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Evaluation of Outcome after Acute Carbon Monoxide Poisoning by Brain CT

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Of 129 patients with carbon monoxide (CO) poisoning, 62(48.0%) had characteristic computed tomographic (CT) findings. The most common finding, seen in 42 patients, was low-density in the cerebral white matter, and the second characteristic feature, seen in 33 patients, was low-density in both globus pallidi. Abnormal CT findings tended to increase in accordance with the duration of unconsciousness during acute CO poisoning, but such findings occurred even when the mental state was clear during acute illness. The prognosis of acute CO poisoning depended on low-density lesions of the cerebral white matter rather than those of the globus pallidus. There also seemed to be a significant correlation between the cerebral white matter changes in the initial CT scan and the development of delayed neurologic suquelae after acute CO poisoning, particularly in middle age or older patients, but no correlation between the CT findings and the clinical outcome of delayed neurologic sequelae.

Key Words: *Carbon monoxide poisoning, Delayed neurologic sequelae, Computed tomographic brain scan, Outcome*

INTRODUCTION

In Korea, carbon monoxide (CO) poisoning is still one of the most important conditions causing brain damage as a result of cellular oxygen lack, although the incidence of CO poisoning is decreasing annually (Hwang an Choi, 1990).

It is widely known that CO has the toxic effects of tissue hypoxia and produces acute neurologic deficits, but severe neurologic reactions may be delayed for days or weeks after anoxic exposure (Shillito et al, 1936; Meigs and Hughes, 1952; Choi, 1983).

The prediction of outcome after acute CO poisoning is difficult in most cases because of variations in age, previous state of health, duration and severity of exposure, individual susceptibility, and manner of treatment.

The initial laboratory findings do not provide any prognostic clues. Recent improvements in neuroimaging tool have made it possible to demonstrate the morphologic changes in the brain during life. Attempts at further predicting the clinical outcome of CO poisoning by means of brain CT scans have been tried (Nardizzi, 1979; Kim et al, 1980; Sawada et al, 1980; Kono et al, 1983; Kobayashi et al, 1984; Miura et al, 1984; Suh et al, 1986), although magnetic resonance (MR) imaging has been shown to be superior in sensitivity to CT scanning in studies of CO poisoning recently (Chang et al, 1986; Horowitz et al, 1987; Vieregge et al, 1989; Chang et al, 1992).

Using brain CT scans, we aimed to determine whether CT findings predicted the outcome of acute CO poisoning or correlated with the clinical course of delayed neurologic sequelae.

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PATIENTS ADN METHODS

Between 1983 and 1991, 360 patients with CO poisoning were admitted to Severance Hospital, Yonsei University Medical Center, Seoul. Of the 360 admitted patients, 129 were examined by brain CT scan. There were 56 men and 73 women. Ages varied from 1 to 92 years (mean, 48.2 years).

All except three of the 360 admitted patients had suffered a period of unconsciousness during acute CO poisoning.

Of the 360 patients, delayed neurologic sequelae occurred in 37, in all of whom brain CT scans were obtained. There were 10 men and 27 women. Ages varied from 37 to 78 years (mean, 60.4 years). The clear period preceding the onset of symptoms varied from 8 to 40 days (mean, 21.6 days), but was between 2 weeks and one month in the majority of cases. Of those 37 patients, 28 completed the one to two-year follow-up study, but 9 were lost to follow up.

129 patients were studied with either a Delta Scanner F.S.50 (Ohio Nuclear Inc) or a GE CT/T 9800 scanner. The first CT scan was obtained within 3 days of acute poisoning in 123 patients, and within 14 days after the onset of delayed neurologic sequelae in 6 patients. Follow-CT scans were obtained in 25 patients with delayed neurologic sequelae, and in 11 patients with prolonged coma after CO poisoning. In delayed neurologic sequelae, follow-up CT scans were done 3 to 8 months after acute CO poisoning in 23 patients, when the patients had recovered clinically, and 18 to 26 months later in remaining two patients.

