

Myoclonus, Delayed Sequelae of Carbon Monoxide Poisoning, Piracetam Trial

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One month following carbon monoxide poisoning, a 39 year-old man developed incontinence, memory impairment, disorientation and emotional instability. He was hospitalized 7 weeks later, and during hospitalization he exhibited myoclonic movements of the neck and lower limbs. He was given piracetam intravenously for 11 days. The myoclonus was significantly reduced by the third day of treatment and had disappeared by the seventh day. There was no recurrence following cessation of treatment.

Key Words: Carbon monoxide poisoning, myoclonus, piracetam.

A variety of neuropsychiatric manifestations appear as delayed sequelae to carbon monoxide (CO) poisoning (Garland and Pearce 1967; Choi 1983). Although chorea is rarely associated with CO poisoning, Choi *et al.* (1984) described such a case. This case report reviews a patient who after CO poisoning presented a number of delayed encephalopathic symptoms including myoclonic movements. These were treated with piracetam with favorable results.

CASE REPORT

A 39 year-old man was admitted to Severance hospital in 1986, with the chief complaints of incontinence and mental impairment. Seven weeks prior to admission he was found in a comatose state in an ondol heated room, transported to a local hospital and treated for CO poisoning. Although he appeared to recover within 30 hours, one month later he exhibited the followings: uneven gait, incontinence, memory loss, disorientation, and emotional instability. There was no history of familial disease or past illness. His only hospitalization was 10 years previously for a herniated lumbar disc.

Upon admission, he was alert, but displayed im-

paired neurological functions involving orientation, memory, calculation, glabellar reflex and ataxia. Vital signs were normal as were chest X-ray and brain CT scan. Laboratory results including CBC, urinalysis, blood chemistry and STS were within normal limits. While there were no significant findings in the EMG or somatosensory evoked response, the EKG showed left ventricular hypertrophy. The abnormal EEG findings were labeled Class 8 (UAB Classification System) and included background suppression. Slow theta to delta waves were seen in the both hemispheres, while spikes were associated with both frontal regions.

Myoclonic jerks of the neck and lower limbs developed spontaneously during hospitalization 8 weeks after poisoning. They occurred while the patient was awake and resting, in a variety of positions, i.e. sitting in a chair, lying in bed on his back or side. They did not occur during sleep and were not evoked by stimulus or action. The movements were irregular in rhythm, rapid, brief, and monophasic or occasionally biphasic contraction-relaxation, and were recorded on a video-cassette tape. Piracetam was administered intravenously 3 days after the onset of myoclonus. The dosage was 12 gm/day for 5 days followed by 9 gm/day for 6 days. By the third day of treatment, the frequency and amplitude of the myoclonic jerks were reduced. They disappeared by the seventh day. There was no recurrence following cessation of treatment.

DISCUSSION

Various neurological sequelae develop after acute

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CO poisoning (Garland and Pearce 1967; Choi 1983). Chorea or choreoathetosis (Jellinger 1968; Choi *et al.* 1984; Schwartz *et al.* 1985; Davous *et al.* 1986), action myoclonus (Lance and Adams 1963; Terwinghe *et al.* 1978; Fahn 1979; Young and Shahani 1979) and Gilles de la Tourette's syndrome (Pulst *et al.* 1983) were described in several cases as delayed posthypoxic sequelae. However, the literature contains no reference to the occurrence of myoclonus while the patient is at rest.

Myoclonus is defined as an involuntary, irregular, lightning-like muscular contraction, single or repeated, of a single fascicle of muscle fibers or of a group of muscles, with or without displacement of a body part (Aigner and Mulder 1960; Lance and Adams 1963; Terwinghe 1981). It can be initiated by several different stimuli (visual, auditory, tactile) or may develop during a sustained motor activity (action myoclonus) (Lance and Adams 1963; Terwinghe *et al.* 1978; Fahn 1979; Young and Shahani 1979). Neuropharmacological research has indicated that there is a decrease in serotonin in the central nervous tissue, which results in a reduction in the level of 5-hydroxyindole acetic acid in the cerebrospinal fluid of patients with posthypoxic action myoclonus. Substitution therapy utilizing L-5-hydroxytryptophan, a precursor of serotonin, gave very good results (Guilleminault *et al.* 1973; Chadwick 1977; Terwinghe 1981; Marsden *et al.* 1985). However, there were severe side-effects. This led to the initial discovery of the therapeutic effect of piracetam in one case of postanoxic action myoclonus (Terwinghe *et al.* 1978; Terwinghe 1981), which was followed by similar studies with beneficial results (Cremieux and Serratice 1979; Terwinghe 1981).

Piracetam has chemical kinship to gamma-aminobutyric acid. Indications are that it enhances the turnover rate of intracerebral ATP, although the exact mechanism is not known. Its physiological distribution and function with regard to neurotransmitters has not yet been established (Nyback *et al.* 1979; Terwinghe 1981; Allikmets *et al.* 1983; Bering and Muller 1985).

This case report reviews the results of treatment with piracetam in a patient who developed myoclonus after CO poisoning. Although the myoclonic jerks occurred in the resting state and were not action myoclonus and there was no recurrence of myoclonus after cessation of piracetam, intravenous piracetam therapy was likely to be an effective method of treatment.

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