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Hemorrhagic Necrosis and Vascular Injury in Carbon Monoxide Poisoning: MR Demonstration

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Summary: MR imaging of a patient 3-years status post-carbon monoxide (CO) poisoning revealed areas of abnormal signal bilaterally in the region of the globus pallidus that had shorter T1 characteristics and longer T2 characteristics than cerebrospinal fluid, probably representing methemoglobin, and that is surrounded by a rim of decreased signal on T2-weighted images, felt to represent hemosiderin. This case demonstrates characteristic findings on MR imaging of CO poisoning, as well as observations that suggest prior focal hemorrhage. Typical findings, neuropathology, and the role of vascular injury and prognosis are discussed.

Index terms: Carbon monoxide; Basal ganglia, magnetic resonance; Brain, necrosis

Computed tomography imaging of the central nervous system following carbon monoxide (CO) poisoning has been well-documented. Findings include unilateral or bilateral and symmetrical or asymmetrical low-attenuation lesions in the globus pallidus and white matter (1–3), which correlate with selective necrosis of the globus pallidus (4, 5) and necrosis or demyelination of the white matter (6) seen on neuropathologic specimens. Early reports suggest that magnetic resonance (MR) imaging is superior to CT in demonstrating CO poisoning (7–11). MR imaging in our case suggests that the necrotic damage is of vascular origin (12).

Case Report

A 29-year-old white man, who had attempted suicide by inserting a hose attached to the tailpipe of his car into his mouth, presented 3 years later with irritability, violent actions, and verbal aggression. The original exposure was significant with respect to the following: 1) CO exposure was estimated to be 30 minutes. 2) The patient required intubation and ventilatory support for 3 weeks. 3) Physical examination revealed perioral cyanosis, labored breathing with a respiratory rate of 28, and, neurologically, the patient was obtunded with response to deep pain by movement

only and had deep tendon hyperreflexia and cog-wheel rigidity in all extremities. He also had marked myoclonus in the right upper extremity and no bowel or bladder control. 4) EEG showed slow dysrhythmia consistent with diffuse encephalopathy. Neurologic evaluation during the current illness showed marked improvement, but deep tendon reflexes were increased, spastic clonic movements of the upper extremities were observed, and there was increased muscle tone in all extremities.

MR imaging was done using a 1.5-T unit. Proton density, 2500/22/2 (TR/TE/excitations), and T2-weighted, 2500/90/2, images were obtained. These revealed diffuse central and cortical cerebral atrophy and diffuse cerebral cortical and subcortical increased signal on T2-weighted images that were more prominent in the temporal regions. There was mild diffuse cerebellar atrophy and a focal area of increased signal on T2-weighted images in the periphery of the left cerebellar hemisphere. Additionally noted were areas of abnormal signal bilaterally in the region of the globus pallidus that had shorter T1 characteristics than cerebrospinal fluid (CSF) on the proton-density images (Fig. 1) and longer T2 characteristics than CSF on the T2-weighted images (Fig. 2). Also noted was a small rim of decreased signal on T2-weighted images surrounding this area in the globus pallidus (Fig. 2).

Discussion

Previously documented characteristic changes on CT evaluation of CO poisoning include bilateral and symmetrical low-attenuation lesions in the globus pallidus and white matter (1, 2). In addition, a case revealing a unilateral low-attenuation lesion in the globus pallidus and marked asymmetrical involvement of the white matter has been reported (3). Previous cases of CO poisoning utilizing MR imaging have revealed abnormalities in the same regions. Observations include increased signal on T2-weighted images in the globus pallidus and white matter (7–11). Also reported in one case was bilateral increased

