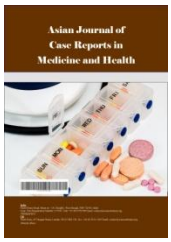

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Cortical White-matter Lesions as Initial Radiologic Finding in Carbon Monoxide Intoxication: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Aims: Present a case of carbon monoxide (CO)-intoxication with initial presence of cortical lesions instead of globus pallidus lesions, to create awareness of atypical or late MRI findings in patients with CO-intoxication.
Presentation of the Case: A 27 year old women with no medical history presents with a reduced level of consciousness due to CO-intoxication. After a lucid interval of two to three days, patient develops altered behavior, headache and loss of short term memory. This symptoms worsen and seven days after discharge she is readmitted to the hospital. MRI of the cerebrum shows cortical hypoxic lesions, but no abnormalities in the basal ganglia. There is only little clinical improvement in the following weeks. Repeated MRI on day 20 shows newly developed lesions in the globus pallidus, pathognomic for CO-intoxication.
Discussion and Conclusion: This case demonstrates how radiologic lesions can develop until weeks after the clinical presentation in patients with CO-intoxication, and that cortical lesions can develop in the absence of globus pallidus lesions. CO-induced cerebral lesions can be explained by several complex mechanisms, not only by hypoxia.

Keywords: Carbon monoxide; intoxication; globus pallidus; cerebral cortex; neuroradiology.

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1. INTRODUCTION

Carbon-monoxide (CO)-intoxication yearly affects many patients worldwide and can lead to high morbidity and mortality. Diagnosis can easily be overlooked as clinical presentation is unspecific and complex. Although elevated carboxyhemoglobin levels confirm diagnosis, clinical presentation does not always correlate with carboxyhemoglobin levels. Furthermore, late neurological symptoms can develop until several days or weeks after intoxication [1].

The treatment for CO-intoxication is oxygen therapy (through a non-rebreather mask or hyperbaric oxygen chamber) in order to restore the reduced oxygen-carrying capacity of the blood and repair the impairment of the cellular respiratory chain.

Here, we report a patient with delayed encephalopathy, due to CO-intoxication, and its atypical radiological findings.

2. CASE PRESENTATION

A 27 year old women presented to the emergency room after she was encountered unresponsive and hypothermic in her bathroom. The patient had no medical history except for the regular use of ketamine and cannabis. Vital signs at presentation included a temperature of 32.6 degrees Celsius, blood pressure of 110/80 mmHg and respiratory rate of 20/min. Neurologic examination showed a Glasgow Coma Scale of 8, a conjugate horizontal eye deviation to the right and pinpoint pupils. Laboratory findings revealed slightly elevated levels of both amphetamine and cannabis. Furthermore, it revealed high levels of carboxyhemoglobin in both venous and arterial blood gasses, respectively 22.6% and 18.2%. Computer Tomography (CT) of the cerebrum showed no abnormalities. Clinically, there were no signs of epilepsy [1]. She was admitted to the Intensive Care Unit for observation of carbon monoxide intoxication and body temperature and received treatment with oxygen therapy. Carboxyhemoglobin levels gradually dropped and the patient showed full recovery within the first 24 hours and the patient was discharged.

The next day, she returned to the emergency room with progressive behavioral disorders, headache, loss of initiative, disordered short term memory and impaired ability to imprint. She was readmissioned at the Neurology Department. During this admission, she showed serious behavioral abnormalities, mainly restlessness and aggression, and no improvement of conjugate gaze movement abnormalities. Magnetic Resonance Imaging (MRI) of the cerebrum which took place a week after initial presentation showed a biparietal hyperintensity on the T2 weighted FLAIR images, with no abnormalities at the basal ganglia (Fig. 1). Electro-encephalography showed significant symmetrical slow wave activity suggesting encephalopathy without any signs for epileptic activity.

At day 25 after CO-intoxication repeated MRI of the cerebrum still showed the aforementioned areas of cortical white matter lesions, but this MRI now also showed bilateral hypoxic lesions in the globus pallidus (Fig. 2), which is pathognomic for a CO-intoxication.

The patient spent a total of six weeks in the hospital before discharge to a neurological rehabilitation center. In the last three weeks of her admission, she showed clear clinical improvements in behavior and cognition, but still suffered from persistent short memory disorders and total amnesia of the first period of her readmission.

