Dramatical Clinical Recovery in a Patient with Carbon Monoxide Poisoning Induced Cerebral Cortical Necrosis: Case Report

Karbon Monoksit İntoksikasyonu Sonrası Kortikal Nekroz Gelişen Hastada Sağlanan Belirgin İyileşme

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ABSTRACT Carbon monoxide (CO) poisoning is an important public health problem, which can cause serious cardiac and neurological sequelae, even death in severe cases. One of the rare complications of CO poisoning is personality change because of the frontal lobe necrosis. In this article, we presented a 28-year-old female patient who had been found in an unconscious position after a natural gas activated water heater poisoning. Despite early application of hyperbaric oxygen (HBO) therapy, the patient remained in coma and developed agitation, personality changes, amnesia and coprolalia. Cortical (especially in the frontal lobe) necrosis was determined in MR imaging. HBO treatments (2 hours at 2.5 ATA with 100% oxygen) were continued and the patient's consciousness and neurological symptoms improved day by day. After 23 HBO treatments, her speech, behavior, agitation and coprolali were completely recovered. After CO poisoning, application of 1 to 3 sessions of HBO therapy is generally recommended, but in selected cases the number of sessions can be extended if the patient's neurological condition doesn't improve with initial HBO treatments.

Key Words: Hyperbaric oxygenation; carbon monoxide poisoning; amnesia

ÖZET Karbon monoksit (CO) zehirlenmesi, kardiyolojik ve nörolojik sekellere yol açabilen, hatta ciddi vakalarda ölümle sonuçlanabilen önemli bir halk sağlığı sorunudur. CO zehirlenmesinin nadir görülen komplikasyonlarından biri de frontal lob nekrozuyla gelişen kişilik değişikliğidir. Bu olgu sunumunda, kısa süreli şofben kaynaklı doğalgaz zehirlenmesi sonrası bilinci kapalı halde bulunan 28 yaşındaki kadın hastadan bahsedilmiştir. Erken dönemde uygulanan hiperbarik oksijen (HBO) tedavisine rağmen, hasta komada kalmış ve sonrasında hastada ajitasyon, kişilik değişikliği, amnezi, koprolali gibi nörolojik bulgular gelişmiştir. MR görüntülemede, kortikal nekroz (özellikle frontal lobda) saptanmıştır. HBO tedavisine devam edilerek (günde 2 saat 2,5 ATA'da %100 oksijenle uygulanan) hastada ciddi bir iyileşme görülmüş olup bilinç durumu ve nörolojik semptomları gün geçtikçe düzelmiştir. 23. seans HBO tedavisi sonrası konuşma ve davranış bozukluğu, ajitasyon, koprolali gibi bulguları tamamen iyileşmiştir. CO zehirlenmesi sonrasında genellikle 1-3 seans HBO tedavisi önerilmektedir ancak, seçilmiş vakalarda hastanın nörolojik durumu başlangıçtaki HBO tedavisiyle düzelmemişse, seans sayıları artırılabilir.

Anahtar Kelimeler: Hiperbarik oksijenasyon; karbon monoksid zehirlenmesi; amnezi

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Carbon monoxide (CO) poisoning is a major public health problem around the world. CO poisoning also is very common in our country, because of socio-economic and climatic reasons.

CO is a colorless, odorless, tasteless and non-irritant gas that is formed as a result of incomplete combustion of carbon-containing fuels, coal etc. CO binds to hemoglobin approximately 200 times faster than oxygen, thus

disrupts transport of oxygen to the tissues.[1] CO poisoning in our country usually occurs due to fuels and the others used in heating systems such as stove, water heater, boiler and fireplace. CO poisoning as a result of suicide attempt is rare in Turkey compared to other countries.[2]

