Clinical Case Report

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A case report on the acute and late complications associated with carbon monoxide poisoning

Acute kidney injury, rhabdomyolysis, and delayed leukoencephalopathy

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Abstract

Rationale: Acute kidney injury (AKI), rhabdomyolysis, and delayed leukoencephalopathy after carbon monoxide (CO) poisoning are very rare. We report a case presenting with AKI, rhabdomyolysis, and delayed leukoencephalopathy after CO poisoning.

Patient concerns: The patient was admitted to our emergency department due to loss of consciousness after CO exposure during a suicide attempt.

Diagnoses: Laboratory findings revealed elevated carboxyhemoglobin, serum creatinine, and serum muscle enzyme levels. Initially, this patient was diagnosed with AKI and rhabdomyolysis due to CO poisoning. A month after the CO poisoning, she showed neuropsychiatric symptoms. Brain magnetic resonance imaging showed white-matter hyperintensity on the T2 flair image. Therefore, she was diagnosed with delayed leukoencephalopathy after CO poisoning.

Interventions: At the same time as diagnosis of AKI and rhabdomyolysis, the normobaric oxygen and hydration therapies were performed. A month later, rehabilitation was started due to delayed leukoencephalopathy.

Outcomes: Her renal function and muscle enzyme levels were completely restored with alert mental status. She could walk with the aid of a walker at last visit.

Lessons: This case shows that we should consider about rare acute and late complications such as AKI, rhabdomyolysis, and delayed leukoencephalopathy after CO poisoning.

Abbreviations: AKI = acute kidney injury, CO = carbon monoxide, COHb = carboxyhemoglobin, CPK = creatine phosphokinase, Cr = creatinine, MRI = magnetic resonance imaging.

Keywords: acute kidney injury, carbon monoxide poisoning, delayed leukoencephalopathy, rhabdomyolysis

1. Introduction

In the past, many carbon monoxide (CO) poisoning accidents occurred due to briquette poisoning in Korea. Recently, the use of briquette has been reduced by industrial developments, thereby reducing CO poisoning incidence. However, CO poisoning has become an issue again as cases of briquette gas suicide increases.[1] The affinity between hemoglobin and CO is approximately 230 times stronger than those between hemoglobin and oxygen.[2] Myoglobin is also known to have a 60-fold higher affinity with CO than oxygen and has a poor ability to utilize oxygen when bound with CO.[3] This can lead to ischemic damage to the heart, muscles, and brain.

This is a case report of a 32-year-old patient who suffered from delayed encephalopathy after recovering from acute kidney injury (AKI) and rhabdomyolysis due to CO poisoning.

2. Case report

A 32-year-old woman visited the emergency department with low consciousness level. She had inhaled anthracite gas due to suicide attempt 7 weeks earlier. At that time, her laboratory findings upon arrival were creatinine (Cr) level of 1.4 mg/dL, creatine phosphokinase (CPK) of 5991 IU/L, myoglobin of 994.8 ng/mL, and carboxyhemoglobin (COHb) of 44.8%. She was diagnosed with AKI and rhabdomyolysis due to CO poisoning. Hyperbaric oxygen therapy was not performed, and only oxygen therapy at a high concentration of 15 L/min using partial rebreathing mask with intravenous fluids was administered. After treatment, her consciousness level, renal function (Cr 0.4 mg/dL), and rhabdomyolysis (CPK 190.0 IU/L, myolgobin 41.5 ng/mL) with COHb of 0% returned to normal. Two weeks before admission, she had

