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Magnetic Resonance (MR) Imaging in Delayed Encephalopathy of Acute Carbon Monoxide Poisoning*

— Comparison with CT —

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〈국문초록〉

急性一酸化炭素中毒後遺症에서의 磁氣共鳴影像*
— 電算化斷層撮影과의 비교 —

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急性 一酸化炭素中毒後遺症을 보이는 11例에서 磁氣共鳴影像(MRI)과 電算化斷層撮影(CT)을 시행하여 각각 그 所見을 관찰 분석하고, 診斷的 價値를 비교 검토하였다.

MRI는 0.15 Tesla 常電導型을 이용하여 部分飽和回復, T2强調스핀에코 또는 反轉回復등의 다양한 펄스시퀀스로 촬영하였다. 白質의 脫髓質을 암시하는 白質의 이상소견이 MRI에서는 4例에서 관찰된 반면, CT에서는 2例에서만 관찰되었다. 白質의 이상소견은 특히 T2强調스핀에코像과 反轉回復像에서 對照度가 가장 뚜렷하였으며 部分飽和回復像과 CT像에서는 對照度가 낮았다.

淡蒼球病變을 보인 1例와 腦萎縮을 보인 3例는 MRI와 CT에서 모두 함께 관찰되었다.

白質病變의 발견율은 CT보다 MRI가 높으므로 一酸化炭素中毒後遺症으로 白質病變이 의심될 때는 MRI를 먼저 시행하는 것이 바람직하다고 생각된다.

Eleven magnetic resonance (MR) and computed tomographic (CT) imagings were performed in nine patients with mild to moderate degree of delayed neuropsychiatric symptoms following acute carbon monoxide (CO) poisoning, to evaluate the capability of MR in demonstrating any additional finding to CT. The MR images were obtained using 0.15 Tesla resistive system with various combination of three pulsle sequences, including partial saturation recovery, T2-weighted spin echo and inversion recovery.

Bilateral white matter abnormalities suggesting demyelination were demonstrated in 4 patients with MR and in only 2 patients with CT. The contrast discrimination between normal and abnormal white matter proved to be better with T2-weighted spin echo and inversion recovery than with partial saturation recovery and CT.

But necrosis of the globus pallidus (1 patient) and diffuse atrophy (3 patients) were equally demonstrated on both MR and CT.

It is suggested that MR be used as a initial imaging method in the evaluation of the delayed encephalopathy following acute CO poisoning, especially for the detection of the possible white matter lesions.

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Acute carbon monoxide (CO) poisoning produces hypoxia by displacing oxygen from hemoglobin and preventing its release from hemoglobin in tissues, often resulting in fetal event.[1]

Victims who survive acute CO poisoning may have various delayed symptoms and signs. Occasionally, an apparent recovery is followed within two days to three weeks by a sudden neurological deterioration.[2] The degree of neuropsychiatric symptoms depends upon the extent and severity of the pathologic changes in the brain.

The pathologic effects of CO poisoning are present in almost all organs of patients. However, the most important changes occur in the brain, which consist of necrosis of the globus pallidus and reticular zone of the substantia nigra, and the degeneration of the cerebral white matter.[2,3]

The diagnostic superiority of magnetic resonance (MR) over CT has already been shown for various neurological disease.[4-7] But we could not find any MR description on CO poisoning in the literatures, even though the computed tomography (CT) findings and their correlation with neuropathologic findings in CO poisoning have previously been described.[8-11]

To evaluate the capability of MR in demonstrating any additional finding to CT in delayed encephalopathy of CO poisoning, we studied MR in nine patients and compared the MR with CT results.

