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# A Clinical Study of Peripheral Neuropathy in Carbon Monoxide Intoxication

**Il Saing Choi**

*Department of Internal Medicine, Yonsei University College of Medicine*
*Seoul Korea*

Twenty cases of peripheral neuropathy as sequelae of carbon monoxide intoxication have been analyzed clinically. The incidence of pheripheral neuropathy was 0.84% in a total of 2,360 cases and 3.64% in 549 admitted cases of carbon monoxide intoxication.

The ratio of male to female was 1:1.2 (9:11). Their ages ranged from 17 to 52 years (mean 29.5 years), with a peak incidence in the 3rd decade (55%).

The lower extremity was exclusively involved, and the left side was more involved than the right. Symptoms were a burning sensation, tingling sensation, shooting pain and weakness. Other associated sequelae were local swelling, acute renal failure, delayed neurologic sequelae, and Volkman's contracture in that order. Of 20 cases, 6 showed abnormal findings in the electromyogram only, and 14 were abnormal in both electromyogram and nerve conduction velocity.

Fifteen cases recoved within 3 to 6 months.

**Key Words** : Peripheral neuropathy, CO Intoxication

In Korea, coal is the main domestic fuel for cooking and under-floor heating and a tremendous number of cases of carbon monoxide intoxication have occurred annually.

Carbon monoxide affects all tissue organs such as brain (Choi, 1982), heart (Corya, 1976), kidney, skeletal muscle (Loughridge, 1958), skin (Long, 1968) and perpheral nerve. There are more than 3,000 bibliographic references on carbon monoxide (Lilienthal, 1950; Finck, 1966), but peripheral neuropathy has rarely been reported following carbon monoxide intoxication (Wilson and Winkleman, 1924; Renfert and Drew, 1955; Snyder, 1970).

During the last 6 years, I have clinically analyzed 20 cases of peripheral neuropathy after carbon monoxide intoxication.

## MATERIAL AND METHODS

From January 1976 to December 1981, 2,360 cases of acute carbon monoxide intoxication were brought to the emergency room or out-patient department, and 549 cases were admitted to Severance Hospital, Yonsei Univer-

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sity Medical Center. Twenty cases of peripheral neuropathy occurred during the same period.

The ratio of male to female was 9:11 and their ages ranged from 17 to 52 years.

Electrophysiological studies were performed on all cases, but no pathologic study was done.

A follow up study of 15 cases was conducted for 1 year but 5 were lost to follow-up.

## RESULTS

The incidence of peripheral neuropathy was 0.84% in a total of 2,360 cases and 3.64% in 549 admitted cases of carbon monoxide intoxication. The ratio of male to female was 1:1.2 (9:11). The ages ranged from 17 to 52 years, with mean of 29.5 years. The most common

**Table 1. Clinical data on 20 cases of peripheral neuropathy after carbon monoxide intoxication**

| Case | Age | Sex | Symptomsns | Involved site | Electrophysiological studies | | Others |
|---|---|---|---|---|---|---|---|
| | | | | | NCV | EMG | |
| 1 | 23 | F | Pain & tingling sense | Rt. lower ext. | A | A | Swelling |
| 2 | 27 | F | Pain & tingling sense | Lt. Lower ext. | A | A | |
| 3 | 27 | M | Pain & weakness | Lt. lower ext. | A | A* | ARF with swelling |
| 4 | 43 | M | Pain & weakness | Rt. lower ext. | A | A | ARF, DNS, swelling |
| 5 | 48 | F | Pain & weakness | Lt. lower ext. | A | A | DNS |
| 6 | 52 | M | Pain & weakness | Both lower ext. | A | A* | ARF, DNS, swelling |
| 7 | 30 | F | Pain & weakness | Lt. lower ext. | A | A | |
| 8** | 32 | M | Pain & wakeness | Lt. lower ext. | N | A* | Swelling |
| 9 | 32 | F | Tingling sense | Both lower ext. | A | A | |
| 10 | 26 | F | Tingling sense | Lt. lower ext. | N | A | |
| 11 | 23 | F | Tingling sense | Both lower ext. | N | A | |
| | | | | Rt. upper ext. | A | A | |
| 12 | 23 | F | Tingling sense | Both lower ext. | | | |
| 13 | 23 | f | Tingling sense | Lt. lower ext. | N | A | |
| 14 | 25 | M | Tingling sense | Both lower ext. | N | A | |
| 15** | 26 | M | Tingling sense | Lt. lower ext. | A | A* | ARF, swelling |
| 16 | 24 | M | Tingling sense | Lt. lower ext. | N | A | |
| 17 | 29 | M | Weakness | Both lower ext. | A | A | ARF, swelling |
| 18 | 40 | M | Pain & tingling sense | Both lower ext. | A | A | ARF, swelling |
| 19** | 17 | F | Pain & weakness | Rt. lower ext. | A | A | Swelling |
| 20 | 19 | F | Pain & weakness | Both upper ext. | A | A | Volkman's contracture & swelling |

A : Abnormal
N : Normal
ARF : Acute renal failure
DNS : Delayed neurologic sequelae
* : Lumbosacral radiculopathy in EMG
** : Asymptomatic neuropathy on opposite side
EMG : Electromyogram
NCV : Nerve conduction velocity

incidence was in the 3rd decade (11 cases). All except one case involved the lower extremity. Nine cases of peripheral neuropathy involved the left side, 4 the right side and 7 both sides.

