CT of the Brain in Acute Carbon Monoxide Intoxication: Characteristic Features and Prognosis

Takashi Miura¹ Masanori Mitomo Ryuji Kawai Kohshi Harada Of 60 patients with acute carbon monoxide (CO) intoxication caused by accidental or intentional inhalation of natural gas, smoke inhalation, or inhalation of automobile exhaust, 23 had characteristic computed tomographic findings. The most common finding, seen in 21 patients, was symmetric and diffuse low density in the cerebral white matter, which was more advanced in the centrum semiovale and varied in degree from slight to severe. In 18 patients there was a symmetric, bilateral, round low-density lesion in the globus pallidus. The size of this lesion varied from small (limited to the globus pallidus) to large (extension to the internal capsule). Of the 23 patients, six made a good recovery, four recovered with some disabilities, eight remained in a vegetative state, and five died. The prognosis depended on the severity of the cerebral white-matter changes and not on the size of the low-density lesion of the globus pallidus.

The diagnosis of acute carbon monoxide (CO) intoxication is usually suggested by the circumstances under which the patient is found and is confirmed by the subsequent demonstration of carboxyhemoglobin in the blood. Consequently, it is easy to make a diagnosis of acute CO intoxication clinically, but it is not easy to determine the prognosis [1]. Recent improvements in computed tomography (CT) have made it possible to demonstrate the morphologic changes in the brain during life [2–5]. The CT features of acute CO intoxication and the accuracy of CT in determining the prognosis of the patient are the topics of this article.

Materials and Methods

Between May 1976 and April 1982, 60 patients with acute CO intoxication were examined by CT in the Department of Radiology at the Osaka University Hospital. All 60 patients were comatose when they were found. Abnormal CT scans were seen in 23 patients aged 3–68 years old; seven were males and 16 were females. In 12 patients, the first CT scan was obtained within 24 hr after the patient was found, in six patients within 48 hr, and in five patients after 48 hr (from 4 to 15 days). Follow-up CT scans were obtained in 15 patients. Intravenous contrast medium was injected in seven cases. All the patients were studied with either an EMI 1000, EMI 1010, or GE CT/T 8800 scanner. The CO intoxication was caused by suicidal inhalation of natural gas in 16 patients, accidental inhalation of natural gas in two, inhalation of smoke from a house fire in two, inhalation of smoke from a coal fire in two, and inhalation of automobile exhaust in one. CO intoxication is the major cause of intoxication in the Japanese home, because man-made gases, which include carbon monoxide, are an important energy source in Japan [1].

Results

Abnormal CT findings were found in 23 of the 60 patients; all 23 were comatose at the time of the CT examination. Of the other 37 patients, 14 were normal or only mildly confused. Of the 37 patients with normal CT scans, 35 were cured by hyperbaric oxygen therapy and treatment of the attendant acidosis, one died of sepsis, and one had a cardiac arrest.

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AJNR 6:739-742, September/October 1985 0195-6108/85/0605-0739 © American Roentgen Ray Society The most common abnormal finding was symmetrically diffuse low density of the cerebral white matter. This was found in 21 of the 23 cases (table 1). The severity of the low-density lesions varied from mild to severe (figs. 1 and 2). Three patients had low densities in the cortical gray matter. In 10 of the 21 cases the decrease in the white-matter density was severe. Three of the 10 patients died and six were in a vegetative state when transferred to another hospital; one patient recovered with severe disabilities. In 11 of the 21 cases the white-matter changes were minor; of these 11 cases, two remained in a vegetative state, three recovered with moderate disabilities, and six recovered completely.

A second characteristic feature in 18 of 23 patients was the presence of symmetric, round, low-density lesions in the globus pallidus. These lesions varied from small (limited to the globus pallidus) to large (extending to the internal capsule) (figs. 3 and 4). Of the 11 patients with large globus lesions, two died, four remained in a vegetative state, three recovered with slight disabilities, and two recovered completely (table 1). In the other seven cases the changes were small; three of

TABLE 1: Relations of White-Matter Changes and Size of Globus Pallidus Lesions to Outcome

CT Finding	No. of Patients $(n = 23)$				
	Total	Full Recovery	Disability	Vegetative	Death
White-matter cha	nges:				
Severe	10	0	1	6	3
Mild	11	6	3	2	0
Total	21	6	4	8	3
Globus pallidus le	sions:				
Large	11	2	3	4	2
Small	7	3	1	3	0
Total	18	5	4	7	2

these patients remained in a vegetative state, one recovered with slight disabilities, and three recovered completely. In the other seven patients the changes were small: three remained in a vegetative state, one recovered with slight disabilities, and three recovered completely. In two patients, a diffuse low density of the whole brain was found (fig. 5). These patients had cardiorespiratory arrest at the time of the accident and died soon after CT.

Follow-up CT scans were obtained in 15 patients 1–10 days after the first study. Resolution of the white-matter changes only occurred in three patients (two patients initially with mild changes and one with severe changes), and their clinical symptoms also improved. In the other cases, no change could be observed clinically or on CT. The low densities of the globus pallidus improved somewhat in one patient, increased in size in five, and further decreased in one but improved later. These seven patients improved clinically. In the seven patients who had contrast-enhanced scans, subtle enhancement was seen in the globus pallidus in one patient (fig. 6).

