

Complications of carbon monoxide poisoning

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ABSTRACT

Objective: Acute carbon monoxide poisoning is a common problem that occurs during winter and leads to serious complications.

Methods: We retrospectively studied 24 consecutive cases admitted with the aim of finding the causes and outcome of acute carbon monoxide poisoning.

Results: The source of poisoning was charcoal in 71% of patients, motor gasoline in 21%, and other causes in 8%. Immediate complications included altered consciousness level in 54% of patients, metabolic acidosis in 46%, pneumonia in 42%, cardiac arrhythmia in 29% and rhabdomyolysis in 25%. Late neurological

complications occurred in 17% of patients. All the patients received 100% oxygen. Eleven patients (46%) required mechanical ventilation. Ultimately, 19 patients (79%) recovered completely, 4 (17%) had neurological or cardiac disorders, and 1 (4%) died.

Conclusion: Immediate and late complications are common in carbon monoxide poisoning cases admitted to the hospital especially when they arrive late. Time lapse between exposure and presentation may have a role in predicting the outcome.

Keywords: Carbon monoxide, carboxyhemoglobin.

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Carbon monoxide (CO) is the most common and serious by-product of combustion and responsible for smoke-related morbidity and mortality.^{1,2} As it is colorless, odorless, tasteless and non-irritating gas, the exposed person is usually unaware of its effect until serious disorders occur.³ Poisoning with this gas does not frequently occur inside Saudi houses, as they are usually equipped with modern heating systems. However, during wintertime, Saudis like to remember their tradition and spend some nights inside closed tents or special-designed villas (Bungalows) in the desert where alternative sources like stove, wood, or charcoal are the usual heating methods. These sources are also relatively cheap and used commonly by poor people who can not afford expensive heating systems. Such

places are closed and not adequately ventilated in order to keep the environment warm. In our hospital, we noticed every winter that a number of patients are admitted with serious acute CO poisoning. Therefore, we retrospectively studied all the cases of acute CO poisoning admitted to our hospital over a 9-year period with the aim of finding the factors associated with increased risk of poisoning and the outcome of these patients.

Methods. All patients who were admitted to our hospital with a diagnosis of acute CO poisoning between April 1986 and May 1995 (9 years) were included in this study. Our hospital is a tertiary care teaching hospital with a capacity of 550 beds. To

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identify the cases, we used code 986 from the international classification of disease.⁴ Twenty-four charts were identified and retrospectively reviewed. The data collection sheet included the following information: source of CO, time lapse between the exposure and arrival to Emergency Department (ED), need for urgent intubation and mechanical ventilatory support, immediate complications, and late complications that persisted until the time of discharge. The level of carboxyhemoglobin on the initial arterial blood gases was recorded. All laboratory parameters were assessed and the radiographs reviewed especially brain computed tomography (CT) and magnetic resonance imaging (MRI) scans, whenever available. Numbers were expressed as mean \pm standard deviation. The Mann-Whitney test was used to compare distribution of continuous variables across 2 groups. Whenever there were data in 2x2 table, chi square test was used.⁵

Results. Twenty-four cases were identified over the study period. The main age was 35.5 years (± 12.5) with a male:female ratio of 3.8:1. The source of acute CO poisoning was charcoal heater in 17 patients (71%), gasoline motor in 5 (21%), fire smoke in 1 (4%), and sheeshah (local water pipe smoking device, Hubbell-bubble) in 1 (4%). Two groups were identified based on the time lapse between exposure and arrival to ED. Seven cases (29%) arrived in less than 5 hours and 17 patients (71%) arrived after 5 hours or the time lapse was unknown. The age for the first group was 31.0 years (± 10.1) and for the 2nd group was 33.2 years (± 13), $P=0.56$. Carboxyhemoglobin level upon arrival to ED was 30% (± 13.5). The level was 32% (± 16.1) for the first group and 30% (± 13) for the second group, $P = 0.62$. Table 1 shows immediate complications secondary to CO poisoning. Late neurological complications have occurred in 4 patients (17%) in the form of dementia (difficulties in orientation and memory) and Parkinsonian Syndrome. Residual cardiac ischemia was noticed in 1 patient (4%).

