

# Chorea Following Acute Carbon Monoxide Poisoning

Soochul Park and Il Saing Choi

*Department of Neurology, Yonsei University College of Medicine, Seoul, Korea.*

The clinical cases of 6 patients suffering with chorea after acute carbon monoxide (CO) poisoning were reviewed. There were 2 men and 4 women, and the age at onset ranged from 11 to 60 (mean 33.0) years. All the patients except one were associated with mild delayed CO encephalopathy. The latency period between CO poisoning and the onset of chorea was 10 to 30 (mean 21.7) days. The duration of chorea after CO poisoning was 14 to 90 (mean 39.8) days. The brain CT findings were bilateral low-density lesions in the basal ganglia and/or in the white matter of the cerebral cortex, and there was no correlation between the lesion sites on the imagings and the development of chorea.

Neuroleptic agents alleviated the chorea and the patients did not relapse after neuroleptic agents were halted.

**Key Words:** Chorea, delayed encephalopathy, CO poisoning

## INTRODUCTION

Chorea refers to a condition that is almost always related to rheumatic fever, but it can result from various causes.<sup>1-3</sup> Various toxins including carbon monoxide (CO) are also able to cause chorea.

Sequelae of CO poisoning range from subtle neuropsychiatric changes to a severe encephalopathy, and extrapyramidal syndrome is a well-recognized sequela from CO poisoning.<sup>4-7</sup> The literature and textbooks do contain descriptions of involuntary movements concerned with CO poisoning, but actual case reports of chorea are rare.<sup>8-14</sup>

In his 1968 review of CO poisoning, Jellinger<sup>14</sup>

was able to collect only 8 references describing choreiform movements or choreoathetosis. Of 21,143 acutely CO intoxicated patients reported by Shillito et al,<sup>6</sup> and 2,360 patients reported by Choi,<sup>7</sup> 43 and 134 exhibited neuropsychiatric sequelae respectively, but chorea was not observed.

The purpose of this presentation is to clarify the clinical characteristics of chorea following acute CO poisoning by analyzing 6 patients who completed brain CT scan studies and by reviewing the literature.

## MATERIALS AND METHODS

Of 3,223 patients with acute CO poisoning examined between 1976 and 2001 at Severance Hospital, Yonsei University Medical Center, Seoul, Korea, chorea was diagnosed in 3 cases only (one was reported previously.<sup>10</sup>). 3 patients with chorea after CO poisoning that completed a brain CT scan study from the literature<sup>11-13</sup> were added in this study.

These patients satisfied the following inclusion criteria : (1) evidence of acute CO poisoning, (2) chorea occurring after a latency of one week or longer, and (3) they completed a brain CT scan study. Patients were excluded if they had any evidence of (1) metabolic or neurodegenerative disease, (2) use of neuroleptic agents before the onset of chorea, (3) previous history of head trauma, stroke, perinatal hypoxia or encephalitis, and (4) a familial history of chorea.

Brain computed tomography (CT) was obtained from the 6 patients with chorea after CO poisoning. Neuroleptic agents such as haloperidol or chlorpromazine were used in attempts to treat

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Reprint address: requests to Dr. Il Saing Choi, Department of Neurology, Yonsei University College of Medicine, 134, Shinchon-dong, Seodaemun-gu, Seoul 120-752, Korea. Tel: 82-2-361-5465, Fax: 82-2-393-0705, E-mail: neuro@yumc.yonsei.ac.kr

chorea in 5 patients.

Chorea after CO poisoning was classified according to age, sex, latency, duration of choreic movements, associated clinical features, brain CT findings and outcome.

## RESULTS

The clinical, radiological and prognostic data are summarized in Table 1.

There were 2 men and 4 women (M:F=1:2). The age at onset ranged from 11 to 60 (mean 33.0) years. Only one subject was older than 40 years. All but one showed encephalopathy with mild impaired cognitive findings during or immediately after delayed CO sequelae. The latency period between CO poisoning and the onset of chorea was 10 to 30 (mean 21.7) days. The duration of chorea ranged from 14 to 90 days (the mean was 39.8 days.).

Of 6 the patients with chorea after CO poisoning, 5 had abnormal CT findings : bilateral low-density lesions in the basal ganglia and/or in the white matter of the cerebral cortex, and one subject was normal. There was no clear correlation between the sites of neuroimaging finding and the development of chorea. Chorea was alleviated by neuroleptic drugs in 5 patients, and one subject's signs disappeared spontaneously within 14 days. None of the subjects relapsed after the neuroleptic symptoms were arrested. All the subjects recovered completely, and they were without any

permanent sequelae.