Discussion

Brain lesions resulting from CO intoxication have been the subject of many classic pathologic studies [6–14]. Lapresle and Fardeau [7] reviewed 22 patients with CO intoxication and classified them into four main types pathologically: (1) 16 had globus pallidus lesions, consisting of varying degrees of necrosis depending on the duration of survival; (2) 16 had white-matter lesions containing many scattered or focal necrotic areas or confluent areas of demyelination; (3) 12 had cerebral cortex lesions, consisting of spongy changes, intense capillary proliferation, degeneration, and reduction of neurons; and (4) 10 had hippocampal lesions, consisting of clearly delimited coagulation necrosis. We found that the frequency of lesions in the cerebral white matter and in the globus

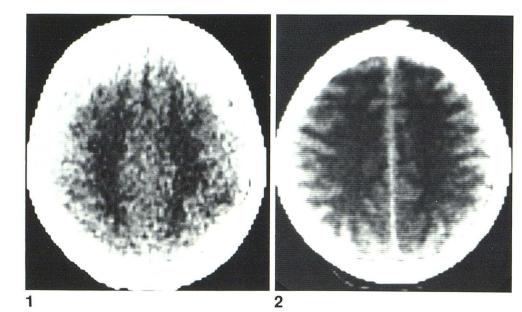


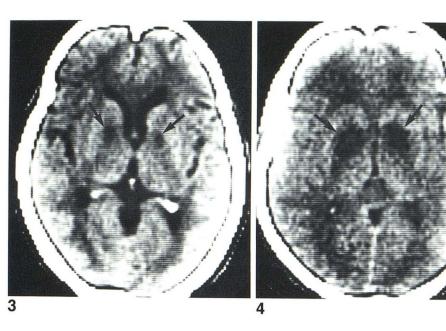
Fig. 1.—Noncontrast CT scan within 24 hr after discovery of patient. Mild, bilateral, symmetric low densities in white matter.

Fig. 2.—Noncontrast CT scan 24 hr after discovery of patient. Severe, bilateral, symmetric low densities in white matter. Cortical gray matter is clearly distinguished from white matter.

Fig. 3.—Noncontrast CT scan 1 day after discovery of patient. Bilateral, symmetric, small low densities in globus pallidus (*arrows*) and low density in white matter.

Fig. 4.—Contrast-enhanced CT scan within 24 hr after discovery. Bilateral, symmetric, large low densities in globus pallidal area (*arrows*) and low densities in white matter.

Fig. 5.—Noncontrast CT scan 8 hr after discovery of patient. Diffuse low density of brain with loss of gray/whitematter differentiation. Compressed lateral ventricles.



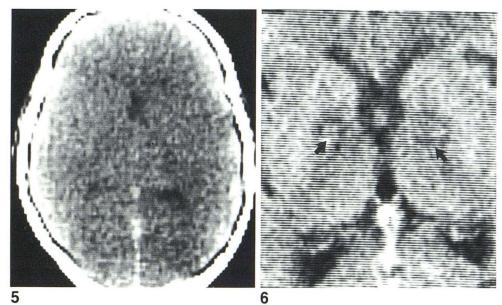


Fig. 6.—Contrast-enhanced CT scan 24 days after discovery of patient. Slight enhancement in center of globus pallidus (arrows).

pallidus as found by CT was similar to that in the pathologic literature. Radiologic demonstration of lesions of the hippocampal area and cortex, however, was less common, possibly because it is difficult to detect lesions near bony structures with low-resolution CT.

Preziosi et al. [6] described acute CO intoxication in dogs monitored physiologically. The circulatory changes, including right-sided heart failure and the consequent elevation of the cerebral venous pressure, were believed to have contributed to the development of the cerebral lesions. According to another experimental study of acute CO intoxication in rhesus monkeys, the extent of the cerebral white-matter damage correlated with the degree of metabolic acidosis and arterial hypotension sustained during CO exposure [15].

The pathogenesis of the globus pallidus lesion remains uncertain. However, in comatose patients who die from acute CO intoxication, the pallidus may appear ischemic, and this ischemia possibly precedes irreparable change in some patients [16]. After a short survival of up to 48 hr, macroscopic inspection of the brain shows an early necrosis of the globus pallidus, which histologically already can be seen after 3 hr [16]. In global central nervous system hypoperfusion, the lesion commonly affects the anterior two-thirds of the globus pallidus and may extend into the adjacent internal capsule, thus suggesting a regional perfusion impairment. With a lowresolution CT scanner, it was difficult to document the extent of early small infarctions [17], and small pallidal lesions could not be seen on early CT scans. Symptoms related to pallidal involvement may immediately follow the acute intoxication, but most often they appear after recovery from coma or are connected with delayed relapse after a latency period of several days to months.

In conclusion, our results suggest that recovery from CO intoxication is possible and that the severity of the whitematter changes appears to provide the best clue to prognosis. The size of the lesions affecting the globus pallidus had no prognostic significance, possibly because of lack of CT sensitivity for small lesions in this region.

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