DELAYED NEUROPSYCHIATRIC SYNDROME AFTER CARBON MONOXIDE POISONING

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INTRODUCTION

Carbon monoxide (CO) is an odorless, tasteless, colorless and nonirritating gas [1]. In Spain, most of the accidents due to CO poisoning are caused by water heaters[2,3,4]. CO binds to hemoglobin with much greater affinity than oxygen, forming carboxyhemoglobin (COHb) and resulting in impaired oxygen transport and utilization.XS In up to 40 percent of patients with significant CO exposure, a delayed neuropsychiatric syndrome (DNS) can arise 3 to 240 days after apparent recovery, characterized by cognitive deficits, personality changes, movement disorders, and focal neurologic deficits, which may persist for a year or longer[2]. COHb levels at the acute intoxication are poorly correlated with DNS development, although the majority of cases are associated with loss of consciousness during acute intoxication[5].

CASE REPORT

We present the case of a 60-year-old man who attempted suicide by inhalation of CO in the kitchen. He lost consciousness after the attempt and went to the emergency room three days later, when he recovered consciousness. He smoked 20 cigarettes a day. He suffered from chronic depressive disorder, with a suicide attempt with anxiolytic medication when he was young. Currently he does not go to consultation.

In emergency blood analysis, levels of carboxyhemoglobin were normal (3%), and no significant alterations were observed in the rest of parameters. EEG and EKG were normal. Finally, the patient was transferred to the psychiatric unit. Day fifteen starting to count from the suicide attempt the patient reported paresthesia in his left foot without any other neurological symptoms. On day 21 an attention disorder, bradypsychia, nocturnal disorientation and echolalia were reported. Cranial CT and lumbar puncture were performed without pathological meaning. On day 34 brain MRI was normal. The patient´s evolution was bad, requiring help with every single activity of daily living. Second brain MRI (10 days after first one) showed In T2 and Flair sequence, as well as in diffusion, extensive high-signal images in periventricular white matter, in semioval centrum and in bilateral globus pallidus (predominantly left) (Figure 1). On day 51 due to feeding difficulties he had to use a nasogastric tube. The diagnostic impression was of a severe cognitive impairment, with motor aphasia. From day 59 a progressive recovery was appreciated, answering simple questions. On Day 64 he started rehabilitation. On day 79 a significant improvement was perceived, with more physical independence and cognitive improvement, being able to maintain a conversation. Finally, on day 119 he was discharged. He was correctly oriented and he could independently perform basic activities of daily living.

DISCUSSION

CO binds to the iron moiety of heme with approximately 240 times the affinity of oxygen. The degree of carboxyhemoglobin (COHb) is in function of the relative amounts of CO and oxygen in the environment, duration of exposure, and minute ventilation. Levels above 3% in nonsmokers and above to 10 to 15 percent in smokers are consistent with CO poisoning[6].

The half-life of CO while a patient is breathing room air is approximately 250 to 320 minutes, while breathing high-flow oxygen via a nonrebreathing face mask is about 90 minutes and with 100 percent hyperbaric oxygen is approximately 30 minutes. Standard pulse oximetry (SpO2) cannot screen for CO exposure, because it does not differentiate carboxyhemoglobin from oxyhemoglobin[7]. A carboxyhemoglobin measurement is essential for determining exposure, but levels are not predictive of DNS.

Symptoms after CO poisoning can be mild or as severe as death from central nervous system or cardiovascular system involvement. Headache, dizziness, nausea, vomiting, diarrhea, asthenia, weakness, decreased level of awareness, rhabdomyolysis, as well as heart rhythm disturbances or cardiac ischemia may appear. After an apparent recovery from acute CO poisoning, neurological or behavioral disturbances may appear after days to several months, which is called DNS.

DNS is divided into cognitive, motor or affective symptoms. Cognitive symptoms are disorientation, concentration and attention deficits, decreased spatial and visual skills, less

verbal fluency and memory impairment, and in extreme cases there may be dementia. Motor symptoms include parkinsonism, dystonia, chorea, and myoclonus, and affective/psychiatric symptoms may include anxiety, depression and irritability that should be clarified if they are present after the poisoning or were previously present, especially in suicide attempts. All these symptoms usually have a favorable evolution from months to a year after the onset of DNS, with a 50-75% recovery in less than a year, although some are left with definitive sequelae[1,7,8].