Subjects and Methods

Nine patients with a diagnosis of delayed encephalopathy of acute CO poisoning were studied. The diagnosis of acute CO poisoning was readily made by overwhelming circumstantial evidence. In Korea, many people have a unique, traditional home heating system using coal briquette as a fuel. CO-containing coal briquette gas can accidentally leak into the room through the cracks, resulting in acute CO poisoning. There are so many victims in the winter season. Our nine patients who survived acute CO poisoning has had mild to moderate neuropsychiatric symptoms and signs, after recovery from the initial coma state. There is no patient with grave symptoms such as vegetative or quadriplegic patients in our series.

Nine patients were aged from 20 to 68 years (5 men and 4 women). One patient (case 1) had 3 studies; the first study in the 2 weeks, the second in the 1½ month and the last in the 5 months after the accident. Four patients were studied 2 to 3 months after the accidents. In the other 4 patients the scans were obtained 7 to 48 months after the accidents.

All the MR studies were performed using a 0.15 Tesla resistive magnet developed by Korea Advanced Institute of Science & Technology, Seoul, Korea, with a aperture of 25cm head coil.

Using a two-dimensional Fourier transform method with 256 phase-encoding data lines, various combinations of following three pulse sequences were used; partial saturation recovery (spin echo TE = 30, TR = 500ms), inversion recovery (TE = 30, TI = 400, TR = 1,400ms) and T2 weighted spin echo (TE = 60, TR = 1,000ms) techniques. A total of 8-12 axial slices of about 13mm slice thickness were obtained in each pulse sequence.

In all patients the X-ray CT scans were obtained within 1 day to 1 week of the MR study. The CT scans were made with an GE CT/T 8,800 and included precontrast and postcontrast enhanced-scans. The CT scanner was operated at 120kVp with contiguous 10mm sections.

Results

The clinical, MR and CT findings were summarized in Table 1.

Four of 5 patients examined within 3 month after the accidents showed symmetrical bilateral abnormalities in the white matter of the periventricular area and centrum ovale. These white matter abnormalities are seen as bright signal intensities on T2 weighted spin echo image and dark, low signal intensities on T1 weighted inversion recovery images, representing long T2 and T1 relaxation times, retrospectively.

In one patient with 3 times follow-up studies (case 1), there were no abnormalities on the initial MR studied in the complete recovery state (lucid internal), 2 weeks after the accident. But on the second MR studied 1 week after the delayed symptoms of urinary incontinence and confusion appeared, symmetrical

Table 1. Summary of Clinical, MR and CT Findings in Delayed Encephalopathy of Acute CO Poisoning

Case No.	Age, Gender	Time from Accident to MR	Clinical Symptoms & Signs	MR	CT
1	39, Male	2 weeks	Poor memroy Poor calculation ability	Normal	Normal
		1½ months	Urinary incontinence confusion	White matter* abnormality	Normal
		5 months	Poor memory	Normal	Normal
2	58, Male	2 months	Urinary incontinence mutism	White matter* abnormality	Bilateral white matter low densities
3	68, Female	2 months	Poor orientation Poor memory	White matter* abnormality	Equivocal bilateral periventricular low densities
4	48, mlae	3 months	Personality change Poor orientation	White matter* abnormality	Bilateal white matter low densities
5	33, Female	15 months	Cogwheel rigidity Bradykinesia	Basal ganglia** abnormality	Bilateral basal ganglia lacunae
6	20, Female	2 months	Depression	Normal	Normal
7	43, Male	10 months	Poor memory	Diffuse atrophy	Diffuse atrophy
8	46, Male	7 months	Poor memory	Diffuse atrophy	Diffuse atrophy
9	40, Female	48 months	Poor memory Bradykinesia	Diffuse atrophy	Diffuse atrophy

* White matter abnormality on MR represents bilateral low signal intensities in inversion recovery images and/or bilateral high signal intensities in T2-weighted spin echo images in the white matter of deep periventricular area and centrum ovale.
** Basal ganglia abnormality on MR represents bilateral focal low signal intensities in globus pallidus on inversion recovery.

bilateral white matter abnormalities were demonstrated. The white matter abnormalities disappeared on the 3rd MR studied 5 month after the accident (Fig. 1).

