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Carbon Monoxide Poisoning: Asymmetric and Unilateral Changes on CT

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Carbon monoxide intoxication is a known cause of damage to the central nervous system. The distribution of pathologic changes has been described [1, 2]. The characteristic CT changes have been described as symmetric lucencies in the globus pallidus and centrum semiovale [3]. We recently encountered a case of proven carbon monoxide intoxication in which the pallidal lesion was unilateral and the white matter involvement showed marked asymmetry.

Case Report

A 41-year-old man was brought to the emergency department severely obtunded after a 6-hr exposure to automobile exhaust fumes in a closed garage. Initial examination revealed blood pressure of 220/128, pulse 150, respirations 40, and temperature 103°. There was flaccid paresis of all but his right arm, which moved only in response to pain. There were bilateral splinter hemorrhages in the retinae. Pupils were 3 mm, equal, and reactive. There was mild impairment of both corneal and caloric responses on the left. Plantar responses were equivocal and deep tendon reflexes were normal.

The patient's initial arterial blood gas examination showed a pH of 7.36, PO₂ of 51.8 mm Hg, and PCO₂ of 22.9 mm Hg. Serum bicarbonate was 12 mg/l. Glucose was 379 mg/dl. A toxic screen was positive for alcohol (0.054 mg/dl) and nicotine. Hydrocarbons were absent. The hematocrit was 56%. CT of the head was done emergently (Fig. 1). There was a low-density lesion in the right globus pallidus interpreted as most likely an infarction. After approximately 5 hr in a pollution-free environment, carboxyhemoglobin (COHb) level was 7.7%. The patient was intubated and placed on 100% oxygen. Arterial blood gas improved to pH 7.36, PO₂ to 196.8 mm Hg, and PCO₂ to 29.4 mm Hg. Decadron was given intravenously. Repeat COHb level after 3 hr was less than 0.1%. Blood cultures were negative. By the third day, the patient was extubated. His left side was flaccid but some activity returned to the right leg. The patient could follow commands and interact but was amnesic for the episode. An EEG showed slowing over the right hemisphere. A repeat CT scan (Fig. 2) 3 days after admission showed diffuse white matter damage in the right cerebral hemisphere with relative cortical sparing. There was also lucency of the centrum semiovale on the left. The right globus pallidus remained lucent, and the left globus pallidus was normal. This constellation of changes confirmed the diagnosis of CO poisoning. A follow-up scan (Fig. 3) done 70 days after the episode

showed a well-developed right hemisphere leukoencephalopathy with minimal left-sided changes. There was no further significant clinical change after day 3.

Discussion

The combination of bilateral hypodense lesions in the globus pallidus and paraventricular white matter are classical for carbon monoxide poisoning, although bilateral pallidal lesions alone have been described in trauma, hydrogen sulfide poisoning, hypoglycemia, cyanide poisoning, barbiturate intoxication, and necrotizing encephalomyelopathy.

Basal ganglia lesions also have been described in Leigh disease (subacute necrotizing encephalomyelopathy) as a degenerative disorder that has no known cause and occurs principally in infants. Basal ganglia lesions are found throughout the CNS; that is, in the spinal cord, brainstem, basal ganglia, dentate nucleus, and cerebellum [4]. Additional rare causes of basal ganglia lesions include hepatolenticular degeneration, Huntington disease, Kearns-Sayre syndrome, acute neurologic dysfunction with striatal necrosis, and methanol poisoning [4].

CO poisoning has three mechanisms: carboxyhemoglobin, which cannot bind oxygen, causes hypoxia; the oxyhemoglobin dissociation curve is shifted to the left, which decreases oxygen release to the tissues, and there is a direct toxic effect on mitochondria via CO binding to cytochrome *a*₃. Levels of carboxyhemoglobin in the blood are useful only as a clinical guide to diagnosis [5]. Permanent neurologic sequelae have been reported with levels of less than 10%. The elimination of carbon monoxide, the CSF, EEG, and clinical findings, the protective effect of ethanol in carbon monoxide poisoning, and the treatment have been previously described [5–10].