We modified the Glasgow Outcome Scale (Jenett and Bond, 1975) and classified into five grades in acute cases: fulll recovery, delayed neurologic sequelae, disability, vegetative state, and death, and into four grades in delayed neurologic sequelae: full recovery, disability, vegetative state, and death.

RESULTS

Abnormal CT findings related with CO poisoning were found in 62 of the 129 patients (48%). The most common abnormal finding was symmetrically diffuse low-density in the cerebral white matter, particularly in the frontal areas. This was found in 42 of the 129 patients (32.6%). The second characteristic feature seen in 33 of 129 patients (25.6%) was the presence of low-density in the globus pallidus bilaterally. Of the 33 patients with pallidal low-density, only one had contrast enhancement in the basal ganglia on CT scan 2 days after acute poisoning (Fig. 1). In addition, 13 had simultaneous low-density lesions of the globus pallidus and cerebral white matter on the initial CT scans. Of the remaining 67 patients, nine had cortical atrophy presumably not related to CO poisoning on CT scans, and 58 had normal CT finding.

All except three had suffered a period of unconsciousness during acute CO poisoning, which was usually severe. They were in deep coma when found but usually awakened within 24 hours. The abnormal

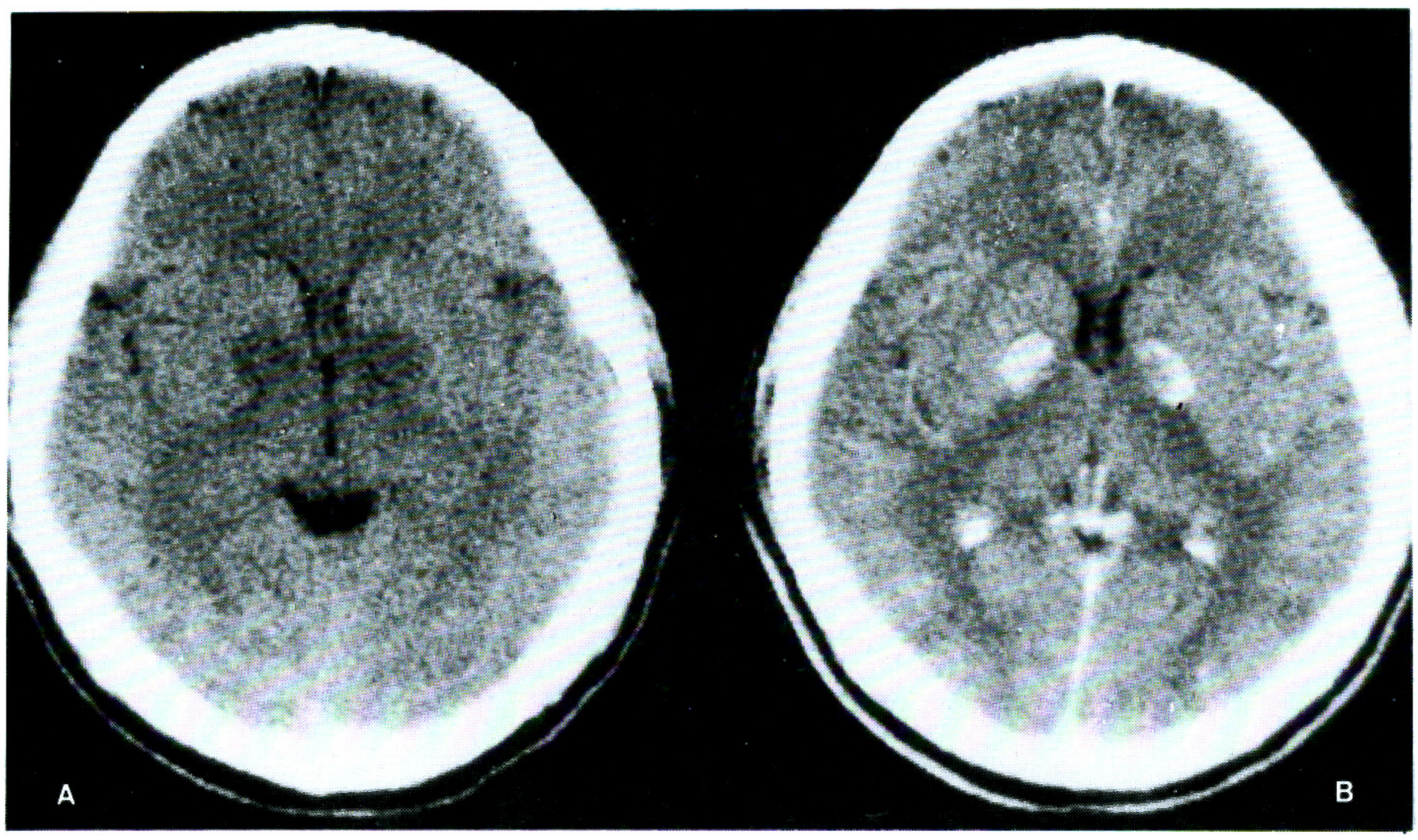

Fig. 1. A. Pre-contrast CT scan 2 days after anoxia showed low-density in both globus pallidi. B. Post-contrast CT scan clearly demonstrated contrast enhancement in both globus pallidi.

CT findings tended to increase in accordance with the duration of unconsiousness during the acute episode, but such findings occurred even when the mental state was clear during acute illness (Table 1).

Of the 62 patients with abnormal CT findings, 17 recovered completely, but the remaining patients had sequelae or died. Of the 42 patients with white matter changes on CT during acute stage, 20 had delayed neurologic sequelae, but only six recovered completely, five of which were below 40 years of age. Of the 33

Table 1. Duration of unconsciousness during acute CO poisoning and initial CT findings.

Duration of unconsciousness, Hours	No. of abnormal CT	No. of normal CT
- 0*	2	1
1-12	5	27
13-24	23	23
25-48	4	2
49-72	6	3
73-	22	11
Total	62	67

*All patients who suffered no unconsciousness were treated in the emergency room or as outpatients and only 3 with delayed sequelae were admitted.