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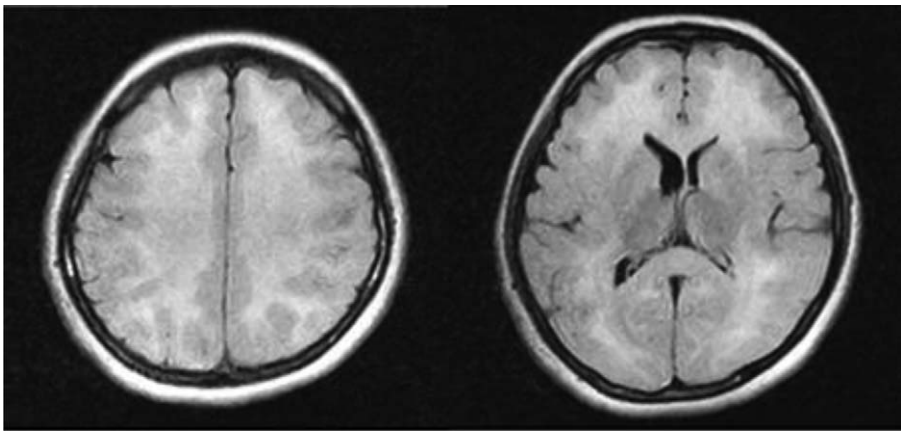

Figure 1. Initial brain MRI on T2 flare – 7 weeks later after CO poisoning. Brain MRI showed confluent T2 hyperintensity in the cerebral white matter that suggested delayed leukoencephalopathy. CO = carbon monoxide, MRI = magnetic resonance imaging.

difficulty in responding to questions and could not walk without help. Since 1 week before admission, she had spent most of her time in bed and was unresponsive.

Upon admission, her blood pressure was 100/60 mmHg, pulse rate 84/min, body temperature 37.1°C, and respiration 22/min. In the neurological examination, the tendon reflex was symmetrical, and the limb movement power was reduced to 2/5. The assessment for her sensory function and gait could not be measured, because she had aphasia.

The patient's laboratory findings were as follows: white blood cell count, 9800/mm^3; hemoglobin, 12.9 g/dL; and platelet count, 260,000/mm^3. Biochemistry showed protein of 6.4 g/dL, albumin 3.7 g/dL, blood urea nitrogen 10.5 mg/dL, Cr 0.4 mg/dL, aspartate transaminase/alanine transaminase 25/40 IU/L, Na/K/Cl 138/4.1/99 mEq/L, lactate dehydrogenase 371 IU/L, CPK 41.52 IU/L, myoglobin 41.52 ng/mL, and C-reactive protein 11.7 mg/dL. Arterial blood gases showed a pH of 7.494, pCO_2 of 32.9 mmHg, pO_2 of 98.5 mmHg, and HCO_3^- of 24.7 mmol/L. Her renal function was not impaired and muscle enzymes such as CPK and myoglobin were within the normal values.

Brain magnetic resonance imaging (MRI) showed confluent T2 hyperintensity in the cerebral white matter, suggesting the possibility of reversible demyelination. No other significant focal lesions were found in the brain parenchyma (Fig. 1).

The patient was diagnosed with delayed leukoencephalopathy due to CO intoxication based on clinical and radiologic findings, such as consciousness deterioration, decreased limb movement, and brain white matter high signal on T2 image. Rehabilitation was started. She did not cooperate during her first visit and only nodded her head when she heard her name. She could not even say a simple word. During 1 week of hospitalization, she complained of leg pain in 2 words. Two weeks after admission, limb movement power increased to 3/5. Although ambulation was impossible, she could speak her name and several other words. She was discharged on the 25th hospital day.

She was transferred to a nearby hospital and continued rehabilitation treatment. After 40 days of symptom onset, she was ambulatory, and on the 70th day after admission, she showed more improved state of cognitive function and could walk with the aid of a walker. She returned for follow-up, and brain MRI was performed 3 months after symptom onset. Although brain MRI showed bilateral cerebral white matter with confluent T2 hyperintensity, it showed significantly lower signal on the T2 image compared to the previous MRI results (Fig. 2).