Factors which predict the development of DNS are older age, duration of exposure to CO, longer time to treatment, transient loss of consciousness, increased serum levels of neuron specific enolase[10], increased levels of myelin basic protein in the cerebrospinal fluid or hyperthermia in the first 24 hours after poisoning[11].

COHb concentration was not a predictive factor for DNS, but some studies have found a higher number of DNS in patients with COHb greater than 24%[8], as well as initial consciousness problems and the modification of the tendon reflexes[12]. Neuropsychological sequelae of CO poisoning could be significantly reduced with one session of hyperbaric oxygen (HBO)[12]. In DNS development, a hypersignal in FLAIR (demyelination and gliosis zones), and in T2 are found at globus pallidus and white matter. Atrophy of the hippocampus is related to memory problems, fornix atrophy is related to verbal memory impairment, and visuospatial and visuoconstructive alterations are related to parietal and globe pallidus damage. Presence of acute brain injury in diffusion-weighted imaging during the acute phase of CO is associated with 14 times higher risk of developing DNS compared to patients without brain injury[13]. The most common location was globus pallidus. Therefore, presence of acute brain injury can help to make the decision to apply HBO in order to prevent the appearance of DNS[14].

HBO has been associated with lower mortality after CO poisoning in the acute phase and should be administered as soon as possible (first 6 hours). There is controversy regarding the prevention of DNS, some recommend it, but others do not find advantages[15]. A Cochrane review[16] recommended HBO, also other studies found less appearance of DNS with HBO[17,18] and another Spanish study did not find statistically significant differences but did improve with HBO in a small sample with COHb higher than 35%[19] .

HBO is also performed in the chronic phase, as the case of a patient who improved in symptoms such as dystonia or parkinsonism and recovery in verbal fluency 14 months after the poisoning and more than 100 sessions with HBO later[20].

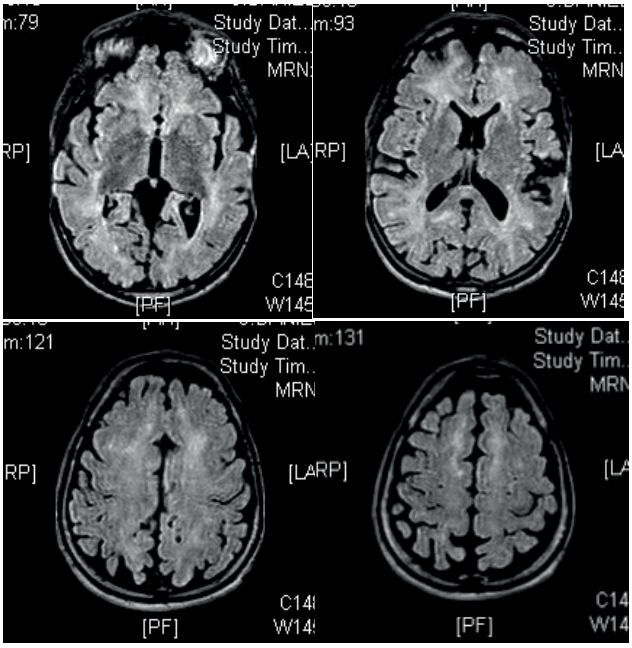

Figure 1 | Second brain MRI

It has been published a case of DNS improvement and disappearance of the MRI lesions, after umbilical cord blood (UCB) stem cells transplantation, combined with injection of nicholine and intranasal inhalation of insulin[21]. UCB transplantation is associated with angiogenesis, neurogenesis, the inhibition of apoptosis and inflammation, and increased trophic support.

CONCLUSION

It would have been important to consider that the patient lost consciousness days before coming to the emergency room in order to treat him with HBO despite the absence of symptoms. It is possible that HBO could have prevented the development of DNS. Once the DNS was established the patient recovered spontaneously and without sequels. Even though there are limited publications about DNS, acute and long-term treatment must be considered as was explained before.

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