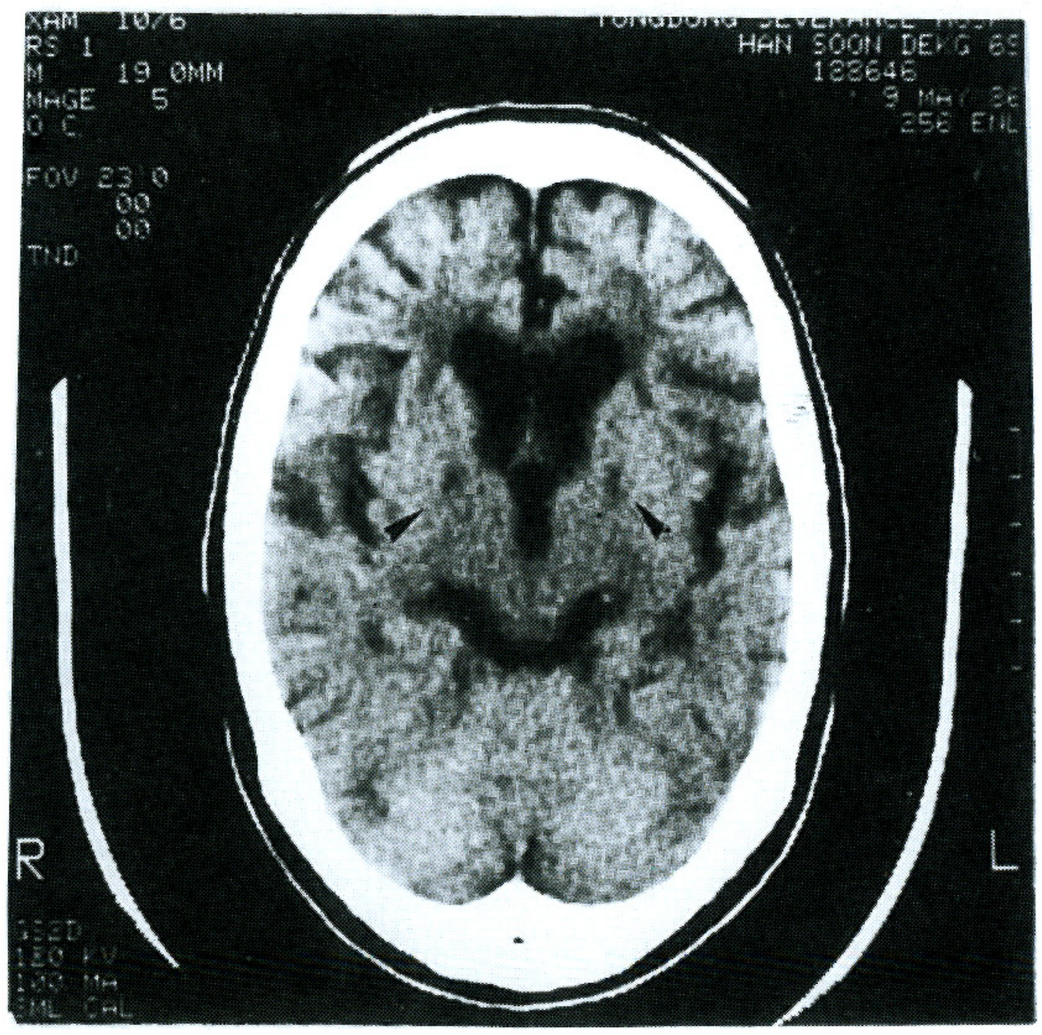

Fig. 2. CT scan 26 months after anoxia still showed low-density in both globus pallidi with cortical atrophy.

Table 2. Correlation between the initial CT findings and the outcome of acute CO poisoning.

Initial CT findings	No. of Patients					
	Full recovery	Delayed sequelae	Disability	Vegetative	Death	Total
White matter changes	5*	17	0	6	1	29
Globus pallidus lesions	11	3	1	4	1	20
Lesions of globus pallidus & white matter	1	3	1	6	2	13
Normal & others	46	14	2	5	0	67
Total	63	37	4	21	4	129

*All except one were below 40 years of age

Table 3. Correlation between the initial CT findings and the outcome of delayed neurologic sequelae after CO poisoning.

Initial CT findings	No. of Patients				
	Full recovery	Disability	Vegetative	Death	Total
White matter changes	7	3	2	0	12
Globus pallidus lesions	2	0	1	0	3
Lesions of globus pallidus & white matter	1	0	0	0	1
Normal & others	6	3	0	3	12
Total	16	6	3	3	28

patients with pallidal lesions, 12 recovered completely, and 21 had sequelae or died. Of the 67 patients with either normal or non-CO-related findings, 46 recovered completely, and 21 had sequelae (Table 2).

Of the 37 patients with delayed neurologic sequelae, 28 completed the follow-up. Of the 28 follow-up patients, 22 (78.6%) recovered, 3 showed no improvement, and 3 died of infection. Of the 22 recovered patients, 5 had parkinsonism, and one had memory disturbance. Of the 13 delayed sequelae patients with white matter changes on CT, 8 recovered completely, 3 had disability, and 2 were in a vegetative state. Of the 4 patients with pallidal lesions, 3 recovered completely, and one was in a vegetative state. Of the 12 patients with normal or other CT findings, 6 recovered completely, 3 had disability, and 3 died (Table 3).

Follow-up CT scans showed no interval change in 14 of 25 patients with delayed sequelae, even up to 26 months after anoxia (Fig. 2), and 8 revealed more a aggravating pattern with cortical atrophy, in contrast

