

Magnetic resonance imaging findings after acute carbon monoxide poisoning[☆]



Hallazgos en resonancia magnética nuclear craneal tras intoxicación aguda por monóxido de carbono

Dear Editor:

Carbon monoxide (CO) is the most common toxic gas. It is odourless, colourless, inflammable, and highly toxic. CO is released during incomplete combustion of carbon-containing substances. This gas is the most frequent cause of death by accidental poisoning, as well as being responsible for a significant percentage of suicides. Prevalence of death by CO poisoning increases during the winter due to absent or deficient ventilation of domestic heaters and the higher incidence of depression during the winter months.¹ CO causes adverse effects by binding to haemoglobin. Once inhaled, it passes into the bloodstream. The affinity of CO for the haem groups is 250 to 300 times that of oxygen, causing it to bind tightly (although not irreversibly) to haemoglobin to form carboxyhaemoglobin.² This leads to decreased blood oxygen content which mainly affects the CNS and myocardium. Toxicity depends on the exposure time and amount inhaled, and it may be either acute or chronic. Initial symptoms range from nonspecific signs such as headache, dizziness, nausea, vomiting, and confusion, to events including loss of consciousness, coma, and death.^{3–5} Morbidity after CO poisoning includes neurological sequelae, mainly cognitive impairment, parkinsonism, and psychiatric symptoms.^{6–8} We present the case of a 38-year-old man with a personal history of paranoid

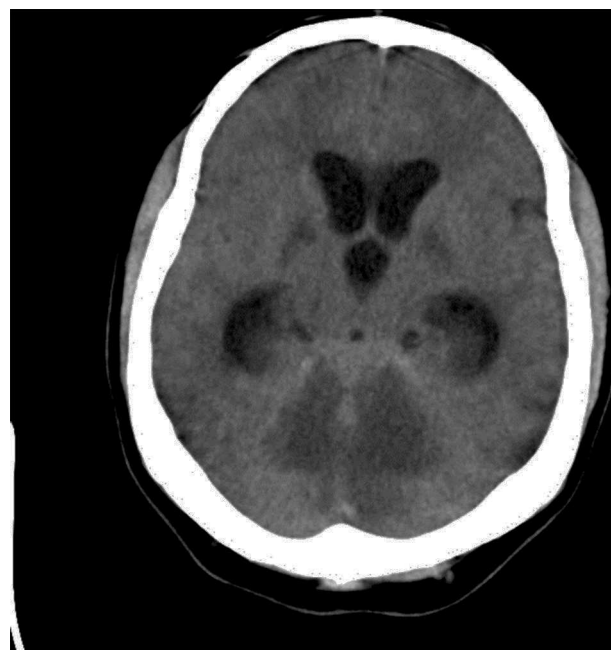


Figure 1 Head CT. Diffuse hypodensities in both cerebellar hemispheres with low attenuation in basal temporal white matter bilaterally and in basal ganglia. Dilated lateral ventricles and third ventricle.

schizophrenia who was admitted to our hospital after having been found unconscious in a closed car and inhaling exhaust fumes. Two empty benzodiazepine blister packs were found beside him. The most relevant findings from the initial examination were low oxygen saturation and Glasgow coma score of 3. Carboxyhaemoglobin concentration was 23%. An initial head CT ([Fig. 1](#)) revealed diffuse hypodense

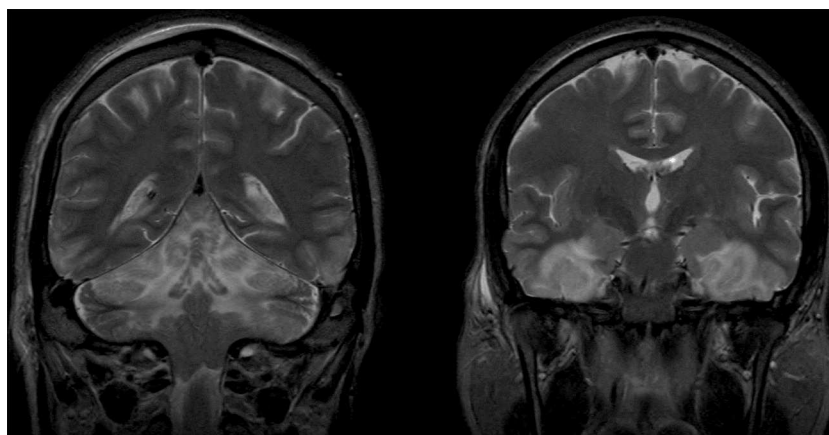


Figure 2 Head MRI. Coronal T2-weighted brain MRI showing multiple anoxic-ischaemic lesions in an early subacute stage. Lesions extensively affect the limbic system, hippocampus, fornix, and temporal and basal area of both hemispheres. Small patchy areas in frontal and parietal lobes. Large lesions in both cerebellar hemispheres. Catheter for ventricular drain was placed in the right lateral ventricle.

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areas in both cerebellar hemispheres, the basal temporal white matter (bilaterally), and basal ganglia. There were initial signs of recovery during the first 48 hours, when the patient's Glasgow coma score rose to 10, after which his symptoms exacerbated. Head CT scans showed severe dilation of the lateral and third ventricles which was compatible with obstructive hydrocephalus and required ventricular drain placement. A subsequent follow-up cranial MRI (Fig. 2) showed that hydrocephalus had resolved. Bihemispheric lesions persisted and significantly affected the limbic system, hippocampus, fornix, and basal temporal areas, the frontal and parietal lobes, and both cerebellar hemispheres. Findings were compatible with multiple bilateral anoxic-ischaemic lesions secondary to CO poisoning. After the acute phase, the patient continued to show severely impaired cortical functions associated with gait ataxia and one-sided akinetic-rigid syndrome. No signs of improvement were observed.

Imaging studies, especially head MRI, have demonstrated high sensitivity for detecting brain abnormalities after CO poisoning.^{1,9} They provide physicians with details regarding characteristics and scope of the damage and correlate better with short- and long-term patient outcomes than any other clinical or analytical parameters. Although the first findings of MRI studies may be normal,¹ they usually show hyperintensities on T2-weighted sequences.¹⁰ The most frequently affected areas are the brain regions with higher levels of iron (which are thus targeted by carboxyhaemoglobin): the basal ganglia and especially the globus pallidus, and the substantia nigra. Periventricular white matter and subcortical white matter of the temporal lobe, including the hippocampus, thalamus, and cerebellum, can also be affected by CO toxicity.^{1,4,9} Our patient presented extensive abnormalities, both supra- and infratentorial, with a devastating neurological outcome.

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