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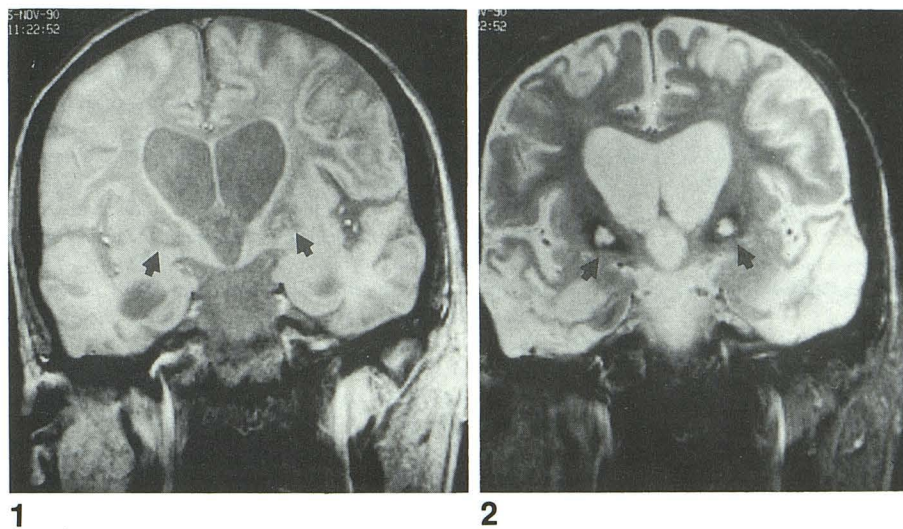


Fig. 1. Coronal proton density image, 2500/22/2 (TR/TE/excitations). Arrows designate areas of abnormal signal bilaterally in the globus pallidus with shorter T1 than CSF, probably representing methemoglobin.

Fig. 2. Coronal T2-weighted image, 2500/90/2. Arrows designate areas of abnormal signal bilaterally in the globus pallidus with longer T2 characteristics than CSF, probably representing methemoglobin, and surrounded by a rim of decreased signal, felt to represent hemosiderin.

signal on T2-weighted images in the anterior thalamus (7).

These imaging observations have correlated with known neuropathology. CO is among a group of toxins, also including cyanide and manganese, that are typically responsible for a selective necrosis of the globus pallidus and a clinical picture, as this case exemplifies, of a parkinsonian syndrome (4, 5). Pathologic changes secondary to CO poisoning have been classified by Lapresle and Fardeau (6). The following are some of their findings: 1) lesions of the globus pallidus involving necrosis; 2) lesions of the white matter involving necrosis or demyelination; 3) cerebral cortex lesions consisting of spongy changes, intense capillary proliferation, degeneration, and reduction of neurons; and 4) lesions of the cerebellum involving necrosis or demyelination.

This case demonstrated previously reported observations that correlate well with known pathology. Additionally, this case revealed areas of abnormal signal symmetrically in the globus pallidus, not of CSF equivalent signal as would be expected for an area of infarction, but rather with areas that have shorter T1 and longer T2 characteristics than CSF. This probably represents methemoglobin mixed with CSF, supporting the idea that hemorrhagic necrosis is responsible for the globus pallidus lesions. The presence of a small rim of decreased signal surrounding the area of increased signal on the T2-weighted images probably represents hemosiderin, localized to this region as a result of a prior focal hemorrhage and hemorrhagic necrosis.

According to Lapresle and Fardeau (6), the cause of the lesions secondary to CO poisoning are difficult to explain on the basis of hypoxia as the sole insult. In their anatomical study, they

noted numerous diapedetic hemorrhages with hemosiderin-laden macrophages in the necrotic regions of the globus pallidus in cases of long survival. Furthermore, two cases of CO poisoning have been reported that involved the transient disappearance of the globus pallidus lesions on follow-up CT, and the authors suggest that this may support the theory that the necrotic damage may be of vascular origin following brain edema (12). They came to this conclusion because their observations on CT resembled the "fogging effect" previously reported for vascular lesions (13).

The prognosis for patients with CO poisoning has generally been considered to be better correlated with the severity of the white matter changes (2, 9). At present, MR is considered superior to CT at observing the changes involving white matter, including those secondary to CO poisoning (7-11). Two cases with long-term follow-up have been reported that are similar to this reported case. In both, patients were reevaluated with MR imaging 1 year after the initial CO poisoning. They presented with pyramidal syndromes, movement disorders, hypertonia in all extremities, and mental deterioration. MR imaging demonstrated increased intensity in the white matter. The authors suggested that these abnormalities were secondary to the inflammatory process that accompanies demyelination, and that this was an active process (10).

With the use of MR imaging, this case demonstrates the previously reported observations that occur with CO poisoning. In doing so, it helps corroborate the idea that prognosis is related to the extent of the white matter disease. However, this case also helps substantiate that lesions secondary to CO poisoning may not be easily ex-

plained by hypoxia alone, and that the damage may be of vascular origin.

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