Case Report

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Delayed Neuropsychiatric Syndrome Following Carbon Monoxide Poisoning: Report of a Rare Case With Response to Treatment

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Abstract

Carbon monoxide (CO) is a colorless, odorless, and non-irritant gas that is lighter than air. Carbon monoxide intoxication is the leading factor of deaths due to toxications. Besides, it is an important health problem due to its morbidities related to the delayed neurological and/or psychiatric syndromes that occur after the acute recovery period. Besides behavioral problems, mood and personality changes, parkinsonism, motor deficits, and dementia syndrome are the most common clinical pictures. Compared with hyperbaric oxygen, which has been proven effective in the acute period in CO intoxication, there is no treatment with definitely proven efficacy for the delayed neuropsychiatric picture. In this paper, a case of CO intoxication followed with a delayed neuropsychiatric syndrome is presented in the light of the literature.

Keywords: carbon monoxide, poisoning, delayed neuropsychiatric syndrome, methylprednisolone, memantine

Introduction

Carbon monoxide (CO) is a colorless, odorless, lighter than air and non-irritating gas. Carbon monoxide poisoning usually presents with a severe clinical course and may result in severe neurological sequelae and even death[1]. Rarely, neurological and/or psychiatric syndromes may be encountered following the recovery period after acute CO poisoning. The delayed neurological or neuropsychiatric syndrome usually emerges within one to four weeks following acute poisoning. The typical radiological feature of this syndrome is mostly symmetrical, demyelinating lesions that are localized to the subcortical white matter[2]. The prevalence of the neuroimaging lesions is closely related to the severity of the clinical picture and the poor prognosis[3].

In this report, a case in whom a delayed neuropsychiatric syndrome had developed following CO poisoning is presented to be discussed in terms of its pathophysiological and clinical features and treatment.

Case Presentation

A 69-year-old female patient was admitted to the emergency department of our hospital with a complaint of nervousness, weakness, and fatigue in the last two weeks. The weakness in both arms and legs had worsened for the last week. She could not walk without support and perform self-care. She was discharged from another hospital with no sequelae after hyperbaric oxygen therapy, where she applied for CO poisoning about six weeks ago. During the neurological examination of the patient, she was conscious, albeit partially disoriented for place and time. We found mild dysarthria and quadriparesis. The standardized mini-mental test score was 20/30. The brain tomography was normal. Cranial magnetic resonance imaging revealed diffuse demyelinating lesions without contrast enhancement. The lesions which were located at the periventricular white matter and centrum semiovale were mildly hypointense in the T1W sequence and hyperintense in T2W and FLAIR sequences (Figure 1). The widespread slow-wave activity was observed in the electroencephalogram (Figure 2). The rest of the laboratory and imaging tests, performed for differential diagnosis were normal. The patient, who was suspected to have late period demyelinating central nervous system involvement, was hospitalized in our clinic. We have decided to manage the clinical picture with intravenous methylprednisolone 1 gram daily for 10 days, memantine 20 mg daily, and physiotherapy. Behavioral and cognitive problems started to improve immediately under these treatments. The patient was discharged in the 4th week of follow-up when she could be independently mobilized and perform daily routine activities.

Discussion

Carbon monoxide is an odorless, colorless, non-irritating gas formed as a result of hydrocarbon combustion. Carbon

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monoxide poisoning is the leading cause of death due to toxins[1]. It binds to hemoglobin with a higher affinity than oxygen and converts to carboxyhemoglobin (COHb), disrupting oxygen transport. Carbon monoxide can cause derangement in many systems but particularly affects the central nervous and cardiovascular systems because of their highest consumption rate of oxygen. In severe CO poisoning, cardiovascular and metabolic complications such as seizures, coma, myocardial ischemia, arrhythmia, pulmonary edema, lactic acidosis, and irreversible neurological deficits can be seen[2]. Acute myocardial ischemia is the most important clinical presentation determining long-term mortality[3].

The mechanism of late pathological changes due to CO poisoning is not clear. White matter lesions may arise based on cerebral vascular damage, cerebral parenchymal cytotoxic edema, and hypersensitivity reaction as a result of the combined effects of acidosis, hypoxia, and hypotension. Direct toxic effects as a result of various intracellular proteins that bind CO is also a contemplated mechanism. It is among the theories that lipid peroxidation caused by toxic oxygen molecules may be another cause[4]. Besides, it was proposed that exposure to hyperoxia as a result of reperfusion during the recovery period after acute CO poisoning may increase the existing oxidative damage[2].

Although the first one to four weeks after acute CO poisoning is the most common time for the development of a delayed neuropsychiatric syndrome, cases may be seen ranging from 3 to 240 days in the literature[5]. Morbidity depends on early neurocognitive impairment a great deal. Delayed neuropsychiatric syndrome and cognitive sequelae, which reach a serious extent, are extremely rare, and it has been reported that sequelae findings may persist for 1 year or longer (6). Our patient had applied to the neuropsychiatry clinic approximately four weeks after the end of the hyperbaric oxygen treatment, and no other etiology that would cause widespread white matter demyelination was detected.

