Hemiplegia resulting from acute carbon monoxide poisoning

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Abstract

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Carbon monoxide (CO) poisoning can cause neurological complications such as movement disorders and cognitive impairment through hypoxic brain damage. Although peripheral neuropathy of the lower extremities is a known complication of CO poisoning, hemiplegia is very rare. In our case, a patient who developed left hemiplegia due to acute CO poisoning received early hyperbaric oxygen treatment (HBOT). The patient had left hemiplegia and anisocoria at the beginning of HBOT. Her Glasgow coma score was 8. A total of five sessions of HBOT at 243.2 kPa for 120 minutes were provided. At the end of the 5th session, the patient's hemiplegia and anisocoria were completely resolved. Her Glasgow coma score was 15. After nine months of follow-up, she continues to live independently with no sequelae, including delayed neurological sequelae. Clinicians should be aware that CO poisoning can (rarely) present with hemiplegia.

Introduction

Carbon monoxide (CO) poisoning can cause neurological complications such as movement disorders and cognitive impairment through hypoxic brain damage. Besides peripheral neuropathy of the lower extremities, a known complication of CO poisoning, hemiplegia due to CO poisoning, is very rare.[1] We present a patient with left hemiplegia caused by acute CO poisoning and whose neurological symptoms were completely resolved after five sessions of hyperbaric oxygen treatment (HBOT).

Case report

The patient consented to her case details being reported.

An 82-year-old female with prior diagnoses of hypertension and Parkinson's disease and who lives alone was found unconscious in her coal-burning stove-heated home by her relatives and transferred to the local hospital's emergency unit. On admission the Glasgow Coma Scale (GCS) was 6 ($E_2V_1M_3$), and her blood pressure was 80/50 mmHg. Arterial blood gas test results were as follows: carboxyhemoglobin (COHb): 36%; pH: 7.18; lactate: 14.4 (mmol·L^{-1}) and the white blood cell count was 21.38 (10^9·L^{-1}).

The patient was not intubated because her peripheral oxygen saturation was 96–98% while breathing oxygen with a reservoir face mask at the rate of 10 L·min^{-1}. Her $PaCO_2$ was 3.54 kPa, respiratory rate was 18·min^{-1} and no pathological respiratory pattern was observed. Her cardiac evaluation was normal. The patient was given a preliminary diagnosis of carbon monoxide poisoning and HBOT was recommended.

In the first examination of the patient at the hyperbaric department, redness and abrasion were observed in the face's left forehead and left cheek area. She had spontaneous respiration, and while breathing 100% oxygen with a mask, her oxygen saturation was 99%, pulse rate was 105–110·min^{-1}, blood pressure was 130/90 mmHg, and GCS was 8 ($E_2V_2M_4$). In the neurological examination the pupils were anisochoric; the left pupil was more dilated than the right pupil. There was no pupillary light reflex on the left side. Additionally, a left hemiplegia was noted with a positive Babinski sign on the left side. Anisochoric pupils and positive Babinski sign suggested central nervous system pathology rather than peripheral neuropathy. There was no response to painful stimulus in the left lower and upper extremities. Bilateral respiratory sounds were natural, and no pathological sounds were heard. The relatives of the patient were interviewed, and it was learned there was no pre-existing mobility issues. Before the patient was accepted for HBOT, a brain computed tomography (CT) scan was performed to exclude intracranial haemorrhage. No pathology was detected on the CT scan. The patient began HBOT at the 5th hour of her admission to the emergency service.

In the examination performed after the first HBOT session she opened her eyes when called by her name, made meaningless sounds, and withdrew to pain. The GCS was 9 ($E_3V_2M_4$). The patient was advised to continue HBOT sessions beginning the next day. In the neurological examination performed after the second session, GCS was 13 ($E_4V_4M_5$); in the motor examination, the strength in the left lower and upper extremities was 0/5, and the pupils were isochoric. Bilateral pupillary light reflex was obtained. Brain diffusion MRI and neurology consultation were requested. No diffusion restriction was detected. Carotid Doppler ultrasound was recommended for differential diagnosis and was normal. No brain pathology was detected in the neurological examination performed by the neurologist. In the examination performed after the third session, GCS was 14 ($E_4V_4M_6$). Pupils were isochoric and the light reflex was detected bilaterally. Left lower and upper extremity motor strength was 2/5. The eyes opened spontaneously and in response to voice. In the examination performed after the fourth session, the motor strength was 4/5; after the fifth session, the motor strength was 4/5, and GCS was 15 ($E_4V_5M_6$). HBOT was terminated after neurological symptoms were completely resolved after five sessions.

Discussion

Carbon monoxide is a colourless, odourless, highly toxic gas with an affinity for haemoglobin 200 to 250 times greater than oxygen. It may cause tissue hypoxia and inhibition of mitochondrial function.[2,3] By reducing oxygen delivery and mitochondrial oxidative phosphorylation, CO can cause ischaemic brain damage and cognitive dysfunction in survivors.[4] Associated excitotoxicity, acidosis, ion imbalance, depolarisation, oxidative stress, nitrative stress, inflammation, and apoptosis can result in brain damage.[5] When cerebral oedema and focal necrosis are seen, more degenerative and demyelinating changes could develop in the brain. The corpus callosum, hippocampus, and substantia nigra are the most affected.[6,7] Demyelination in the cerebral cortex, which has perivascular spread, is a common neurological finding.[6] Also, neuropathy can be seen in peripheral nerves.[7]

Although the brain constitutes approximately 2% of the body mass, it uses 20% of all oxygen taken into the body.[8] The brain needs continuous and sufficient oxygen to perform its functions.[9] For this reason, the brain is extremely sensitive to hypoxia and ischaemia. Hypoxic brain injury, also called hypoxic-ischaemic encephalopathy, is a serious consequence of cerebral ischaemia due to carbon monoxide poisoning or other causes (such as myocardial infarction or cerebrovascular event).[10] Carbon monoxide poisoning may result in serious morbidity and mortality.[11] As a result, deterioration in brain function causes motor, cognitive, behavioral, and functional disorders.[12] In the case we present, we believe that the patient developed left hemiplegia and anisocoria due to hypoxic injury and mitrochondrial impairment with consequent injury in the brain stem. Other common causes of the presentation were excluded by the various investigations performed. Very few cases of hemiplegia due to acute CO poisoning are reported.[1,13] In our case, early treatment with HBOT was associated with a complete recovery. After nine months of follow-up, she continues to live independently with no sequelae, including delayed neurological sequelae. We report this rare presentation of CO poisoning, which can easily be misattributed to other causes, to raise awareness of the possibility among clinicians.

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