Pathologically, the most common lesion is bilateral necrosis of the globus pallidus. Injury to Ammon's horns, white matter lesions, and cortical damage are also found [1]. CT has demonstrated symmetric low-density lesions of the basal ganglia. Diffuse white matter low-density areas evolve from

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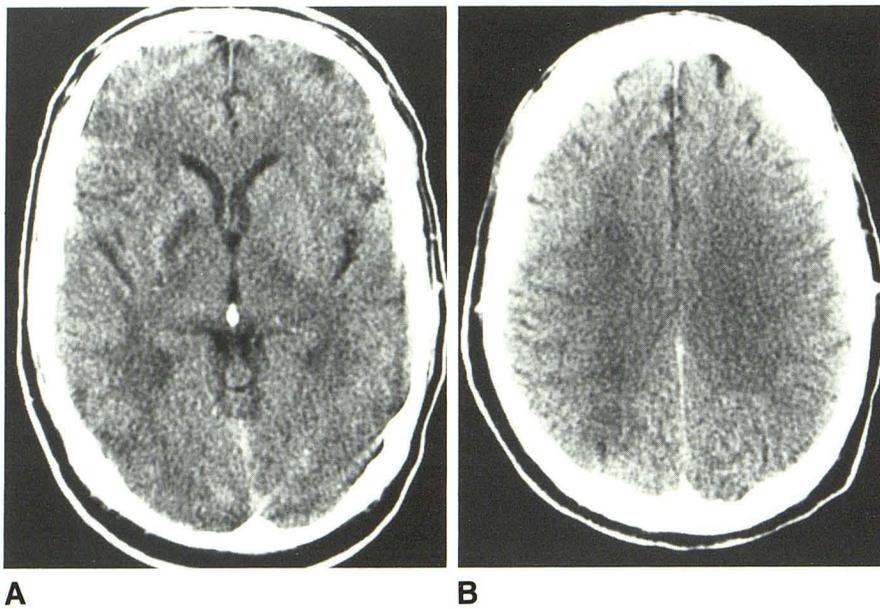


Fig. 1.—A and B, Initial CT scans (6 hr) after beginning of exposure reveal well-defined lucent zone in right globus pallidus.

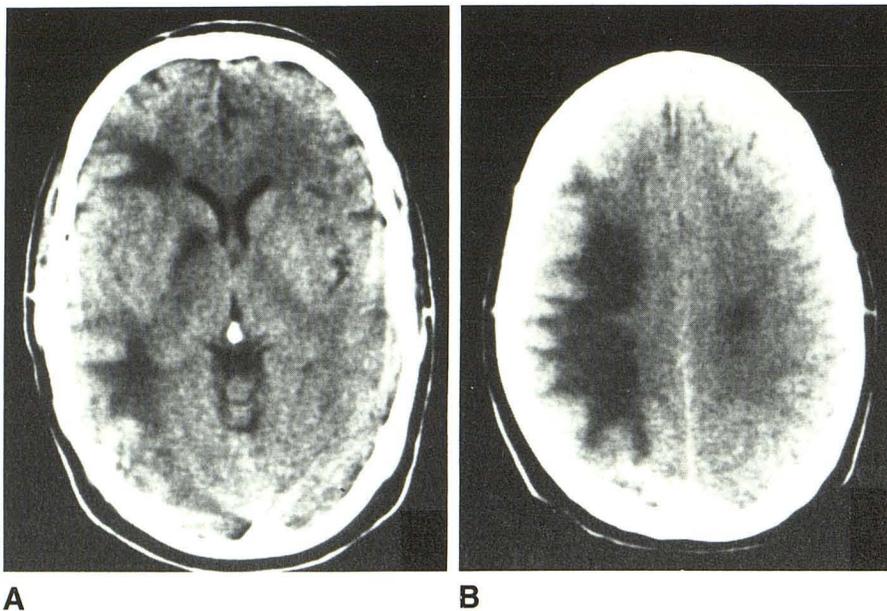


Fig. 2.—A and B, On CT scans done 3 days after Fig. 1, right pallidal lesion is more evident and there is extensive leukopathic change on the right with a much less prominent change on the left.

early, faint, poorly defined areas, to better-defined lucent areas, sparing the cortex, in the late stages.