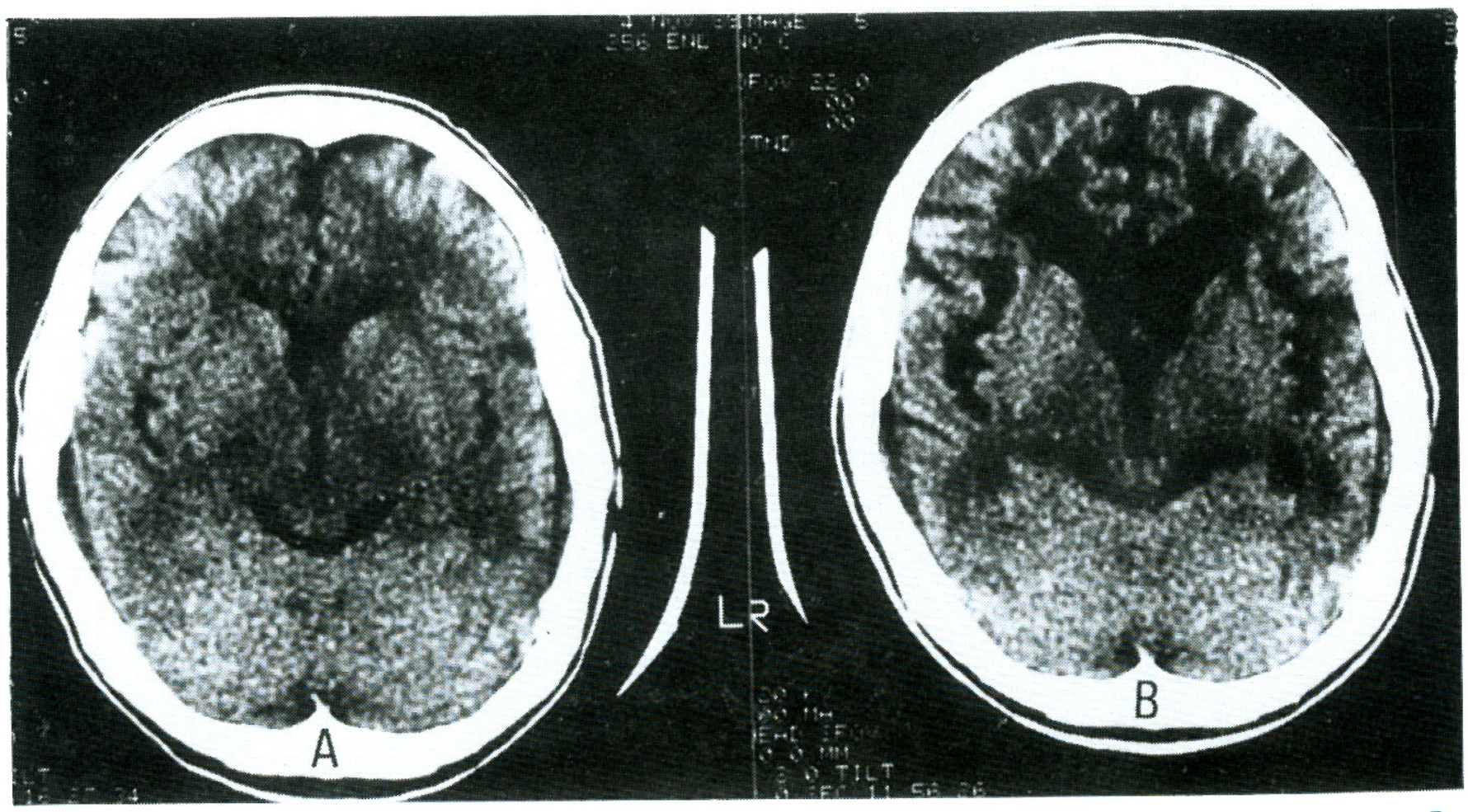

Fig. 3. A. CT scan 4 days after acute CO poisoning showed low-density in cerebral white matter. B. CT scan 7 months after anoxia revealed a more aggravating pattern with cortical atrophy.