In general, the T2 weighted spin-echo and inversion recovery techniques were useful in detecting the white matter abnormality itself. Although partial saturation recovery images provided excellent anatomic definition, they were not helpful in separating the lesion from normal surrounding structures.

On the CT scans, only two of 4 patients with the white matter abnormalities on MR showed diffuse periventricular white matter low densities. The other two patient showed normal to subtle low densities on CT, which is very difficult to differentiate the normal from the abnormal white matter.

Contrast discrimination between normal and abnormal white matter was more apparent on MR than were changes of attenuation on CT. Subtle low densities on CT were easily demonstrated on MR (Fig. 2).

Only one patient who was examined 15 months after the accident demonstrated bilateral symmetrical low densities in the globus pallidus, which was seen on the both CT and inversion recovery images of MR (Fig. 3).

Three patients examined 7 months or more after the accidents demonstrated dilatation of the lateral ventricles and widening of the sulci, indicating diffuse atrophy.

Discussion

The neuropathologic changes including necrosis of the basal ganglia and degeneration of the white matter in CO poisoning have been well documented in autopsy specimens and experimental animals. The le-

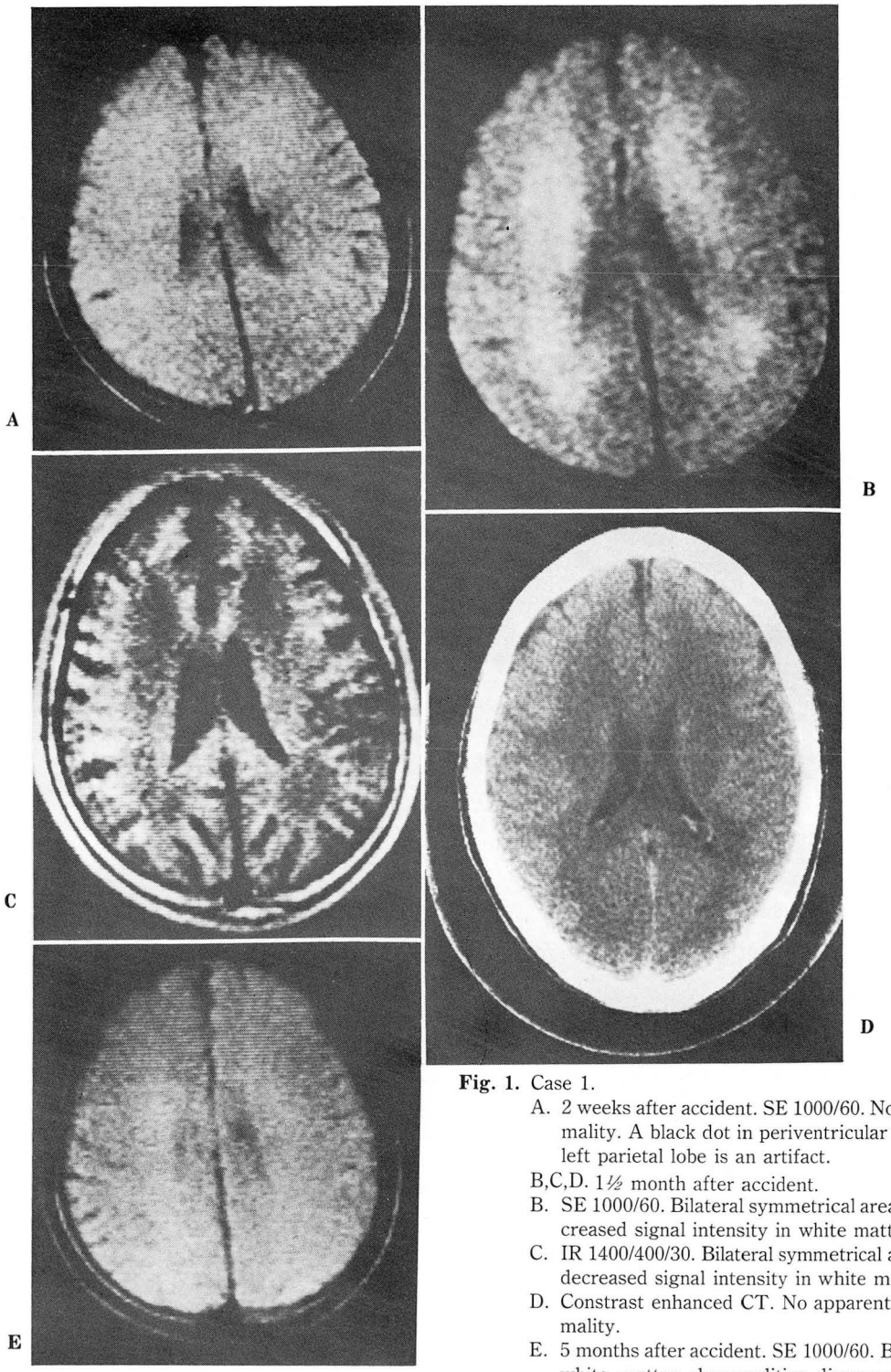

Fig. 1. Case 1.
A. 2 weeks after accident. SE 1000/60. No abnormality. A black dot in periventricular area of left parietal lobe is an artifact.
B,C,D. 1½ month after accident.
B. SE 1000/60. Bilateral symmetrical areas of increased signal intensity in white matter.
C. IR 1400/400/30. Bilateral symmetrical areas of decreased signal intensity in white matter.
D. Constrast enhanced CT. No apparent abnormality.
E. 5 months after accident. SE 1000/60. Bilateral white matter abnormalities disappeared. A black dot in left periventricular area is an artifact.

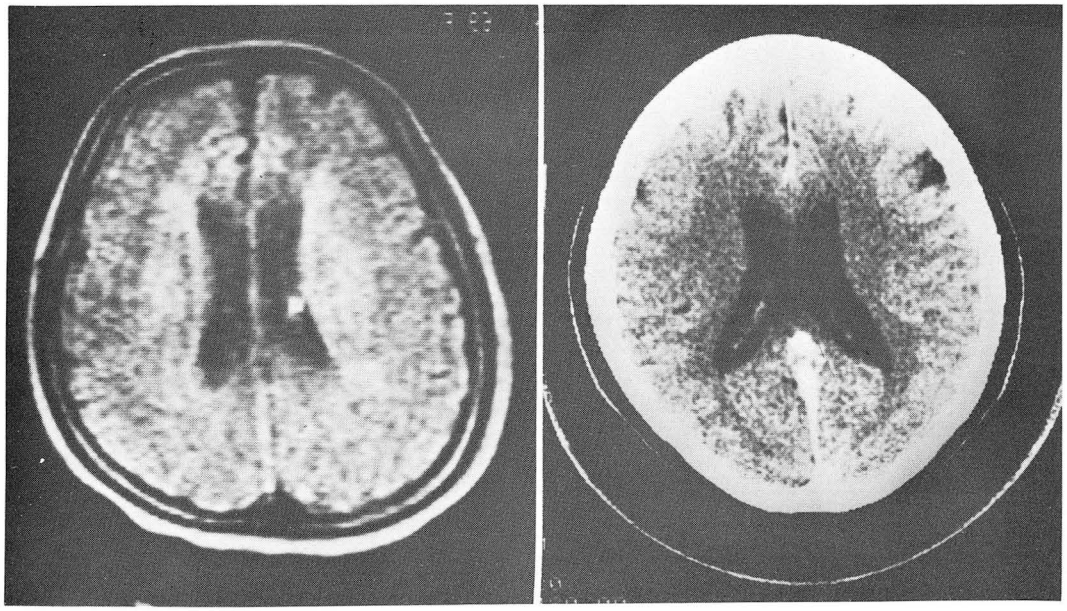

Fig. 2. Case 3. 2 months after accident.
A. SE 1000/60. Bilateral symmetrical areas of increased signal intensity in white matter. A white dot in left lateral ventricle is an artifact.
B. Contrast-enhanced CT. Equivocal white matter lesions showing bilateral periventricular low densities.