Main treatment of CO poisoning is oxygen therapy. Normobaric oxygen (NBO) or hyperbaric oxygen (HBO) treatment is applied according to the clinical status of the patient. HBO treatment was discovered in 19th century and it was firstly used in 1960's. Initially; although acceleration of removal of carbon monoxide from hemoglobin was aimed, other effects were recognized by the time.[3] HBO treatment is recommended in patients with one of the following findings:

- History of unconsciousness,
- Clinical, neurological, cardiac, respiratory or psychological symptoms or signs,
- Pregnancy.[4]

In this article, we presented a patient who suffered loss of consciousness after CO poisoning. Despite the recovery of her consciousness on the 3rd day of the HBO treatment; some symptoms such as agitation, personality change, amnesia and coprolali developed. Therefore we continued HBO treatments. After a month, the control T2-weighted MRI series, showed findings of cortical thickening however the patient was free of symptoms.

The reason we reported this case is three folds. First, although HBO therapy is generally applied only 1 to 3 sessions in CO poisoning, we recommend prolonged use of HBO therapy in selected cases when initial treatments fail to improve patient's neurological symptoms. Second, short-term exposure to CO can also cause severe poisoning. Last but not the least, MR imaging is very useful and remarkable in the follow-up of patients with CO poisoning.

CASE REPORT

A 28-year-old female patient was found unconscious by her sister 40 minutes after entering the bathroom to take a shower. The patient was taken to the nearest hospital by her relatives.

At admission, she was unconscious and had superficial breathing. Her Glasgow Coma Scale was (GCS):4, therefore she was intubated. Test results revealed; carboxyhemoglobin [COHb]: 47.5%, pO_2: 19.2 mmHg, pCO_2: 38.4 mmHg, HCO_3: 9 mmol/L, pH: 6.99, WBC: 24.4 $10^3/\mu L$, K: 2.78 mmol/L, glucose: 349 mg/dL, ALT: 29 IU/L and AST: 65 IU/L. Arterial blood pressure was 108/66 mmHg. Heart rate was 120/minute and body temperature was 36°C. Her electrocardiography (ECG) showed supraventricular tachycardia. Lungs were clear to auscultation. No rales, ronchi or wheezes were heard. Abdominal muscular defense or rebounds were not observed. The pupillary light reflex in both eyes was intact and Babinski's signs were absent.

After the evaluation, the patient was diagnosed with CO poisoning. Oxygen therapy at a rate of 12 L/min was started and she was transferred to our center for HBO therapy. She received normobaric oxygen for 2 hours with a mask at the emergency department and during transport. She was being transferred to our center within 3 hours after the exposure of CO. She received HBO therapy for 2 hours at 2.4 absolute atmospheric pressure (ATA) in a multiplace hyperbaric chamber. After HBO therapy, no improvement was observed. Her GCS was 4. She was transferred to intensive care unit in our hospital. HBO therapy was applied once a day for the first 3 days as usual.

On the fourth day after the poisoning, the patient's consciousness began to open and spontaneous breathing returned. However, we decided to continue HBO therapy and to start additional treatment with intravenous piracetam (3x2 mg) because of the patient's neuropsychological problems, such as hallucinations, memory loss and agitation. Control ECG, which was taken on the 5th day of poisoning, was normal.

Brain diffusion-weighted MRI was taken on the 6th day of poisoning. The cortical areas of the frontal, parietal and temporal lobes showed signal intensity changes diffusion-weighted images and the ADC (apparent diffusion coefficient) maps consistent with the acute-subacute infarcts (Figure 1a, b), signal intensity of the basal ganglia region was

found normal. The cortical and subcortical areas of the bilateral frontal, temporal, and parietal lobes were slightly hypointense on T1-weighted images and hyperintense on T2-weighted and FLAIR (fluid attenuated inversion recovery) sequences, due to sub-acute infarcts. Subacute infarct had led to edema which caused gyral swelling and sulcus narrowing (Figure 2a-c).

