Delayed Encephalopathy after Carbon Monoxide Intoxication: Prognosis is Still Uncertain

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In acute carbon monoxide (CO) intoxication, the majority of patients are either rapidly fatal or recover without neurological sequelae[1]. Among the few remaining patients with neurological sequelae, some progress to a semi-vegetative and akinetic state and remain in such condition for months to years before death. Others develop neuropsychiatric encephalopathy weeks after initial recovery. Still others have a variety of permanent mental and/or neurological symptoms.

Clinical studies of delayed encephalopathy following acute CO intoxication, particularly by Choi[2] on 2380 victims and by Min[3] on 738 victims, have shown that the incidence is estimated to be about 3%, and the onset is relatively sudden after a lucid interval ranging from 2 to 40 days (mean 22 days). The peak ages are between 5th and 7th decades, indicating that young persons are infrequently affected. The occurrence of delayed encephalopathy is also related to the duration of initial coma which is significantly longer for the progressive type than for the delayed relapse type[4].

The frequent symptoms of delayed encephalopathy are disorientation, irritability, amnesia, apathy, apraxia, urinary and/or fecal incontinence, gait disturbance, mutism, hypokinesia, expressionless face, small-stepped gait, bizarre behaviors, retropulsion and increased muscle tone[1-4]. The majority (about 75%) of cases with delayed encephalopathy recover within one year. Permanent parkinsonism occurs in about 5% of those with delayed neurological sequelae. Noticeably missing in the extrapyramidal syndrome is hyperkinesias, including dystonia, chorea, athetosis and tremor[5].

In this issue, Hsiao et al.[6] reported a retrospective study on delayed encephalopathy after CO intoxication emphasizing the long-term prognosis and its correlations with clinical manifestations and neuroimaging findings. Twelve patients were identified to have delayed encephalopathy among 89 patients with acute CO intoxication from 1985 to 2000. Initial disturbance of consciousness was severe (deep coma) only in 4 patients, indicating that the remaining 8 patients probably had not had severe CO intoxication. This might partly explain the good prognosis of this study. Unfortunately, mini-mental and other cognitive tests were not performed, particularly in the early stage of delayed encephalopathy.

The observed clinical symptoms of delayed encephalopathy were cognitive impairment, akinetic mutism, sphincter incontinence, gait ataxia and extrapyramidal syndrome. In the latter, dystoria was surprisingly as frequent as parkinsonian features, but the explanation was not provided. MRI showed common involvement of globus pallidus and subcortical white matter that would resolve with time, particularly the latter. The order of recovery was sphincter incontinence,

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cognitive impairment, and extrapyramidal features. Such phenomenon of recovery was explained by the reversibility of subcortical white matter lesions and the partial reversibility of basal ganglia lesions. To be convincing, the relationship between cognition and subcortical white matter needs to be further explored.

This study has several short-comings in data collection. The sample of patients was small and the retrospective record review would have some unfortunate consequences: i.e. Formal cognitive tests were not adequately performed; MRI evaluation was not obtained in the follow-up of half of the patients; Neuropsychiatric symptoms were not adequately evaluated and categoried, particularly cognitive and psychiatric symptoms. Therefore, this study is of value in providing the clinical manifestations of delayed encephalopathy in Taiwan, but still fails to provide new insight into the pathogenesis and prognosis of delayed encephalopathy after CO intoxication.

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