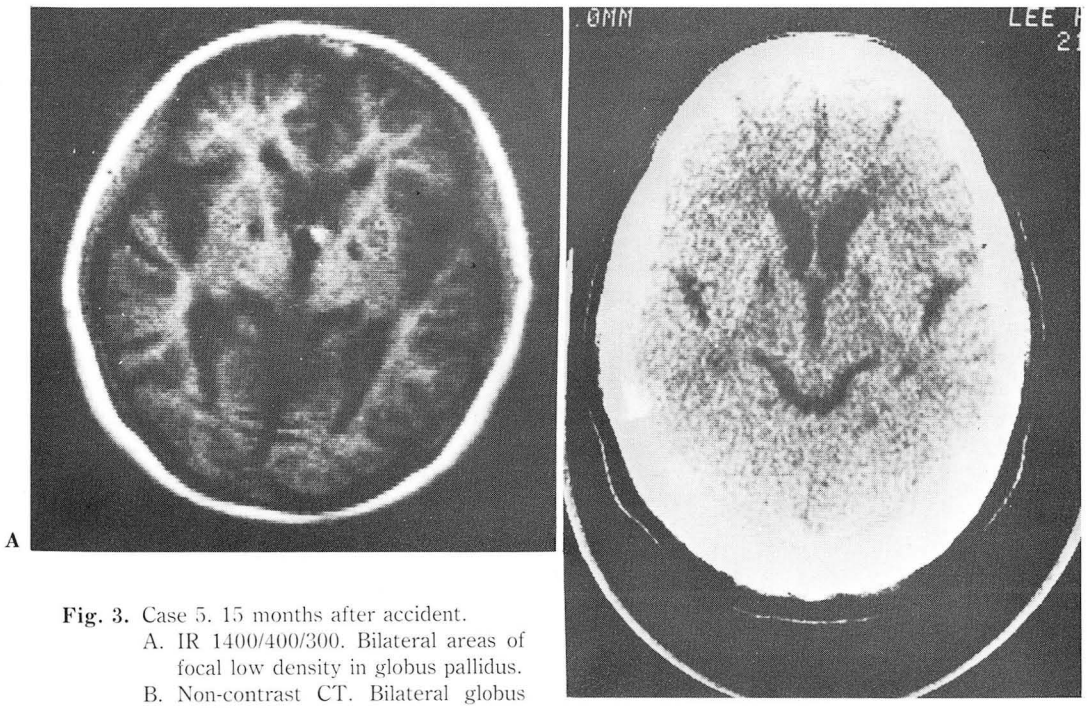

Fig. 3. Case 5. 15 months after accident.
A. IR 1400/400/300. Bilateral areas of focal low density in globus pallidus.
B. Non-contrast CT. Bilateral globus pallidus lacunae.

sions are symmetrical, and involve the globus pallidus and the deep white matter of the periventricular centrum semiovale and the corpus callosum. In acute state of CO poisoning the pathological alteration is characteristic of the hypoxic change producing acidotic oligemic edema, which progresses to incomplete necrosis causing demyelination and gliosis, or complete necrosis terminating in cystic lesions in later state.[2,3] All the abnormal MR findings in our cases were attributed to the late pathologic change; the white matter abnormalities in our 4 cases were probably due to the demyelination, but not to the acute edematous change. The demyelination may be reversible. In our case 1 with 3 times follow-up MR, the white matter abnormalities shown only on the 2nd MR (1½ month) highly suggest reversible, incomplete demyelination.