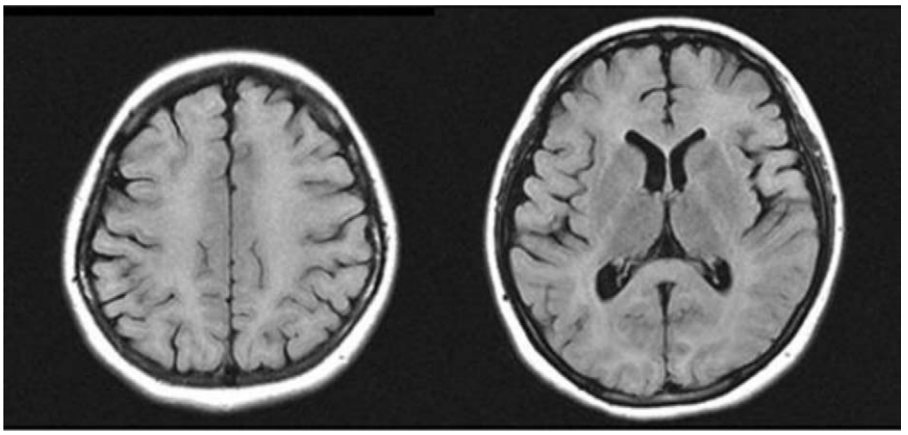

Figure 2. MRI on T2 flare – 3 months later. Brain MRI showed bilateral cerebral white matter with confluent T2 hyperintensity, significantly reduced signal on T2 image compared to the previous MRI. MRI = magnetic resonance imaging.

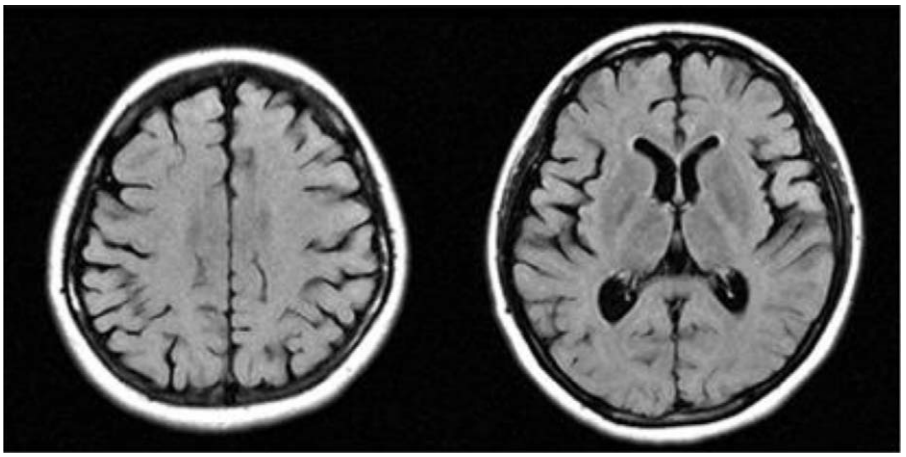

Figure 3. Brain MRI on T2 flare – 1 year later. Brain MRI showed confluent T2 hyperintensity normalized in cerebral white matter, but gray–white matter differentiation was unclear. MRI = magnetic resonance imaging.

A year later, she still needed a walking aid to walk, but her sensory function was intact. Follow-up brain MRI was performed, which showed confluent T2 hyperintensity normalized in the cerebral white matter, but the gray–white matter differentiation was unclear. It was thought to be sequelae of CO poisoning; however, no focal lesions were observed on the brain parenchyma (Fig. 3).

3. Discussion

Since the mid-1950s, Korean society started to use coal briquettes as fuel for cooking and heating. Then, CO poisoning incidence increased during the 1960s. This trend had continued until the 1980s. In the 1990s, CO poisoning was dramatically reduced as coal briquettes were replaced by oil. However, CO poisoning using burning coal briquettes during suicide attempts has recently increased. The number of suicides associated with CO poisoning were 34 among the total 10,653 suicide cases (0.3%) in 2006, but 1125 among the total 14,159 suicidal cases (7.9%) in 2012.[4] Furthermore, >50,000 patients with CO poisoning visit the emergency departments in the United States per year.[5]