## Case 1

A 39-year-old housewife was admitted to Severance Hospital with the chief complaint of unconsciousness, and she was treated with hyperbaric oxygen therapy under the diagnosis of acute CO poisoning, which was made by a complete history and by checking the blood carboxyhemoglobin levels (21%). She completely recovered 8 hours later, but 28 days after the acute insult, choreic movements of her extremities developed and gait disturbances followed 3 days later. She was then readmitted to Severance Hospital.

On admission, she was alert, but restless. Her memory and calculation were mildly impaired. Involuntary, nonrhythmic, brief, and rapid movements were evident in the extremities, face and tongue.

Her speech was clear, but she had difficulty modulating the volume. When performing finger to nose testing, she grasped the examiner's finger. She also had short-step gait without retropulsion. There were no other neurologic abnormalities.

Laboratory findings including CBC, urinalysis, serum electrolytes and liver function tests were normal. The chest X-ray and ECG were normal, and a brain CT scan and EEG revealed no abnormality.

The choreic movements spontaneously ceased 14 days after onset, and the encephalopathy after CO poisoning recovered 1 month later.

**Table 1.** Clinical Summary of 6 Patients with Chorea after CO Poisoning

No.	Sex/Age	Latency of chorea (days)	Associated findings	Duration of chorea (days)	Brain CT findings	Outcome	Reference
1	F/25	30	mild encephalopathy	40	gp & wm	recovery	10
2	M/17	10	dysarthria, hyperreflexia, mild mental changes	14	cn,p & gp	recovery	11
3	F/33	10	hypotonia, hyperreflexia, dysarthria, dysphagia	90	gp & ic	recovery	12
4	F/24	24	mild encephalopathy	60	gp & wm	recovery	13
5	F/39	28	mild encephalopathy	14	normal	recovery	presents
6	F/60	28	mild encephalopathy	21	gp	recovery	presents

cn, caudate nucleus; p, putamen; gp, globus pallidus; ic, internal capsule; wm, white matter of the cerebral cortex.

## Case 2

A 60-year-old woman was admitted to Severance Hospital with the chief complaint of unconsciousness for 6 hours, and she was treated with hyperbaric oxygen therapy under the diagnosis of acute CO poisoning, which was made by taking her history and by determining the blood carboxyhemoglobin levels (18%). One day later, she completely recovered, but 28 days after acute anoxia, restlessness and involuntary movements of her extremities developed. Four days later, urinary incontinence and gait disturbance followed.

On admission, she was alert, but confused. Her cognitive functions such as memory and calculation were mildly impaired. Involuntary, nonrhythmic, and rapid movements were evident in the face, tongue and extremities. She also had the grasping reflex and short-step gait with retropulsion. Her finger to nose testing showed a coarse tremulousness of both upper extremities. The tendon reflexes were very brisk, and there was no overt sensory deficit. Ankle clonus and Babinski sign were not elicited. Laboratory findings including CBC, urinalysis, serum electrolytes and liver function tests were normal.

Chest x-ray, ECG and EEG were also normal. Her brain CT scan showed low-density in both globi pallidi (Fig. 1).

Haloperidol (3 mg/day) was started, and the choreic movements ceased 21 days after medication. Encephalopathy and gait disturbance recovered 3 months later.

## DISCUSSION

Although this presentation is a limited case study, we can clarify the clinical characteristics of chorea following acute CO poisoning.

The incidence of chorea after CO poisoning is still unclear, but it may be extremely rare. The age of onset usually is younger than 40 years, and women are more often affected. All patients usually have delayed encephalopathy with mild cognitive dysfunctions, and the patients usually recover from this within several months. The latency period until the onset of chorea after CO

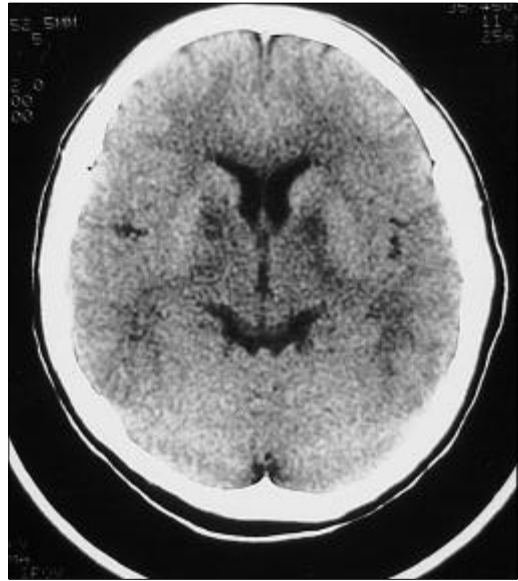


Fig. 1. CT scan 3 days after chorea that followed an acute CO poisoning shows low density in both globi pallidi (case 2).

poisoning is usually within a month.

The duration of the chorea after CO poisoning can range from days to months, and the chorea is usually alleviated by neuroleptic agents. There were no relapses after neuroleptic cessation and none of the subjects had permanent sequelae.

