

# Rhabdomyolysis as a complication of carbon monoxide poisoning

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## Abstract

Carbon monoxide (CO), a highly toxic gas produced by incomplete combustion of hydrocarbon, is a relatively common cause of human injury. When the exposure history is absent, or delayed, the diagnosis is difficult. A 66-year-old woman, who use the briquette boiler in home, presented with rhabdomyolysis. Diffusion-weighted MRI showed high signal intensities in globus pallidus bilaterally. Her symptoms and high creatine kinase level were improved by conservative management. Our case report shows that the prognosis of CO poisoned patient greatly depends on timely and appropriate treatment, severity of damage to other organs, and success of the treatment of complications. (*J Med Life Sci* 2016;12(2):85-87)

**Key Words** : Carbon monoxide, Creatine kinase, MRI, Rhabdomyolysis

## Introduction

Carbon monoxide (CO) intoxication is one of the most common types of poisoning in the modern world<sup>1)</sup>. CO is a tasteless, odorless, non-irritating but highly toxic gas. Because of these properties and it lacks a unique clinical signature, CO is difficult to detect and can mimic other common disorders. The diagnosis of CO intoxication is frequently made obviously by the patients own history or by presentation of others who shared a common environment.

The symptoms of CO poisoning are non-specific<sup>2)</sup>. Mild exposure to CO causes headache, myalgia or dizziness whereas severe exposure will result in confusion, loss of consciousness or death. Every organ in human body could be damaged due to CO poisoning. However, the brain and heart with high metabolic rate are most susceptible to it. Hypoxic brain damage predominates in the cerebral cortex, cerebral white matter, and basal ganglia, especially in the globus pallidus<sup>3)</sup>. A few reports have described the association of rhabdomyolysis with CO poisoning<sup>4-6)</sup>. We report a patient who developed muscle necrosis and showed characteristic MRI findings following CO intoxication.