Investigations of the patient had no change at the end of the 8th day, piracetam per oral (3x2 mg) and HBO therapy were continued, in addition to mannitol 20% (2x250 mg) and dexamethasone (4x4 mg) therapy which were started due to brain edema observed on MRI. On the 15th day, patient's general condition improved and she was transferred from department of neurology to intensive care department with the findings of limitation of cooperation, disorientation, agitation and coprolalia. Because of the psychiatric symptoms, olanzapine (2x10 mg) was started. On the 21st day, there were not any complaints except from mild agitation and amnesia. On the 24th day after 19th HBO session, agitations and

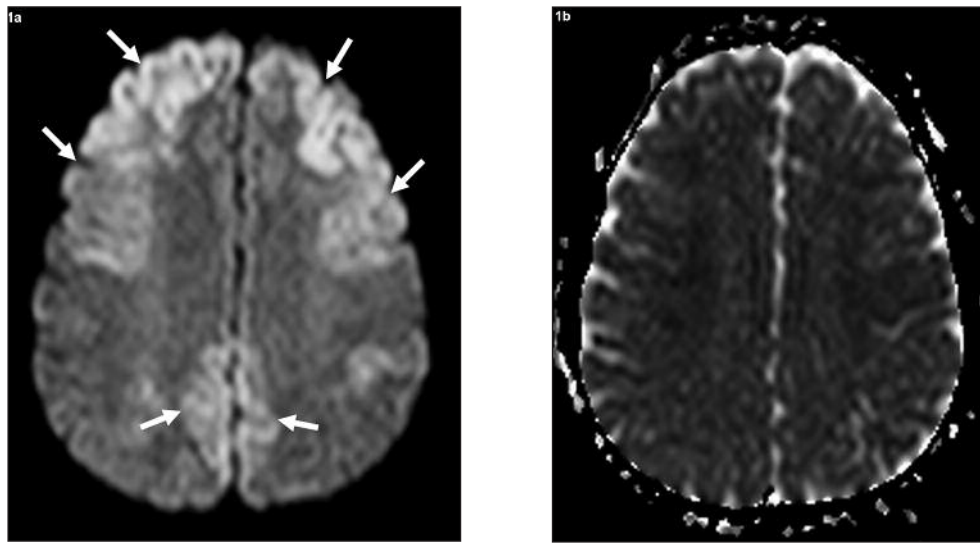

FIGURE 1: On the 6th day, **[a]** b1000 diffusion-weighted images and **[b]** ADC map images, at the cortical areas of the bilateral frontal, parietal and temporal lobes, we observed hyperintense on diffusion-weighted image **[a]** and acute-subacute seen as isointense to slightly hypointense infarct areas [arrows] on ADC maps.