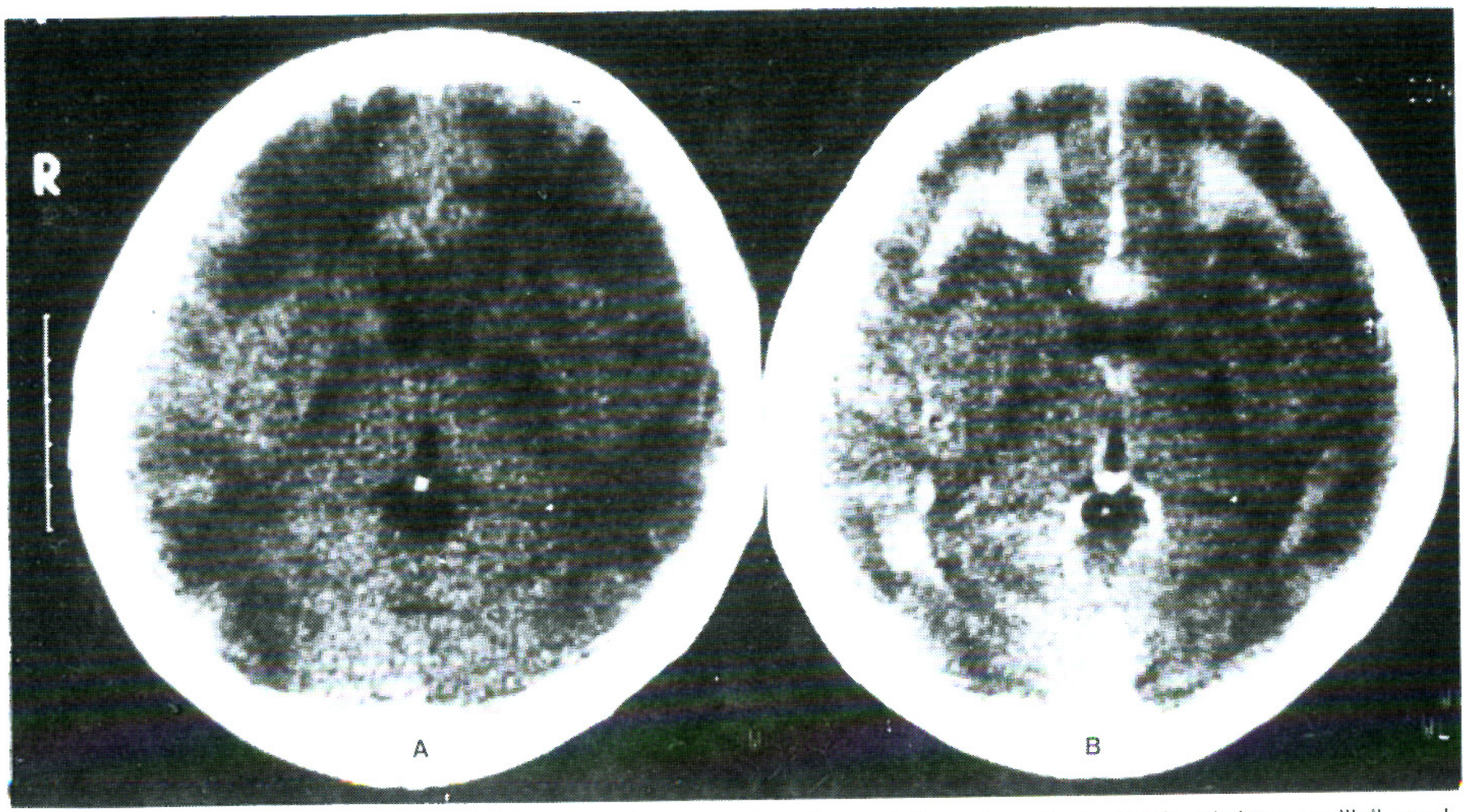

Fig. 4. A. Pre-contrast CT scan 15 days after anoxia showed low-density in both globus pallidi and cerebral white matter. B. Post-contrast CT scan clearly demonstrated contrast enhancement in the areas of subcortical white matter, particulary the frontal areas.

to the clinical improvement (Fig.3). Three patients with initial normal CT finding had low-density lesions in the cerebral white matter or globus pallidus at follow-up. Of the 11 patients with prolonged coma after anoxia, one had the subcortical white matter enhancement on follow-up CT 15 days after anoxia (Fig.4).