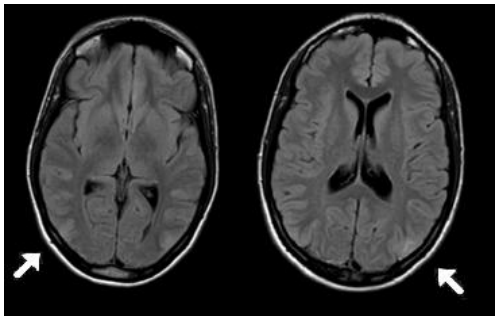

Fig. 1. MRI cerebrum seven days after CO-intoxication with parietal-occipital hyperintensities on a T2 weighted FLAIR image

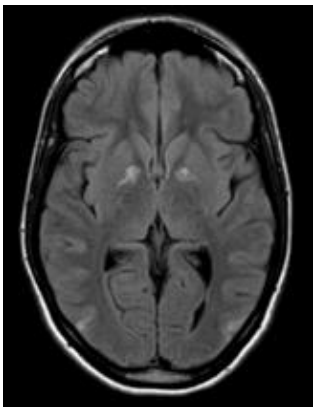

Fig. 2. MRI cerebrum 3-4 weeks after CO-intoxication with parietal-occipital hyperintensities and the typical lesions in de globus pallidus

In the differential diagnosis, withdrawal symptoms of cannabis use was considered. However during the first admission, cannabis use was discontinued but symptoms still improved drastically in the first 24 hours. Laboratory findings during the second admission consequently revealed elevated cannabis levels, meaning patient did use cannabis during this period, but symptoms did not improve until after three to six weeks into her readmission. This makes withdrawal as a cause of the symptoms highly unlikely.

3. DISCUSSION

CO-intoxication yearly affects approximately 300-500 patients in the Netherlands and 50.000 patients in the United States of America [2,3]. It can lead to high morbidity and mortality. CO arises from the incomplete burning of hydrocarbons. It has an affinity for the hemoglobin molecule which is approximately 240 times higher than that of oxygen, and thus replaces bounded oxygen, reducing the oxygen-carrying capacity of hemoglobin. The attendant tissue hypoxia is considered to be the major mechanism of CO-toxicity [4,5]. However, severity of first clinical presentation of CO-intoxication has been noted not to correlate with the blood carboxyhemoglobin levels and clinical improvement does not correlate with clearance of it. This implies hypoxia is not the only factor contributing to tissue damage [4,5]. It is suggested that some toxic effects of CO are the result of CO binding to enzymes, inhibiting the electron transport chain [6]. As well, immune-modulation processes can lead to tissue injury in the brain even after lowering the carboxyhemoglobin concentration [7].

In this case presentation we report a patient, in which radiologic findings at MRI seven days after CO-intoxication were limited to two symmetrical cortical white matter lesions. Typical lesions of the basal ganglia did not show at initial scans, but only at repeated MRI after three weeks.

Delayed encephalopathy has been described before, occurring several days or weeks after CO-intoxication [8]. Case reports describe patients developing behavioral abnormalities to up to four weeks after the initial presentation [9-11]. Delayed radiologic findings within six weeks of discharge occur in about 26% of patients with CO-intoxication. Radiologic abnormalities are most commonly found in the globus pallidus, as this structure is typically most vulnerable to hypoxia. Cortical lesions without involvement of the basal ganglia have never been described: previous studies show cortical involvement is only related with bilateral involvement of the globus pallidus [12,13].

Hypoxic lesions in the globus pallidus will normally be seen on MRI shortly after CO-intoxication, as the globus pallidus is susceptible for tissue hypoxia. The occurrence of cortical ischemic lesions in absence of globus pallidus lesions can be explained by the fact that, in addition to tissue hypoxia, other pathophysiological events contribute to cerebral tissue damage.

One of the mechanisms which could be responsible for the development of delayed tissue damage is the formation of cytotoxic edema and reactive oxygen species as a result of tissue hypoxia. CO-induced cardiac dysfunction could contribute to the development of focal embolic infarcts, which could cause

multiple infarcts in various vascular territories. CO causes myocardial injury through impairment of oxidative phosphorylation [14]. Additional mechanisms on the formation of intracardiac thrombi following CO-intoxication have been described: CO-intoxication attenuates the susceptibility of clot lysis, as CO makes the thrombus resistant to tissue plasminogen activator by enhancing alfa-2-antiplasmin interaction. CO-intoxication promotes the formation of oxygen free radicals, which alter platelet aggregation and blood flow, contributing to endothelial damage. Endocardial ischemic injury caused by the impairment of oxygen delivery by hemoglobin can induce secondary intracardiac thrombus formation, as a result of cardiomyopathy or dysrhythmias [15].

In this case we confirmed the CO-intoxication before the MRI's were made, which is why we focused on differential diagnosis of hypoxic lesions in combination with CO-intoxication. If no diagnosis is found, consider to extend the differential diagnosis with all causes for young ischemic stroke, including carotid or vertebral dissection, atherosclerosis, but also vasculitis and neurosarcoidosis [16,17].

4. CONCLUSION

In conclusion, cortical white matter lesions as the initial radiologic finding in patients with CO-intoxication has not been described often but can be explained by several complex mechanisms. The multitude of causative and contributory factors of cerebral tissue damage explains the potential development of lesions in brain areas not susceptible to hypoxia. Although recognizing hypoxic lesions as a part of CO-related damage, treatment of both CO-intoxication and (delayed) encephalopathy does not differ for different causes.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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