CO poisoning causes acute and late complications. Symptoms of acute CO poisoning may include headache, myalgia, dizziness, or neuropsychological impairment as well as confusion, loss of consciousness, or death depending on the CO exposure level.[6] Additionally, CO poisoning can induce tissue and organ damage, such as the cardiovascular and respiratory systems, muscles, liver, and kidneys. Major causes of tissue and organ damage is not only hypoxia, but also oxidative stress, formation of oxygen reactive species, neuron necrosis, apoptosis, and abnormal inflammation.[7]

AKI is a clinical syndrome that decreases renal function, resulting in retention of nitrogenous waste products and dysregulation of extracellular volume and electrolytes. This case is categorized as stage 3 on the AKI Network. AKI is rarely caused by CO poisoning. The main mechanism is the ischemia–reperfusion injury most likely caused by toxic effects on the vascular endothelium, which led to the release of reactive chemical compounds with neutrophil activation and/or adhesion. These reactive chemical compounds caused progressive damage to the blood vessels according to enhanced lipid peroxidation.[8]

Rhabdomyolysis is another acute complication of CO poisoning and has no definite diagnostic criteria; however, elevated CPK levels of over 5 times of its upper normal limit and/or myoglobinuria can be used as its diagnostic factors. Rhabdomyolysis can progress to AKI because of the direct nephrotoxic effect of myoglobin, renal vasoconstriction, and tubular obstruction.[1] In this case, the patient suffered from AKI and rhabdomyolysis due to acute CO exposure. Her serum Cr level was 1.4 mg/dL upon arrival at the emergency room after CO exposure. After treatment, it was normalized to 0.4 mg/dL. The risk of rhabdomyolysis-induced AKI is increased when the serum CPK level is >15,000 IU/L and fractional excretion of sodium is approximately <1%. Upon arrival at the emergency room, the serum CPK level is 5991 IU/L and fractional excretion of sodium is 1.96%. Therefore, we believe that AKI and rhabdomyolysis developed independently due to CO poisoning rather than rhabdomyolysis-induced AKI.

Delayed leukoencephalopathy is a rare complication of ischemic encephalopathy found in patients with CO poisoning.[9] Its pathophysiology is unclear. However, hypooxygenation of the subcortical white matter is believed to interrupt adenosine triphosphate-dependent enzymatic pathway involved in myelin turnover resulting in demyelination.[10] The "leukotoxin" can be a mediator of this response, including CO, alcohol, solvents, and cranial irradiation.[11] Between the level of COHb concentration after CO exposure and delayed onset of leukoencephalopathy, there were no significant correlation.[12] In a small randomized controlled trial, hyperbaric oxygen therapy could not reduce delayed leukoencephalopathy compared to normobaric oxygen therapy.[13] CO poisoning especially induced delayed leukoencephalopathy that can lead to severe sequelae, such as globi pallidi necrosis.[14] No apparent curative treatment was available for leukoencephalopathy. Delayed leukoencephalopathy's prognosis was not correlated with the duration of unconsciousness.[12] If serious complications such as globi pallidi necrosis occur, prognosis can be very poor.[15] The treatment of delayed leukoencephalopathy is focused on symptomatic treatment and rehabilitation. In this case, the patient did not suffer from serious sequelae, such as consciousness deterioration and motor weakness, during the 1-year follow-up, which was very fortunate.

4. Conclusion

CO poisoning may cause delayed leukoencephalopathy. Therefore, a series of follow-up and monitoring of neuropsychological sequelae are essential. In patients with late neuropsychological symptoms or signs, brain MRI can help diagnose leukoencephalopathy. We report a case of delayed leukoencephalopathy after AKI and rhabdomyolysis as acute complications of CO poisoning.

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Validation: Seong Gyu Kim, Gun Woo Kang.
Writing – original draft: Seong Gyu Kim, Gun Woo Kang.
Writing – review & editing: Seong Gyu Kim, Gun Woo Kang.

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