DISCUSSION

It is well known that the most common characteristic features of CT scans in CO poisoning are symmetrical low-density lesions in the globus pallidus and /or cerebral white matter, but the initial CT scan may fail to detect such findings.

Of the series of Miura et al (1985), abnormal CT findings were found in 23 of 60 patients (38.5%) with acute CO poisoing. This was lower than those of Choi (1983), Sawada et al (1980), and ours.

The literature contains descriptions of various CT findings in CO poisoning, but most of them were reports predicting the clinical outcome of acute CO poisoning by means of CT scan (Nardizzi, 1979; Kim et al, 1980; Sawada et al, 1980; Kono et al, 1983; Kobayashi et al, 1984, Miura et al, 1984; Vieregge et al, 1989).

Kono et al (1983) mentioned that there seemed to be a significant correlation between cortical atrophic changes on CT and the duration of unconsciousness during acute CO poisoning. Of the series reported herein, all except three of the admitted patients had suffered a period of unconsciousness during acute illness, which was usually severe. As seen in table 1, abnormal CT findings tended to increase in accordance with the duration of unconsciousness, but abnormal findings occurred even when the mental state was clear during the acute episode.

The prediction of outcome after acute CO poisoning is difficult in most cases, and there are no important contributing factors except age and the severity of anoxia (Choi, 1983). Therefore, in the past an attempt was made to provide more useful prognostic data by evaluating the changes on the brain CT scan during the acute stage (Sawada et al, 1980; Kobayashi et al, 1984; Miura et al,1985). Sawada et al (1980) mentioned that the outcome of CO poisoning correlated with the pallidal low-density of the CT scan, but Miura et al (1985) believed the prognosis of acute CO poisoning depended on the cerebral white matter lesions and not on the low-density lesions of the globus pallidus. The results reported herein were comparable to those of Miura et al (1985). In addition, there seemed to be a significant correlation between the cerebral white matter changes on aucte stage CT and the development of delayed neurologic sequelae after CO poisoning. In the series reported herein, delayed neurologic sequelae occurred within a month after acute insult in the majority of cases with the cerebral white matter changes on CTs of acute illness, particularly in middle age or older patients.

The outcome of delayed neurologic sequelae is relatively good. About one half to 3/4 of patients with delayed sequelae recovered within one year (Shillito et al, 1936; Choi, 1983). In the series reported herein, of the 16 delayed sequelae patients with abnormal findings on initial CT, 10 recovered completely, 3 had disability, and 3 showed no improvement. Of the 12 patients with normal or other findings on initial CT, 6 recovered completely, 3 had disability, and 3 died. This means there seemed to be no correlation between the abnormal CT finding and the clinical outcome of delayed neurologic sequelae after CO poisoning.

The initial CT lesions may either have disappeared, diminished, or remained unchanged at follow-up (Sawada et al, 1980; Miura et al, 1985; Vieregge et al, 1989). All the initial CT lesions reported herein did not disappear at follow-up, even up to 26 months after anoxia, moreover, some revealed more aggravating findings with cortical atrophy, in spite of the clinical improvement. This suggests that the majority of low-density on CT scans in acute CO poisoning consisted essentially of areas of necrosis rather than those of edema, as Lapresele and Fardeau (1967) demonstrated pathologically, and cortical atrophic changes did not affect the clinical outcome of delayed neurologic sequelae after CO poisoning.

The results of this clinical study show that abnormal CT findings tended to increase in accordance with the duration of unconsciousness during acute illness, and the prognosis of acute CO poisoning depended on the cerebral white matter changes rather than the pallidal lesions. There seemed to be a significant correlation between the cerebral white matter changes on initial CT scan and the development of delayed neurologic sequelae, but no correlation between the CT findings and the clinical outcome of delayed neurologic sequelae.

Although it is a weak point that CT scan may fail to detect low-density lesions of the globus pallidus and cerebral white matter in approximately one half of the patients with CO poisoning, CT scans can be used for evaluating the outcome of acute CO poisoning. But concomitant study using MRI is needed, because MRI is more sensitive in the detection of the pallidal and white matter lesions of CO poisoning compared with CT.

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