Diffusion-weighted MR imaging findings in carbon monoxide poisoning

Abstract Diffusion-weighted MR imaging (DWI) of two patients with carbon monoxide (CO) poisoning demonstrated white matter and cortical hyperintensities. In one patient, the changes on the FLAIR sequence were more subtle than those on DWI. The DWI abnormality in this patient represented true restriction. In the second patient, repeated exposure to CO caused restricted diffusion. DWI may be helpful for earlier identification of the changes of acute CO poisoning.

Keywords Carbon monoxide poisoning · Magnetic resonance imaging · Diffusion-weighted imaging

Introduction

Carbon monoxide (CO) is a colorless, odorless gas that is the most common cause of poisoning death and morbidity in the United States [1]. Clinically, patients with CO poisoning experience a wide range of neuropsychiatric abnormalities including psychosis, personality change, dementia, concentration deficit, gait disturbance, fecal or urinary incontinence, mutism and memory loss [2, 3].

The most characteristic neuropathological findings in CO poisoning are bilateral necrosis of the globi pallidi and multifocal areas of demyelination in the periventricular white matter, with sparing of the subcortical U fibers [2].

Magnetic resonance imaging (MRI) is very sensitive to changes in white matter, and its superiority over computed tomography has been reported in CO poisoning on conventional MR imaging [4]. The most common MR findings are bilateral hyperintensities of the periventricular white matter and centrum semiovale, ischemia or infarct of the basal ganglia, and diffuse hypointensity of the thalami on T2-weighted images suggesting iron deposition [2].

We report diffusion-weighted MR imaging (DWI) findings in two patients and discuss the potential utility of DWI in diagnosis and prediction of prognosis in acute CO poisoning.

Case reports

Case 1

A 21-year-old man was admitted to our hospital with unresponsiveness. He was found after sleeping overnight in the cabin of a cruise boat with the motor running. Two of his friends were found dead from CO poisoning at the scene. Vital signs were unremarkable. Neurological examination revealed a decrease in motor strength and coordination. Cardiovascular examination revealed sinus tachycardia. Abnormal laboratory values were as follows: carboxyhemoglobin (COHb) 24% (N: 0–2); creatine kinase, 2,764 U/l (N: 25–90); AST 80 U/l (N: 15–40).

MRI was performed on a 1.5-T unit 48 h after the patient was found. Turbo fluid-attenuated inversion recovery (TFLAIR), 6,500/105/1 (TR/TE/NEX) images demonstrated subtle, increased signals in the subcortical white matter of the parietal lobes bilat-
erally (Fig. 1a). DWI was obtained with single-shot spin-echo echoplanar technique in three orthogonal diffusion gradients with a b value of 1,000 s/mm² as well as a b value of 0 s/mm². The imaging parameters were as follows: 4,000/100/5 (TR/TE/NEX); slice thickness 5 mm; field of view 24×24 cm; and matrix 96×200. Total acquisition time for DWI was 20 s. DWI demonstrated obvious abnormal signals in the subcortical white matter of the parietal lobes bilaterally and also in the cortex of the right medial frontal lobe (Fig. 1b). Apparent diffusion coefficient (ADC) values of the abnormal regions in the parietal subcortical white matter and right medial frontal lobe cortex were 0.30×10⁻³ mm²/s and 0.51×10⁻³ mm²/s, respectively, while ADC values of normal white matter and cortex were 0.82×10⁻³ mm²/s and 0.80×10⁻³ mm²/s, respectively (Fig. 1c). Hyperbaric oxygen therapy at 2.4 atm for 90 min was administered three times in the first 24 h. At the end of treatment, COHb level was 2%. Although the patient had developed a severe neurological syndrome with both decerebrate posture and coma, substantial improvement in the level of consciousness was observed after completion of the hyperbaric oxygen treatment. His hospital course was complicated by severe adult respiratory distress syndrome (presumably on the basis of aspiration), rhabdomyolysis, metabolic acidosis and myocardial injury. Symptoms from these complications almost completely improved over his 5-day hospital stay and the patient was discharged with residual memory and coordination difficulties. The patient was recommended to see his cardiologist in 2 weeks due to myocardial injury secondary to acute CO poisoning.

Case 2

A 32-year-old man was admitted to our hospital with mental status changes, ataxic gait, urinary and fecal incontinence. The patient was not taking any medications upon admission. Neurological examination revealed cognitive impairment and wide-based gait with small steps. Electroencephalogram showed absence of posterior rhythm and background slowing consistent with nonspecific diffuse cerebral dysfunction.

MRI was performed using a 1.5-T unit. T1FLAIR 6,500/1,05/1 (TR/TE/NEX) images demonstrated bilateral diffuse areas of increased signal in the posterior limbs of internal capsules as well as in the periventricular and supratentorial cerebral white matter bilaterally (Fig. 2a). DWI was obtained with the same technique and parameters as in the first case. DWI demonstrated hyperintense signal corresponding to the abnormalities on FLAIR (Fig. 2b). An ADC map did not demonstrate definite restriction (Fig. 2c). ADC values in the hyperintense white matter areas in the frontal and temporal lobes were 0.79×10⁻³ mm²/s and 0.74×10⁻³ mm²/s, respectively, while the ADC value of normal white matter was 0.84×10⁻³ mm²/s. The MRI findings were interpreted as nonspecific but likely secondary to toxic or metabolic white matter disease. The patient’s cognitive impairment showed mild improvement during his hospital stay despite the absence of a specific diagnosis and therapy. Following his discharge, during return visits he demonstrated further improvement. However, 2 months after his original admission, he presented with headache, nausea, vomiting and mental status changes. Neurological examination again revealed cognitive impairment and ataxic gait. Laboratory results were again unremarkable. An MRI study was performed with the same parameters as in his first study. DWI again demonstrated hyperintense lesions in the posterior limbs of internal capsules as well as in the periventricular and supratentorial cerebral white matter bilaterally (Fig. 2d). This time, however, the lesions were more hyperintense than on the prior MRI study. ADC values in the hyperintense white matter areas in the right and left frontal lobes were 0.71×10⁻³ mm²/s and 0.74×10⁻³ mm²/s, respectively, while the ADC value of normal white matter was 0.93×10⁻³ mm²/s. An ADC map demonstrated subtle restriction of water diffusion in the cerebral white matter bilaterally (Fig. 2e). He was discharged for outpatient follow-up on day 3 on his second hospital stay. Upon a further, more rigorous, examination of his medical history, it was found that the patient had been homeless throughout the period of his symptoms and had lived in an abandoned apartment. He became ill every time he went back to his apartment. He had exacerbation of symptoms in the morning and would improve throughout the day. His symptomatic period was during winter, and there appeared to be a backdraft of air from a garage below his upstairs apartment.

![Fig. 1a-c](image1.png)

**Fig. 1a-c.** A 21-year-old man presenting with unconsciousness from acute carbon monoxide poisoning. **a** Axial TFLAIR image (with slight motion artifact) demonstrates subtle hyperintense lesions in the subcortical white matter of the parietal lobes bilaterally (arrows). **b** Axial diffusion-weighted image demonstrates obvious lesions in the subcortical white matter of the parietal lobes bilaterally (small arrows) and in the right medial frontal lobe cortex (large arrow). **c** Axial ADC map reveals restricted water diffusion in the subcortical white matter of both parietal lobes (small arrows) and right medial frontal lobe cortex (large arrow).