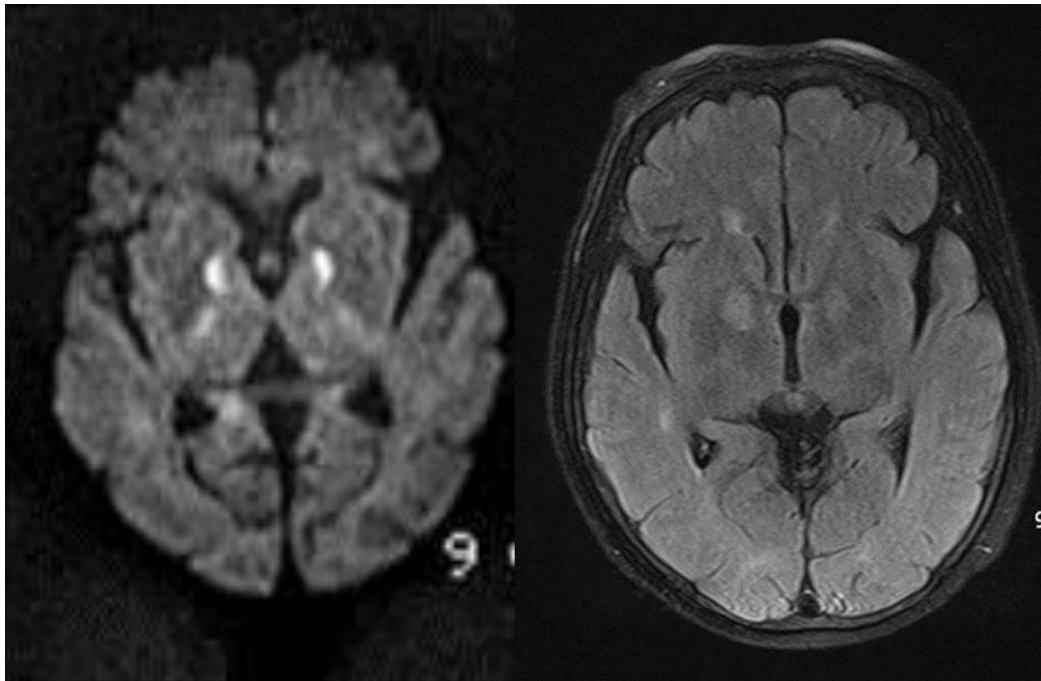
## Case Report

A 66-year-old woman presented with general weakness and headache. Two days ago, when she woke in the morning, she could not stand and had difficulty in raising the arms. Her home used the briquette boiler as heating. On arrival in the hospital, she was alert and oriented. Vital signs were stable. Cherry-red discoloration of the skin and cyanosis were not observed. Neurological examination showed bilateral and symmetric motor weakness, which were more severe in proximal muscle than distal. Sensory and cranial nerve examination were normal. Deep tendon reflexes were hypoactive in lower extremities. No pathologic reflexes were observed. Arterial blood gas examination was normal at this stage. ECG revealed inferolateral T wave inversion. Chest X-ray was normal. She had mild renal failure and a markedly elevated creatine kinase (CK) level of 6270 IU/l. Urine toxicology screen was negative. Acute phase reactants, such as ESR and CRP, were highly elevated. Brain MRI showed increased signal intensities bilaterally in the putamen and globus pallidus on diffusion-weighted and FLAIR images (Fig.1). Apparent diffusion coefficient image demonstrated low signal intensity. Electroencephalograph revealed mild diffuse cerebral dysfunction. Although carboxyhemoglobin (COHb) level was not measured due to short half life of COHb, CO intoxication was suspected from brain imaging findings and circumstances in briquette boiler use. During the course of the first week of hospitalization motor weakness was

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improved to independent gait. CK level was gradually declined and normalized 8 days after admission. Similarly

her renal failure resolved with intravenous fluids.



**Figure 1.** MRI findings. Diffusion-weighted (left) and fluid-attenuated inversion recovery (right) images obtained 1 day after exposure to carbon monoxide show symmetrical high-signal intensity lesions in the bilateral globus pallidus.

## Discussion

Carbon monoxide is one of the common fatal poisons in Korea. The affinity of hemoglobin for CO is 200 to 250 times as great as its affinity for oxygen. CO toxicity is dependent on the concentrations of CO and oxygen in the ambient air and the duration of exposure<sup>2)</sup>. Diagnosis of CO intoxication requires a high level of suspicion. Epidemiological history with information about other affected individuals or pets as well as circumstances suggestive of possible exposure is of paramount importance.

Because of their high metabolic rate, the brain and the heart are most susceptible to CO toxicity<sup>3)</sup>. The clinical symptoms of CO intoxication are often non-specific and can mimic a variety of common disorders. The severity ranges from mild flu-like symptoms to coma and death. CO intoxication has previously been associated with amnesia, encephalopathy, parkinsonism, peripheral neuropathy, cardiotoxicity and muscle necrosis with renal failure<sup>7)</sup>. The association of muscle necrosis with CO intoxication has been reported previously<sup>4-6)</sup>. Pressure necrosis of the muscles

occurs with immobilization and is probably enhanced by the impairment in oxygen delivery due to the accumulation of carboxyhemoglobin<sup>8)</sup>. Cardiac muscle in our case appeared to be less sensitive than skeletal muscle to the carboxyhemoglobin-induced anoxia since only mild changes were noted on serial electrocardiogram.

Treatment of CO poisoning begins with inhalation of a high concentration oxygen and aggressive supportive care. Hyperbaric oxygen therapy accelerates the dissociation of CO from hemoglobin and may prevent delayed neurologic sequelae<sup>9)</sup>. Nevertheless, the indications for hyperbaric oxygen therapy for CO poisoning remain controversial, and the ideal regimen of oxygen therapy is yet to be determined, and significant controversy exists regarding hyperbaric oxygen therapy protocols.

In a large MRI study of 19 patients with acute CO intoxication, the globus pallidus was most commonly involved. Diffusion weighted images (DWI) in brain MRI show white matter high signal intensities consistent with restricted diffusion in acute CO intoxication<sup>10-12)</sup>. On corresponding ADC maps, low signal intensity and low ADC

values were noted at these regions. Our experience with this case suggested that DWI is superior to MR imaging with respect to lesion detection during the acute stage of CO intoxication.

We describe a case of CO poisoning that led to rhabdomyolysis and pallidoreticular damage. This case indicates that muscle necrosis can be developed as a result of CO poisoning and diffusion-weighted imaging is useful for early identification of the effects of acute CO poisoning.

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