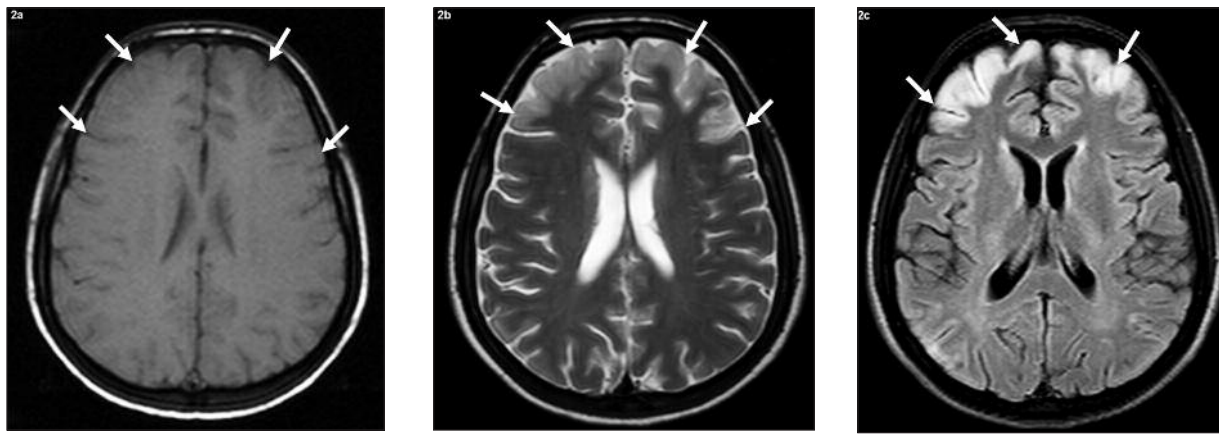

FIGURE 2: On the 6th day, we observed subacute infarcts [arrows] on **[a]** T1-weighted, **[b]** T2-weighted and **[c]** FLAIR sequence images of the cortical and the subcortical areas, slightly hypointense on T1-weighted images and hyperintense on T2-weighted images.

coprolalia were regressed mostly and the patient was discharged from the hospital. On the 30th day, HBO treatment was terminated after 23rd sessions. At the end of the treatments, patient's orientation and cooperation completely recovered; she could recognize their relatives and colors, she could solve the puzzles. Speech and behavior disorders, read and write capabilities, fully returned to normal. After a month, control MRI revealed subacute hemorrhage products, showing Gyral-pattern linear distribution, on old infarct areas (Figure 3). The patient had an increased appetite and extreme quiet mood. The existing complaints are thought to be due to medical therapy. She had no residual symptoms but neurological and psychiatric controls have been continued for a while. Informed consent was obtained from the patient and her sister to report this case.

DISCUSSION

To diagnose CO poisoning, it is required to suspect from the CO intoxication at the health centers which the patients seen first. If the patient's history suggests CO exposure, high COHb level (normal range: <3% in non-smokers and <10% in smokers) is enough for the diagnosis. ECG, cardiac enzymes and echocardiography also must be investigated in severe cases because of the cardiac effects of CO.[5,6]

CT imaging is the best method for imaging bone, calcification, acute hemorrhage or foreign bodies. On the other hand, MR imaging is better for imaging edema, infection or tumor. For cytotoxic edema, diffusion-weighted MRI is the best choice to see ischemia.

In CO poisoning, CT must be taken in the acute phase in severe cases with mental status changes. CT has been used more often in cases of delayed encephalopathy, after CO poisoning. Cerebral atrophy may be seen on CT taken in the delayed period. An initial normal CT scan in a comatose patient does not rule out CO poisoning. After CO poisoning, serial CT scanning showed no lesions until three days.[6] MR imaging is more commonly recommended to visualize and follow focal lesions and white matter demyelinization.[7] In acute CO poisoning, MRI is considered more sensitive examination than serial CT scan and it can demonstrate bilateral edematous lesions in the globus pallidus.[6,8]

CO poisoning may cause, different structural disorders in brain tissue. Ischemic lesions and necrosis, which are often bilateral in gray matter and especially in the globus pallidus, are the most common changes.[9,10] Globus pallidus, putamen, thalamus, substantia nigra, hippocampus and corpus callosum are known as the most being affected sections. Involvement of globus pallidus particularly leads to memory loss. In our case, despite memory deficits we consider microinfarct, because there is not diagnosis of involvement of globus pallidus in MRI. White matter abnormalities are seen on MRI of nearly 1/3 of the patients with serious poisoning.[6]

It is not known whether HBO therapy, applied after 24 hours of poisoning, has an effect on clinical recovery or neurocognitive sequelae. Most HBO specialists, do not suggest HBO therapy if more than 24 hours pass after CO poisoning.[11,12] In our