The superior sensitivity of MR to CT has been reported previously in a variety of demyelinating disease including multiple sclerosis, subcortical arteriosclerotic encephalopathy, progressive multifocal leukoencephalopathy, myelinolysis, methotrexate encephalopathy and anoxic change.[4-7] The demyelinating process usually demonstrated prolonged T1 and T2 on the MR images.[4]

In our two patients with mild clinical symptoms (case 1 & case 3) the MR could detect the white matter lesions which were not evidently demonstrated on the CT. Bilateral symmetrical myelinopathic lesions as a delayed change of CO poisoning correspond with the areas of low signal intensity in IR image (prolonged T1) and of high signal intensity in SE images (prolonged T2) in our cases.

Our cases proved that MR is more sensitive and better than CT in the contrast discrimination between the normal and abnormal white matter of delayed encephalopathy of CO poisoning.

Although the characteristic CT findings of CO poisoning including bilateral symmetric areas of low density in the basal ganglia and/or diffuse low density in the white matter has been previously reported in the many literatures,[8-11] it seems to be sometimes difficult to identify the subtle changes of the white matter degeneration on the CT scan which may show apparently normal or equivocal low density in the periventricular white matter, especially for the patients with mild neuropsychiatric symptoms and signs, like those in our patients.

Most of the patients showing prominent abnormal CT features reported in the literature had grave neurologic sequelae.[8]

With exception of the white matter lesions, the MR findings appeared to be generally correlated with the CT findings. The basal ganglia lacunae and diffuse atrophy could be equally demonstrated in both MR and CT.

The MR is suggested to be used as a initial imaging method in the evaluation of the delayed encephalopathy following acute CO poisoning, especially for the detection of the possible white matter lesions.

REFERENCES

1. Lilienthal JL: *Carbon monoxide. Pharmacol Rev 2:324-354, 1950.*
2. Lapresle J, Fardeau M: *The central nervous system and carbon monoxide poisoning. II. Anatomical study of brain lesions following intoxication with carbon monoxide (22 cases). Prog Brain Res 24:31-75, 1967.*
3. Ginsberg MD, Myers RE, McDonagh BF: *Experimental carbon monoxide encephalopathy in the primate. II. Clinical aspects, neuropathology, and physiological correlation. Arch Neurol 30:209-216, 1974.*
4. Bydder GM, Steiner RE, Young Ir et al: *Clinical NMR imaging of the brain; 140 cases. AJR 139:215-236, 1982.*
5. Brant-Zawadzki M, Davis PL, Crooks LE et al: *NMR demonstration of cerebral abnormalities: Comparison with CT. AJNR 4:117-124, 1983.*
6. Bradley WG, Waluch V, Yadley RA, Wycoff RR: *Comparison of CT and MR in 400 patients suspected disease of the brain and cervical spinal cord. Radiology 152:695-702, 1984.*
7. Zimmerman RA, Bilaniuk LT, Goldberg HI et al: *Cerebral NMR imaging: Early results with a 0.12 T Resistive Sysl;tem. AJNR 5:1-7, 1984.*
8. Kim KSK, Weinbergn PE, Suh JH, Ho SU: *Acute carbon mnonoxide poisoning: Computed tomography of the brain. AJNR 1-399-402, 1980.*
9. Kabayashi K, Isaki K, Fukutani Y, et al: *CT findings of the interval form of carbon monoxide poisoning compared*

with neuropathological findings. Eur Neurol 23:34-43, 1984.

10. Nardizzi LR: *Computerized tomographic correlate of carbon monoxide poisoning. Arch Neurol 36:38-39, 1979.*
11. Sawada Y, Sakamoto T, Nishide K et al: *Correlation of pathological findings with computed tomographic findings after acute carbon monoxide poisoning. New Engl J Med 308(21):1296, 1983.*