Table 1 - Immediate complications secondary to carbon monoxide poisoning.

Immediate complications	Number	Percentage
Altered level of consciousness	13	54%
Metabolic acidosis	11	46%
Pneumonia	10	42%
Cardiac arrhythmia	7	17%
Rhabdomyolysis	6	25%

Computerized tomographic findings in the patients with neurological complications consisted mainly of basal ganglia necrosis and cortical atrophy. All late complications occurred in those who arrived to ED in more than 5 hours or their time lapse was unknown.

All patients received 100% oxygen by a non-rebreather mask upon arrival to ED. Eleven patients (46%) required intubation and mechanical ventilation, one patient belonging to the first group (8%) and 10 belonging to the second group (59%). Due to the unavailability of hyperbaric oxygen (HBO) in our hospital, only 4 patients (17%) were managed with this modality in another hospital. All the patients who received HBO were from the second group. Of the 24 patients admitted to our hospital, 19 (79%) recovered completely, 4 (17%) had late neurological sequelae or cardiac ischemia, and 1 (4%) died despite intensive therapy.

Discussion. Carbon monoxide causes poisoning by its high affinity to hemoglobin and its ability to replace oxygen which renders the hemoglobin to be useless in terms of its oxygen carrying capacity.⁶ Carbon monoxide also alters the molecular configuration of hemoglobin and decreases 2-3 DPG of red blood cells, resulting in unfavorable left shift of the oxyhemoglobin dissociation curve.⁶ The other major toxic effect of CO is the binding of CO to the iron variety of cytochrome (Cytochrome a, a₃).^{1,7} Carbon monoxide also binds to other hemoproteins and myoglobin. There is some evidence that CO causes direct myocardial toxicity which would explain acute cardiac insult in patients exposed to CO poisoning.^{3,7} This shows the importance of the 'tissue level' of CO, which unfortunately cannot be assessed though it has a major role on tissue toxicity and the occurrence of late complications.^{1,6} The half-life of CO in the body is approximately 4-5 hours and this can be reduced to 60-80 minutes by giving 100% oxygen, or to approximately 25 minutes by HBO.² At the cellular level, the cytochrome oxidase enzymes have greater affinity for oxygen than for the CO. Hence, by supplying a high concentration of oxygen in victims of CO poisoning, CO can be replaced by oxygen and tissue hypoxia can be relieved.⁷

The main sources of CO poisoning in Saudi Arabia are found to be charcoal and motor gasoline which is different from the sources found in the western countries (motor vehicles, smoke from fire or fumes, or from faulty heating systems).⁸⁻¹⁰ Carbon monoxide level was not a significant determinant of outcome in our study, a finding similar to previous studies.¹¹ Carbon monoxide level is certainly important in diagnosing CO poisoning but does not predict the immediate or late sequelae. Patients with CO poisoning need careful initial evaluation and attention to disease progression. The documentation

of the time lapse between exposure and presentation to the ED needs to be documented as it is significantly associated with outcome. Even though there are no controlled studies to document the benefit of HBO in acute CO poisoning in terms of morbidity or mortality, HBO leads to faster reduction in CO levels.⁷ However, there are conflicting results by various authors on the long-term benefits of HBO on late neurologic sequelae.¹³ Patients with late neurological sequelae had abnormal CT brain in the form of basal ganglia necrosis and cortical atrophy. The finding in our study is consistent with data presented by Silver and colleagues where they found seven of 18 patients with CO poisoning had abnormal brain CT. It occurred in the form of hypodensity in the globus pallidus and the white matter areas.¹⁴ Such a finding can be avoided by proper application of HBO.^{12,15}

In conclusion, immediate and late complications are common in carbon monoxide poisoning cases admitted to the hospital especially when they arrive late. Time lapse between exposure and presentation may have a critical role in predicting the outcome.

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