The incidence of the delayed neuropsychiatric syndrome was reported to be 2.75% in a case-serial study including 2360 cases[7]. However, it has been emphasized that the frequency may reach 10% in recent studies[8]. Carbon monoxide poisoning causes nonspecific symptoms such as headache, visual disturbances, dizziness, nausea, and vomiting in the acute period. Mental status changes ranging from mild confusion to coma often accompanies. Cognitive disorders, dementia, mood abnormalities, and personality changes can be seen in the late period following CO poisoning. In many cases in the literature, the primary neurological symptom had been parkinsonism in addition to cognitive loss[9]. In our case, it was known that during the acute CO poisoning period, there were no neurological or cardiac symptoms other than headache and confusion. However in the late period, there were quadriparesis, dysarthria, and agitation in addition to cognitive impairment.

Hyperbaric oxygen administration is the accepted and applied treatment in the acute phase of CO poisoning. However, there is still no commonly applied and accepted method in the treatment of delayed neuropsychiatric syndromes. Re-application of hyperbaric oxygen administration was evaluated in a meta-analysis study, but its effect could not be demonstrated[10]. Therefore, corticosteroid applications have come to the fore intending to reduce inflammatory and oxidative stress. This approach has resulted in a favorable response in a small portion of the patients. In a few patients, steroids have been used successfully in combination with

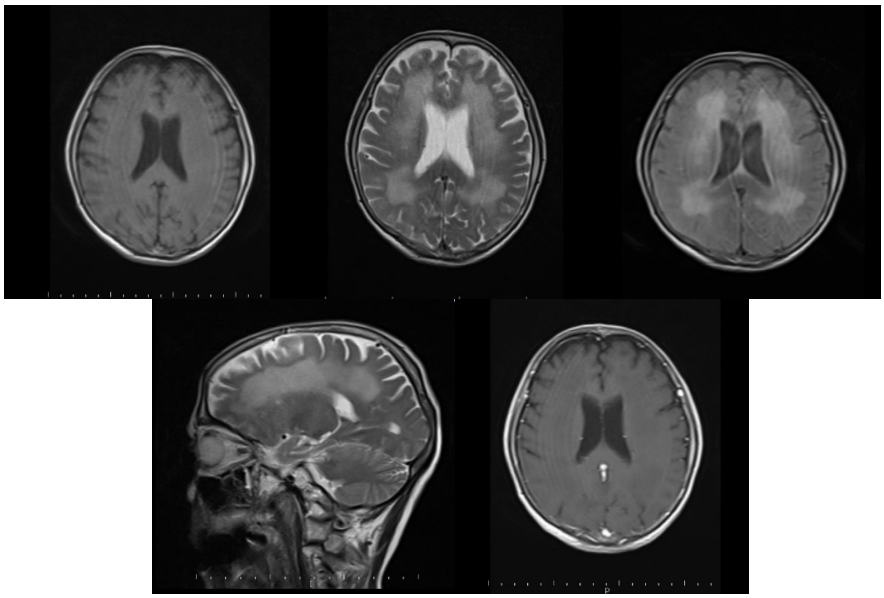

Figure 1. Diffuse demyelinating lesions without contrast enhancement at periventricular white matter and centrum semiovale mildly hypointense in the T1W sequence and hyperintense in T2W and FLAIR sequences.

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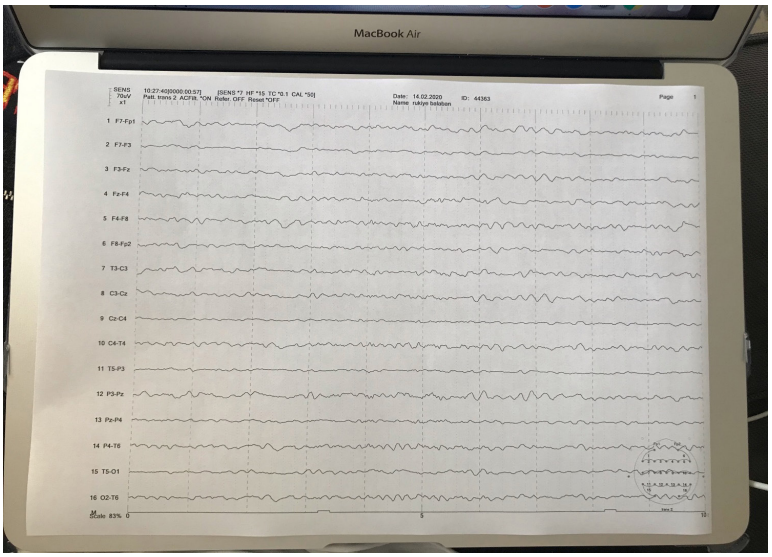

Figure 2. The widespread slow-wave activity in the electroencephalogram.

memantine as in our case. Parkinsonism is a common motor deficit and hence, a levodopa trial is proposed. However, the results of levodopa are frustrating[11].

Conclusion

Acute CO intoxication is known to be life-threatening, with rare but serious complications. Consequently, CO poisoning should be considered and questioned by emergency department personnel in patients who admit signs of the delayed neuropsychiatric syndrome. This approach avoids the risk of morbidity and death in such cases.

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