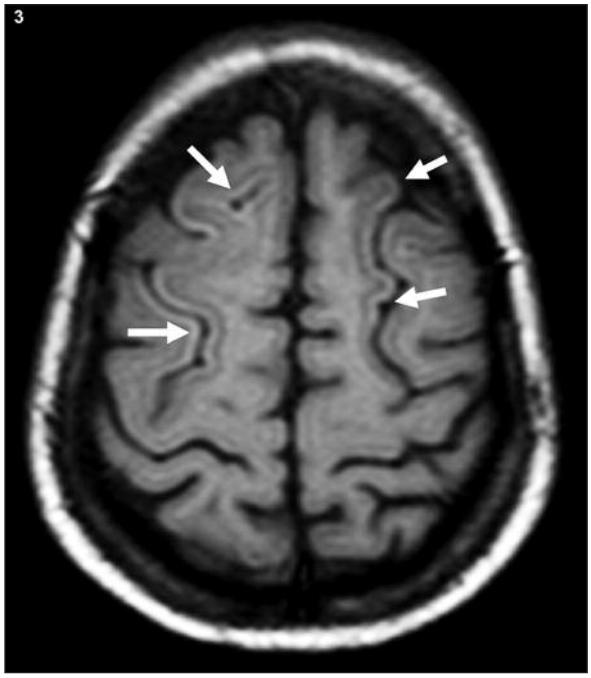

FIGURE 3: In the first month control MRI, on T1-weighted series, subacute hemorrhage products in hyperintense areas which showed gyral-pattern linear distribution were seen.

case, in spite of early HBO therapy, which started after 3 hours of poisoning, clinical status did not improve quickly. However, slow but significant improvement was observed with the continuation of HBO therapy. HBO therapy accelerates the recovery with different mechanism in CO poisoning. HBO therapy provides the elimination of CO 4 times faster than NBO therapy.[13] In addition, HBO treatment has advantages such as improvement of mitochondrial oxidative metabolism, inhibition of lipid peroxidation and reduction of the leukocyte adhesion at microvascular injury sides.[14-16] HBO also reduces cerebral edema.[6] In our case, although patient was arrived to the hospital in less than an hour and HBO therapy was applied within 3 hours, the consciousness was not regained for a long time. It was considered that the initial sessions, HBO especially accelerated the elimination of the CO. The additional sessions of HBO provided the clinical improvement with other mechanisms that aforementioned.

A lucid interval can be seen in CO poisoning.[9,17] It is reported that lucid interval can vary between 2-40 days.[18-20] It is known that prognosis is generally good in delayed encephalopathy. Choi informs that 75% of these cases recover completely in one year.[21] In our case, we continue neurological and psychiatric follow-up for delayed neurological sequelae.

In early stages of ischemia, depending on cytotoxic edema, we can see hyperintensity on diffusion-weighted images and hypointensity on ADC maps. Otherwise edema due to vasogenic cases, we observe hyperintense lesions on FLAIR, T2 and ADC maps and isointense lesions on diffusion MRI.[22-25] In our case, acute infarct areas of laminar necrosis were seen in the first month. It shows that the natural process of infarct areas has been continued and does not support clinical improvement provided by HBO and medical therapy.

As widely accepted, the specific treatment of vasogenic edema is steroid treatment and the most effective treatment of acute CO intoxication is HBO therapy. We used these treatments together in our case and the patient healed completely. We think that HBO therapy can be useful in similar cases in the treatment of delayed neurological sequelae and reduce morbidity. If the patient regains consciousness after initial treatments, HBO therapy should not be extended. Also steroid treatment can be applied only if vasogenic edema occurs after intoxication.

There is no specific treatment for delayed neurological sequelae. The lipid peroxidation is mostly responsible for the development of delayed neurological sequelae. HBO therapy improves brain injuries by inhibition of lipid peroxidation.[26-30]

CONCLUSION

The reason we reported this case is three folds. First, although HBO therapy is generally applied only 1 to 3 sessions in CO poisoning, we recommend prolonged use of HBO therapy in selected cases when initial treatments fail to improve patient's neurological symptoms. Second, short-term exposure to CO can also cause severe poisoning. Last but not the least, MR imaging is very useful and remarkable in the follow-up of patients with CO poisoning.

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