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Hyperbaric Oxygen Treatment for Visual Loss Due to Carbon Monoxide Intoxication: A Case Report

Karbon Monoksit Zehirlenmesinin Neden Olduğu Görme Kaybı Için Hiperbarik Oksijen Tedavisi

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ABSTRACT

High oxygen (O_2) consuming organs such as the brain and the heart are easily affected by carbon monoxide (CO) intoxication. The most frequent changes are bilateral ischemic lesions and necrosis in the grey substance of brain tissue, particularly in the globus pallidus. Depending on the severity of intoxication, cerebral and cardiac findings may occur as well as visual impairment, although rare. Visual disorders may develop in patients recovering from coma. In this case report, we present a case with severe loss of vision consequent to CO intoxication, who demonstrated a complete response to long-term hyperbaric oxygen treatment.

Keywords: Carbon monoxide intoxication, visual loss, hyperbaric oxygen treatment
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ÖZET

Beyin ve kalp gibi yüksek oksijen (O_2) harcayan organlar çok kolay bir şekilde karbon monoksit (CO) toksisitesinden etkilenirler. Beyin dokusunda gri maddede ve özellikle globus pallidusta sıklıkla bilateral olan iskemik lezyonlar ve nekroz en sık görülen değişikliklerdir. Zehirlenmenin şiddetine bağlı olarak serebral, kardiyak bulgular oluşabileceği gibi nadiren de olsa görme kusurları oluşabilir. Görme bozuklukları genellikle komadan çıkan hastalarda görülür. Bu sunumda CO zehirlenmesi sonucu ileri derecede görme kaybı gelişen ve yapılan uzun süreli hiperbarik oksijen tedavisine tam yanıt veren bir olgudan bahsettik.

Anahtar Kelimeler: Karbon monoksit zehirlenmesi, görme kaybı, hiperbarik oksijen tedavisi
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Introduction

Hypoxia induced by carbon monoxide intoxication is an important cause of cellular damage and death. High oxygen (O_2) consuming organs such as the brain and the heart are easily affected by carbon monoxide (CO) intoxication. The most frequent changes are bilateral ischemic lesions and necrosis in the grey substance of brain tissue, particularly in the globus pallidus. CO, with its colorless, odorless, tasteless and non-irritant characteristics, is a toxic gas that is easily absorbed through the lungs (1, 2). Findings and symptoms following CO intoxication are nonspecific and variable. The most frequent symptoms are headache, nausea, vomiting and influenza-like complaints. According to the severity of intoxication, it may cause severe cerebral, cardiac, visual disorders or coma (3, 4). In this manuscript, we present a case with CO intoxication complicated with visual loss which responded well to hyperbaric oxygen treatment.

Case Report

A 46-year-old male patient presented to the emergency unit with general poor health in a comatose state. The case was a probable intoxication by a gas heater. In the physical examination, arterial blood pressure was 110/70 mmHg, pulse: 110/minute, axillary temperature was 36.7^{0}C, and the finger tip saturation of O_2 was 95%. On neurological examination, the Glasgow Coma Scale (GCS) score was E_2 V_3 M_4. Other system examinations were unremarkable. Normobaric oxyxen (NBO) was given to the patient by a reservoir mask at a rate of 10 L/minute. Cranial computed tomography was normal. On arterial blood gas analysis,