Chorea is symptomatic of many diseases of the nervous system, but most of them are related to rheumatic fever. Typical choreic movements appear in Sydenham's chorea and in the variety of that disease that is associated with pregnancy. Chorea is a major features of Huntington's chorea, in which the movements tend to be more typically to be choreoathetotic. Intoxication with phenothiazine drugs or haloperidol and rarely, hyperthyroidism, polycythemia vera, lupus erythematosus, cerebral arteritis, or stroke may also cause chorea.<sup>1-3</sup>

The anatomical basis for abnormal movements in CO poisoning is not known, although it is usually suggested that (1) a lesion in the putamen, or its afferent or efferent projections, may lead to dystonia, (2) chorea may be associated with caudate lesions, and (3) parkinsonism may result from bilateral globus pallidus lesions.<sup>8,9,14</sup>

In 1986, Schwartz et al<sup>11</sup> reported a case of choreoathetosis with infarction of the neostriatum on CT scan, suggesting a relationship between this

structure and the abnormal movements. However, the brain CT findings in this study showed bilateral low-density lesions in the basal ganglia and/or in the white matter of the cerebral cortex, yet these lesions can also be seen in non-choreic patients with CO poisoning;<sup>15,16</sup> therefore, there is no correlation between the neuroimaging findings and the development of chorea.

Of the rare cases of chorea following CO poisoning, some of them are transient,<sup>11</sup> and others have a chronic course.<sup>10,12,13</sup> The fact that chorea does not relapse after neuroleptic cessation may suggest the patients have a functional rather than an anatomical impairment. The pathophysiological pattern of the choreic movements may probably result from excessive dopaminergic output on the receptors of the striatum since chorea is alleviated by neuroleptic agents.<sup>12</sup>

The results of this study show that chorea after CO poisoning is extremely rare, and it appears as a part of delayed CO encephalopathy. The latency of onset for chorea is within a month, and the duration of the choreic movements ranges from 2 weeks to 3 months. Chorea is usually alleviated by neuroleptic drugs, and it do not relapse after neuroleptic cessation. The prognosis for these cases is excellent.

## REFERENCES

1. Aron AM, Freeman JM, Carter S. The natural history of Sydenham's chorea. *Am J Med* 1965;38:83-95.
2. Greenhouse AH. On chorea, lupus erythematosus, and cerebral arteritis. *Arch Intern Med* 1966;117:389-94.
3. Saris S. Chorea caused by caudate infarction. *Arch Neurol* 1983;40:590-1.
4. Lilienthal JL Jr. Carbon monoxide. *Pharmacol Rev* 1950;2:324-54.
5. Raskin N, Mullaney OC. The mental and neurological sequelae of carbon monoxide asphyxia in a case observed for fifteen years. *J Nerv Ment Dis* 1940;90: 640-59.
6. Shillito FM, Drinker CK, Shaughnessy TJ. The problem of nervous and mental sequelae in carbon monoxide poisoning. *JAMA* 1936;106:669-74.
7. Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. *Arch Neurol* 1983;40:433-5.
8. Scott BL, Jankovic J. Delayed-onset progressive movement disorders after static brain lesions. *Neurology* 1996;46:68-74.
9. Choi IS, Cheon HY. Delayed movement disorders after carbon monoxide poisoning. *Eur Neurol* 1999;42:141-4.
10. Choi IS, Hwang YM, Kim KW. Chorea as a clinical manifestation of delayed neurologic sequelae in carbon monoxide poisoning. *J Korean Neurol Assoc* 1984;2: 91-3.
11. Schwartz A, Hennerici M, Wegener OH. Delayed choreoathetosis following acute carbon monoxide poisoning. *Neurology* 1985;35:98-9.
12. Davous P, Rondot P, Marion MH, Gueguen B. Severe chorea after acute carbon monoxide poisoning. *J Neurol Neurosurg Psychiatry* 1986;49:206-8.
13. Kim JY, Kim JE, Jung BW, Suh CK. A case of neuroleptic malignant syndrome associated with choreo-ballism due to the delayed complication of carbon monoxide intoxication. *J Korean Neurol Assoc* 1994;12: 556-9.
14. Jellinger K. Degenerations and exogenous lesions of the pallidum and striatum. In: Vinken PJ, Bruyn GW, editors. *Handbook of Clinical Neurology*, vol 6. Amsterdam: North Holland; 1968. p.632-3.
15. Sawada Y, Ohashi N, Maemura K, Yoshioka T, Takahashi M, Fusamoto H, et al. Computerized tomography as an indication of long-term outcome after acute carbon monoxide poisoning. *Lancet* 1980;ii: 783-4.
16. Miura T, Mitomo M, Kawai R, Harada K. CT of the brain in acute carbon monoxide intoxication: characteristic features and prognosis. *AJNR* 1985;6:739-42.