

## An Unusual Cause of Rhabdomyolysis: Acute Carbon Monoxide Poisoning

Rabdomiyolizin Nadir Bir Sebebi: Akut Karbon Monoksit Zehirlenmesi

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### Abstract

Carbon monoxide (CO) is a toxic gas produced by the incomplete combustion of carbon-containing compounds. Exposure to high concentrations of CO can be lethal and is the most common cause of death from poisoning worldwide. Rhabdomyolysis caused by CO poisoning is uncommon. Early recognition of rhabdomyolysis in such cases is important, since this complication can be fatal as it leads to acute renal failure. However, the prognosis is excellent if proper medical management is provided. In the present study, we discuss rhabdomyolysis caused by CO poisoning in a 68-year-old man.

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**Key words:** Carbon monoxide poisoning, carboxyhemoglobin, rhabdomyolysis

### Özet

Karbon monoksit (CO); karbon ihtiva eden bileşiklerin tam yanmaması ile meydana gelen toksik bir gazdır. CO'ye yüksek konsantrasyonlarda maruz kalmak öldürücü olabilir ve dünya genelinde zehirlenme ölümlerinin en sık nedenidir. CO zehirlenmesinin sebep olduğu rabdomiyoliz nadirdir. Böyle olgularda rabdomiyolizin erken tanınması önemlidir. Çünkü bu komplikasyon akut böbrek yetmezliğine yol açarak ölümcül olabilir. Ancak, uygun tıbbi yönetimi sağlanırsa, prognozu mükemmeldir. Bu çalışmada, CO zehirlenmesi nedeniyle rabdomiyoliz gelişen 68 yaşındaki bir adamı değerlendirdik. (*JAEM 2013; 12: 43-5*)

**Anahtar kelimeler:** Karbon monoksit zehirlenmesi, karboksihemoglobin, rabdomiyoliz

### Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless, and non-irritant gas produced primarily as a result of incomplete combustion of any carbonaceous fossil fuel. The clinical symptoms of CO poisoning are diverse and easily confused with many diseases, such as viral illness, benign headache, and various cardiovascular and neuropsychiatric syndromes (1). Exposure to high concentrations of CO can be lethal and is the most common cause of death from poisoning worldwide (2). CO poisoning can cause a wide variety of clinical effects in humans, but rhabdomyolysis due to CO poisoning is extremely rare. We present a case of CO poisoning that resulted in rhabdomyolysis, and discuss the implications of this complication in the management of patients poisoned with CO.

### Case Report

A 68-year-old man was admitted to our emergency department with altered consciousness as a consequence of acute domestic exposure to CO from a stove. Upon preliminary evaluation, he was unconscious, his Glasgow Coma Scale score was 8 (M4, V2, E2), body temperature was 37.5°C, pulse rate was 115 beats per minute (bpm),

respiratory rate was 22 breaths per min, and blood pressure was 158/80 mmHg. According to the information acquired from his relatives, he had no other diseases and did not use any drugs.

A pulse oxymetric measurement performed using a signal extraction pulse CO-oximetry device (Masimo Co., USA) revealed a serum carboxyhemoglobin (COHb) level of 47%. Oxygen treatment was started immediately. Hyperbaric oxygen therapy was planned for the patient, but hyperbaric oxygen was not available either at our hospital or at another local hospital. Therefore, we used therapeutic red cell exchange (TRES) by automated aphaeresis to treat our patient. The TRES lasted 45 minutes and used 12 packed red cells (each packed red cell is approximately 400 mL in volume). The COHb level was reduced to 12% after TRES and the Glasgow Coma Scale score increased to 12 (M5, V4, E3). His Glasgow Coma Scale score was 15 by the tenth hour after TRES. Physical examination at this time identified weakness in the arms and legs, and the weakness progressed over the next 7 days; muscle strength returned within the next 35 days with the aid of physiotherapy.

The preliminary blood analysis revealed a white blood cell count (WBC) of 24.1  $10^3/\mu\text{L}$ , a hemoglobin (HGB) level of 15.9 g/dL, potassium 5.8 mmol/L, calcium 7.6 mg/dL, creatine kinase (CK) 41774 U/L, creatine kinase MB isoenzyme (CK-MB) 1117 U/L, troponin T 0.549

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ng/mL, creatinine 1.26 mg/dL, urea 74 mg/dL, aspartate aminotransferase (AST) 345 U/L, alanine aminotransferase (ALT) 202 U/L, and lactate dehydrogenase (LDH) 1135 U/L. Other complete blood cell count parameters, coagulation parameters, and biochemical values were normal. The urinalysis showed a pH of 6. The urine was dark brown in color, and showed 1+ protein and 8-9 red blood cells (RBCs) per high power field. However, all other parameters were negative. The urine and blood myoglobin levels were not measured due to the unavailability of a laboratory kit. These findings suggested to us that rhabdomyolysis had occurred due to CO poisoning. Intravenous crystalloid (300 mL/h) was administered immediately, and two ampoules of sodium bicarbonate were added to each liter of normal saline. In addition, a 20% mannitol infusion (0.5 g/kg) was given over a 15-minute period and subsequently followed by an infusion at 0.1 g/kg/h.

The patient was managed with optimum rehydration and forced alkaline diuresis. He improved rapidly and made a complete recovery, with all laboratory parameters normalized by the twelfth day (Table 1). He was discharged with a follow-up physiotherapy program after twelve days of hospitalization.