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the carboxyhemoglobin (COHb) level was found to be 47.4%. On the first day of hospitalization, he was referred to hyperbaric oxygen treatment (HBOT). On the first session, HBOT was administered for 120 minutes under two atmosphere absolute (ATA) pressure. Then, NBO treatment was continued. At the ninth hour of admission, GCS score was 15. He complained of loss of vision 12 hours after the CO exposure, although he had no previous visual disorder. On ophthalmological consultation, the visual acuity was 0.2; he had no anteroposterior camera pathology; the visual field could not be assessed; no retinal damage was detected. On request from the neurology consultation, a brain diffusion magnetic resonance imaging (MRI) was taken (Figure 1), it revealed infarct areas in diffusion tests in the cortical-subcortical white substance of the right parieto-occipital lobe covering a 6 cm area, two infarct foci of 5 and 6 mm in diameter in the subcortical white substance of the right frontal lobe, a 9 mm diameter area of infarct at the level of the right caudate nucleus head, a 3 cm diameter area of infarct in the cortical-subcortical white substance of the medial part of the right temporal lobe, a 9 mm diameter area of infarct in the cortical-subcortical white substance of the posterior part of the left parietal lobe, a 23 mm diameter area of infarct in the cortical-subcortical white substance of the left parieto-occipital region, and a 3 cm diameter area of infarct of millimetric size in the middle section of the vermis in the cerebellar hemisphere. For continuation of treatment, he received a second session of HBOT at the 12th hour of admission. A total of 14 sessions of HBOT were administered. A gradual recovery was observed on daily ophthalmological examinations, and on the fourteenth day, the visual disorder was seen to have been completely ameliorated. On diffusion brain MRI performed one month later (Figure 2), signal intensity changes consistent with encephalomalacia changes were observed in the cortical-subcortical white substance of the right parieto-occipital area and no additional pathological signal intensity changes were observed in the other supratentorial-infratentorial neural parenchyma in diffusion tests. The patient was placed on control follow-ups for six months.

Discussion

Carbon monoxide gas was first discovered in 1776 by the French chemist Lassone. The molecular structure of CO composed of carbon and oxygen was defined by William Cruikshank in 1880 and the toxic effects were defined by Claude Bernard in 1865 (5).

As the CO concentration and the exposure time increase, symptoms ranging from confusion to coma may occur. The diagnosis of CO intoxication is made by clinical suspicion and blood COHb levels. Following CO intoxication, 3-20% of cases display delayed neurological sequelae (6). In this case, the COHb level was 47.4% and GCS score was 9.

In CO intoxications, ventricular dysrhythmias and neurological sequelae are responsible for mortality. Among the neurological and/or psychiatric findings, disorders involving anterograde and retrograde amnesia accompanying confabulation may frequently be observed. Emotional lability, impaired judgment and decrease in cognitive functions may be seen in patients (7, 8). In the presented case, sudden loss of vision 12 hours after exposure was recorded.

The most frequent changes in the grey substance, particularly the globus pallidus, are bilateral ischemic lesions and necrosis. Similar necrotic lesions may be found in the purkinje cells of the cerebellar cortex, dentate nucleus and the cortex. The prevalent psychiatric and neurological symptoms are speech disorders, delirium, epileptic seizures, Parkinsonism, agnosia, ataxia, apraxia and amnesic disorders. In about 40% of cases, milder memory disorders and permanent sequelae such as personality changes have been mentioned (5).

Findings of CO intoxication usually occur due to its effect on the basal ganglia (particularly in the globus pallidus), frontal lobe and the cortical periventricular white substance. MRI is superior to brain tomography in determining the neurological injury (9). In the presented case, while brain tomography was interpreted as normal, diffusion MRI revealed manifest pathologies.