Twelve had only sensory symptoms such as a burning sensation, tingling sensation and shooting pain, 1 had a motor symptom, and 7 had combined symptoms. Peripheral neuropathy after carbon monoxide intoxication was associated with other sequelae: local swelling (10 cases), acute renal failure (6 cases), delayed neurologic sequeale (3 cases) and Volkman's contracture (1 case).

Nerve conduction velocity and electromyographic studies of 20 cases were performed. Six showed denervation potentials such as fibrillations and positive sharp waves in the electromyogram but normal in nerve conduction velocity and 14 had abnormal findings in both nerve conduction velocity and electromyogram.

In 3 cases, an asymptomatic extremity was possibly an asymptomatic peripheral neuropathy as suggested by electromyographic study. Four showed evidence of involved lumbosacral roots (Table 1).

A follow up study of 15 cases was conducted for 1 year. All recovered within 3 to 6 months.

## DISCUSSION

Since in 1857, Claude Bernard first described the toxic effect of tissue hypoxia, and in 1895, Halden described the underlying mechanism for carbon monoxide toxicity when he demonstrated that carbon monoxide reversibly interacted with hemoglogin, blocking the binding of oxygen to hemogobin, thus causing tissue hypoxia (Astrup, 1972), there are numerous bibliographies on carbon monoxide, but little has been written regarding peripheral neuropathy apparently resulting from acute carbon monoxide poisoning.

The precise pathogenesis of neuropathy remains in doubt, but 4 different mechanisms may be responsible for the changes found in the peripheral nerve: compression, ischemia due to hypoxia, petechial hemorrhage and carbon monoxide itself (Olsen, 1956; Snyder, 1970).

Histologic examination of clinically affected peripheral nerves in cases of carbon monoxide intoxication has shown demyelination with preservation of axons (Wilson and Winkleman, 1924), but this finding is rather non specific, and diverse forms of peripheral nerve injury can manifest as demyelination (Mayer and Denny-Browm, 1964).

From my electrophysiological studies, I believe that the axon and the nerve root may also be involved, as well as the peripheral nerve.

Reviewing case reports about carbon monoxide intoxication (Wilson and Winkleman, 1924; Renfert and Drew, 1955; Synder, 1970), there was no article citing peripheral neuropathy, after carbon monoxide intoxication.

From my clinical experience, peripheral neuropathy after carbon monoxide intoxication has the following characteristics; It equally affects both sexes, commonly occurs in the young adult, exclusively involves the lower extremity, has motor and sensory symptoms, involves the nerve root as well the perpipheral nerve, and complete recovery occurs within 3 to 6 months.

## REFERENCES

Astrup P: *Some physicological and pathological effects of Moderate carbon monoxide expasure. Brit Med J 4:447, 1972*

Choi IS: *A study of neurologic sequelae in carbon monoxide intoxication. J Korean Med Assoc 25:341, 1982*

Corya BC, Black MJ and McHensy PL: *Echocar-*

*diographic findings after acute carbon monoxide poisoning. Brit Heart J 38:712, 1976*

Finck PA: *Expose to carbon monoxide: Review of the literature and 567 autopsies. Milit Med 131:1613, 1966*

Long PI: *Dermal changes associated with carbon monoxide intoxication. JAMA 205:50, 1968*

Lilienthal JL: *Carbon Monoxide. Pharmacol Rev 2:324, 1950*

Longhridge LW, Leadear LP and Bowen DAL: *Acute renal failure due to muscle necrosis in carbon monoxide poisoning. Lancet 1:349, 1958*

Mayer RF and Denny-Brown D: *Conduction velocity in peripheral nerve during experimental demyelination in the cat. Neurol 14:714, 1964*

Olsen CW: *Lesions of peripheral nerves developing coma, JAMA 160:39, 1956*

Renfert H and Drew A: *Peripheral neuritis as a sequela of carbon monoxide asphyxiation. A case report. Ann Intern Med 42:942, 1955*

Snyder RD: *Carbon monoxide intoxication with peripheral neuropathy. Neurol 20:177, 1970*

Wilson G and Winkleman NW: *Multiple neuritis following carbon monoxide poisoning. JAMA 82:1407, 1924*