## Discussion

Carbon monoxide is a toxic gas produced by the incomplete combustion of carbon-containing compounds. Sources of poisoning include, but are not limited to, accidental exposure to high doses of CO through indoor burning of charcoal for barbecues or heating, automobile exhaust, industrial solvents, and gasoline-powered generators (1).

Carbon monoxide binds to hemoglobin with an affinity more than 200 times that of oxygen and causes a leftward shift in the oxygen-hemoglobin dissociation curve. This shift, combined with CO inhibition of cytochrome P450-mediated cellular respiration, produces tissue hypoxia, anaerobic metabolism, and lactic acidosis (1, 3). The non-specific symptoms after CO exposure include headache, nausea, vomiting, palpitations, dizziness, and confusion. As exposure increases, patients develop more pronounced and severe symptoms, with highly oxygen-dependent organs showing the earliest signs of injury. A high index of suspicion is essential for making a diagnosis of occult CO poisoning. Serum COHb levels should be obtained from patients suspected of CO poisoning (1, 3). The preliminary evaluation indicated that our patient was unconscious, with a Glasgow Coma Scale score of 8 (M4, V2, E2). He was diagnosed with CO poisoning based on the information acquired from his relatives and on his increased COHb level.

The standard treatment for CO poisoning is administration of 100% oxygen and should be administered until a normal carboxyhemoglobin level is restored (3). Typically, hyperbaric oxygen (HBO) is used to treat patients with severe CO poisoning; however, this intervention may not be available in most places. In these cases, TREX is an alternative treatment method (4). In our study, 100% oxygen treatment was started immediately, but hyperbaric oxygen was not available at our hospital or another local hospital, so we used TREX to treat our patient.

**Table 1.** Trend in laboratory parameters over time

	Day 1	Day 3	Day 6	Day 9	Day 12
CK	41774 U/L	31030 U/L	8335 U/L	1734 U/L	196 U/L
AST	345 U/L	267 U/L	206 U/L	79 U/L	24 U/L
LDH	1135 U/L	1297 U/L	1173 U/L	825 U/L	239 U/L

LDH: Lactate dehydrogenase; CK: Creatine kinase; AST: Aspartate aminotransferase

Carbon monoxide poisoning may result in rhabdomyolysis, potentially as a direct toxic effect of CO on skeletal muscle. Rhabdomyolysis is a potentially life-threatening syndrome characterized by the breakdown of skeletal muscle. It results in the release of large amounts of muscle cell contents into the circulation. These cell contents include enzymes such as CK, AST, LDH, aldolase, the heme pigment myoglobin, and electrolytes such as potassium and phosphates. Rhabdomyolysis is most often caused by muscular trauma or crush injuries (5). Other significant causes of rhabdomyolysis include drugs and toxins, excessive muscular activity, extremes in temperature, muscle ischemia (CO poisoning), prolonged immobilization, infections, electrolyte and endocrine abnormalities, genetic disorders, and connective tissue disorders (5). In the past, the most common causes of acute rhabdomyolysis were from crush injuries during wartime and following natural disasters. More recently, as noted in one published series, drugs and alcohol have become frequent causative agents in up to 81% of cases of rhabdomyolysis (6). The classic presentation of rhabdomyolysis is clinically characterized by muscle weakness and pain and, at times, tea-colored urine which is non-specific and may not always be present (5, 7). The diagnosis therefore rests upon the presence of a high level of suspicion of any abnormal laboratory values in the mind of the treating physician. The key to making the diagnosis is to detect increased creatine kinase activity in the plasma or myoglobin in the urine. In addition, hyperkalemia, hypocalcemia, and increased AST, LDH, and aldolase activities may be present. The management of the condition includes prompt and aggressive fluid resuscitation, elimination of the causative agent(s) and the treatment or prevention of any complications that may ensue (5).

Extensive rhabdomyolysis may cause acute renal failure unless treated immediately. Acute renal failure is the most serious complication occurring in the days following the initial presentation of rhabdomyolysis (7). The treatment for rhabdomyolysis is intense rehydration and forced alkaline diuresis (5, 7). In our study, the patient's potassium, calcium, CK, AST, and LDH levels were increased, but the urine and blood myoglobin levels and serum aldolase activity were not measured due to the unavailability of a laboratory kit. These results suggested to us that rhabdomyolysis had occurred due to CO poisoning. Intravenous crystalloid (300 mL/h) was started immediately, and two ampoules of sodium bicarbonate were added to each liter of normal saline. The patient was treated with intense rehydration and forced alkaline diuresis for twelve days.

Previous reports of rhabdomyolysis due to CO poisoning are extremely rare. The current report shows that CO poisoning may cause severe rhabdomyolysis (8-11). The mechanisms of CO-induced rhabdomyolysis are as follows: I) decreased hemoglobin carrying capacity of oxygen; II) CO-inactivated cytochrome oxidase, which leads to an inability to meet the energy requirements necessary to maintain aerobic respiration of working muscle cells, and III) CO interference with the normal binding of oxygen to myoglobin, which is an essential oxygen reservoir within muscle tissue (11).

## Conclusion

Rhabdomyolysis is a serious complication of carbon monoxide poisoning. It may lead to acute renal failure unless treated immediately. Early recognition of rhabdomyolysis is important; however, the prognosis is excellent, as our case report has illustrated, if proper

medical management is provided. The follow-up period of patients should include daily measurements of serum CK, AST, LDH, and electrolyte levels.

#### Conflict of Interest

No conflict of interest was declared by the authors.

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