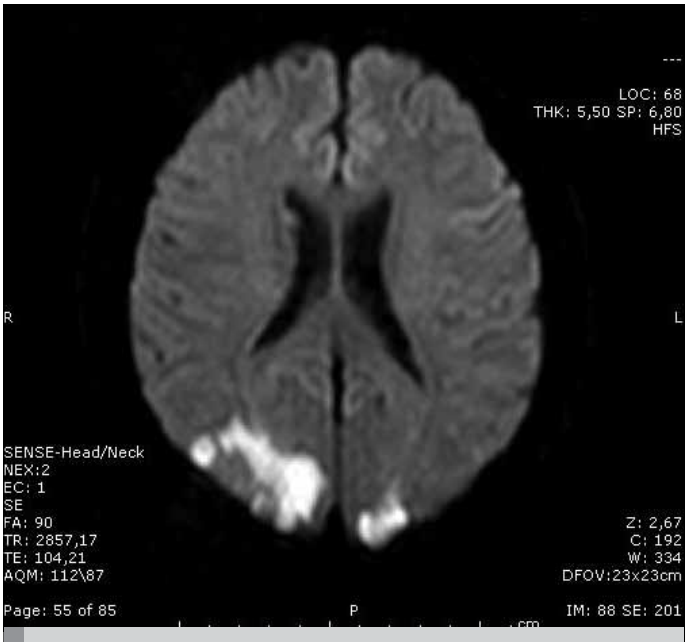

Figure 1. Brain diffusion MRI in the first day

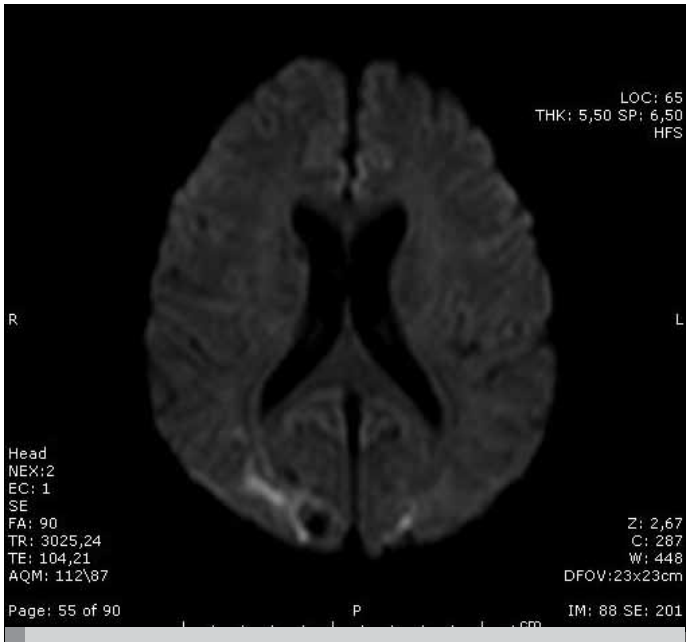

Figure 2. Brain diffusion MRI for control (after 1 month)

Loss of vision in CO intoxication may be due to optic neuropathy or affected visual centers in the occipital lobe. In this case, retinal and optic examinations were within normal limits. Therefore, cerebral causes were suspected as responsible for the loss of vision. On diffusion MRI, the ischemic areas in the occipital lobe explained the loss of vision. Ersanli et al. (10) reported improved vision by HBOT in two cases with loss of vision due to CO toxicity. Improved vision was seen up to the level of 20/100 in one case and to the level of 70/100 in the other. The improvement may not reach a desired level, either due to late commencement of HBOT, or a severe injury occurring consequent to CO toxicity. In the present case, vision was initially at the level of 20/100 and following 14 sessions of HBOT, it reached the level of 100/100. Furthermore, on control diffusion MRI, the previously observed ischemic areas were seen to have disappeared.

Hyperbaric oxygen treatment is the only non-hormonal treatment method approved by the FDA for human tissue repair and regeneration. A faster response has been obtained by HBOT in the treatment of traumatic brain damage. In animal studies and many cellular studies, the positive effect of HBOT has shown the need for it in the r management of traumatic and ischemic brain injury (11).

In the first six hours following intoxication, NBO or HBOT application at 1 atmosphere pressure has decreased the mortality from 30% to 14%, and has reduced the development of neurological disorders (12). 23% of 204 care centers investigated in America were found to have administered more than one HBOT sessions automatically for CO intoxication cases; 10% of these centers administered three sessions. Among institutions, 67% applied multiple sessions, while 8% never applied more than one session (13). In another study, 74% of CO intoxications were treated by more than one HBOT session (13, 14).

Conclusion

In this report, we present a case of CO intoxication complicated with visual loss who responded well to hyperbaric oxygen treatment. It was shown that long-term HBOT therapy for CO intoxication, such as 14 sessions, in the presence of serious complications such as cortical amaurosis, may help to improve the visual findings.

Conflict of interest

No conflict of interest was declared by the authors.

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