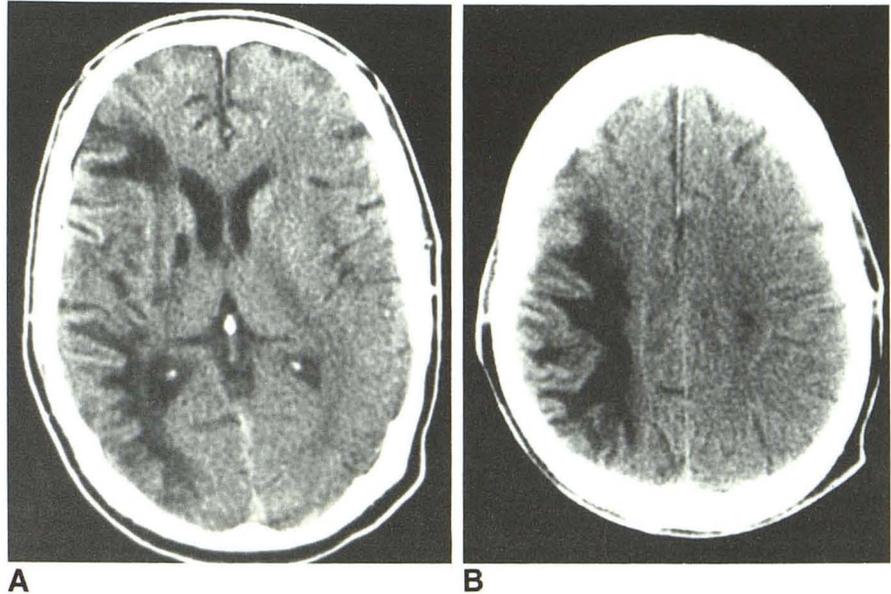
Experimentally, neither the intensity nor the duration of exposure has a strong relationship to the severity of white matter damage [11]. The lesions of the pallidum are predominantly in the anterior two-thirds and probably result from the sensitivity of this functional end-arterial area to hypoxemia-hypotension [1]. Extensive white matter lesions are most often seen with acidosis, hypoxemia, and hypotension [12].

A method to determine the initial carboxyhemoglobin level and its elimination rate exists, but requires that the exact concentration of CO inhaled as well as length of exposure be known. Since this information is rarely available in clinical

situations, there is no way to estimate accurately the original COHb level in patients. The half-life ranges from about 5 hr on room air to as short as 40 min on pure oxygen; and multiple factors affect elimination. Therefore, extrapolation from the patient's first COHb level drawn to his level when found is not possible.

The association of unilateral pallidal and centrum semiovale lesions is unusual, regardless of cause. Unilateral lesions of the global pallidus or lentiform nucleus have been described after head trauma [13], postencephalitic postmeningitic lesions [14], basal ganglia tumors, stroke, and subacute necrotizing encephalomyelopathy (Leigh disease) [4]. While localization and unilaterality are easily understood in stroke and

Fig. 3.—A and B, On CT scans done 70 days after Fig. 1, right hemisphere changes of CSF-density white matter lesions are more defined. Compensatory enlargement of regional CSF spaces and ventricles is evident. Left hemisphere shows a minimal focus of leukopathic change.



tumor, no generally accepted explanation or postulated mechanism has been put forth for the other causes. Maki et al. [13] suggest a vascular mechanism in childhood trauma whereas some researchers have postulated a "selective vulnerability of the brain" as an explanation for the predilection for the basal ganglia to be affected by toxic and metabolic insults [4]. Embolic infarcts might mimic this pattern but would be expected to involve cortex as well, and they would not be expected to be evident as early on CT. Nevertheless, the predominant unilaterality of the lesions in this case might well reflect an ischemic component, possibly as the result of preexisting disease or vascular compression during the toxic episode. The distribution of the CT lesions does not reflect the bilaterality of early clinical findings but does correlate well with the final outcome. The white matter lesions, but not the pallidal lesions, are known to have predictive value [1].

At presentation, the clinical differential included infarction, hemorrhage, drug ingestion, sepsis, and carbon monoxide poisoning. The drug screen was negative, and the level of CO was only 7.7%. An erroneous CT diagnosis of infarct was made on the initial CT scan. Subsequent evolution of CT changes included bilateral although markedly asymmetric deep white matter lesions, thus confirming the diagnosis on CT of CO poisoning.

This case is significant in that it demonstrates unilaterality of pallidal injury and marked asymmetry of white matter lesions. These changes are known to occur pathologically but have not been reported on CT. Furthermore, there was a delay in the appearance of the white matter changes, which, with the unilateral nature of the pallidal lesion, lead to an erroneous diagnosis.

Awareness of the potential for unilateral pallidal lesions in CO poisoning and the delayed appearance of white matter

changes should lead to more accurate CT interpretation and may have significant forensic implications.

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