

Acute Carbon Monoxide Intoxication. A Case Study in Laboratory Practice and Postmortem Diagnostics

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Abstract

Introduction: The Carboxyhemoglobin (COHb) study is fundamental to the diagnosis of carbon monoxide poisoning, even after death, and to clarify the cause of intoxication.

Aim: The aim is to present a case of a person dying of an acute (fulminant) form of carbon monoxide (CO) poisoning that has occurred under unusual conditions.

Case Report: We present the case of a 44-year-old man who was removed dead from a vertical hole, nailed to the rope by which he descends into the hole. Illegally TNT an explosion was triggered the previous day to widen the opening. The man was pulled dead by firefighters, 2 hours after the descent. The Dräger X-AM 5000 measured the presence of toxic gases in the air at a depth of 10 meters. The blood levels of HbCO were determined spectrophotometrically on a CO-oximeter ABL 800 (Radiometer).

Results: The CO values in the rock cavity air exceed the dynamic range for CO measurement ($>> 2290 \text{ mg/m}^3$). An increase in the levels of other toxic gases is observed (methane and hydrogen cyanide). The amount of COHb in corpse blood was 77.3%. At these high values, acute tissue anoxia and block oxidation processes in the cells occurred. CO is a cell poison.

Conclusion: The leading cause of death is carbon monoxide poisoning resulting from very high levels of CO in the inhaled air. The study of COHb levels with the ABL 800 CO oximeter is a reliable method for detecting CO intoxication in live and dead individuals.

Keywords: CO; COHb; Co Oximetry; Oxygen; Methane; Hydrogen Cyanide

Abbreviations

COHb: Carboxyhemoglobin; O₂Hb: Oxyhemoglobin; CO: Carbon Monoxide; CH₄: Methane; HCN: Hydrogen Cyanide; Hb: hemoglobin

Introduction

Carbon monoxide (CO) is a colourless gas, odourless, lighter than air, easily combustible. It is one of the most widespread atmospheric pollutants. It is formed by incomplete combustion and is an integral part of the flue gases produced in industrial plants, explosive gases, car exhausts, tobacco smoke and more. The air in big cities is characterised by a high level of CO [1]. The Carbon monoxide enters the body by inhalation. It has about 200 - 290 times greater affinity for binding to haemoglobin (Hb) than O₂ [2,3]. The created bond is covalent and difficult to break (under hyperbaric conditions only) [4]. The dissociation of carboxyhemoglobin is 3600 times slower than

oxyhemoglobin (O₂Hb), with a half-life of 4 - 5 hours under normobaric conditions [5]. In our previous study, we found that there was no significant change in the concentrations of COHb when the sample was stored well-closed until seven days. In the range of 0 - 60% COHb, the values in arterial (COHba) and venous blood (COHbv) are similar, i.e. it does not matter what type of sample will be analysed [6]. The formed COHb turns Hb into an inactive form to connect O₂, resulting in hypoxia in the body. CO is a blood and cell poison. It also blocks the enzyme chains in the cell (cytochrome and cytochrome oxidase), which are involved in cellular respiration and the flow of vital intracellular metabolic processes. Caused direct and indirect hypoxia leads to rapid clinical manifestations of the Central Nervous System and the cardiovascular system, which are most sensitive to ischemia. Severe poisoning leads to marked hypotension and deadly arrhythmias, which thought to be responsible for a large number of deaths. COHb over 12 - 15% show signs of intoxication and over 65%, the poisoning is fatal. Usually, fatal exposure to CO causes coma, convulsions and cardiorespiratory arrest.

Clinical Case

We present the case of a 44-year-old man who was removed dead from a vertical hole in a rock, tied to a rope with which it was descended. He was probably looking for a hidden treasure. The previous day, it is caused an explosion in the gap with the TNT to widen the opening. The participants in the incident are two men. One helped with the descent and stayed upstairs. After 5 - 6 minutes, reaching almost to the bottom of the hole, the lowered man began to signal and make sounds. He probably had difficulty breathing. The man upstairs was not able to subtract it alone. The body was removed after 2 hours by firefighters.

The forensic report states that the deceased's face is hyperemic with an enlargement at the conjunctival vessels.

The next day, the air in the hole for toxic gases was measured with the Dräger X-AM 5000. The detector is lowered to a depth of 10 - 11 meters, almost to the bottom. At the very bottom, there is water, which when illuminated with a spotlight shows that bubbled. The instrument is kept for about 2 minutes. The measured concentration levels are given in table 1.

Methane (CH ₄)	Oxygen (O ₂)	Carbon monoxide (CO)	Hydrogen cyanide (HCN)
6%	16.5%	↑↑↑	8.3 mg/m ³
*0 - 100%	*0 - 25 vol%	*0 - 2000 ppm	*0 - 55 mg/ m ³

Table 1: Concentration levels of measured toxic gases with the Dräger X-AM 5000.

**The following line lists the measurement ranges for the respective gases according to the manufacturer's specifications.*

The carcass sample obtained for the study was taken on the day of death in a vacuum container with the addition of Li Heparin anticoagulant. It was delivered to the laboratory well packaged and tested on the fourth day. Visually, the blood is bright red (crimson red) with a liquid consistency and a blood-like viscosity. The results obtained when examining the biological material using the ABL 800 (Radiometer) CO-Oximeter are given in table 2.

ctHb	Ht	O ₂ Hb	COHb	HHb	MetHb
132 g/L	0,405 L/L	10.0%	77.3%	7.0%	5.7%

Table 2: Haemoglobin levels and haemoglobin derivatives in the carcass sample.

The measurement of Hb fractions is based on the principle of an optical system. At its base is a spectrophotometer with wavelengths in the measuring range 478 - 672 nm. The various haemoglobin derivatives have specific absorption spectra. The spectrophotometer is coupled via optical fibre to a combined hemolysator and measuring chamber. CO-oximetry is widely used in laboratory practice, but mainly for the analysis of blood from living donors. In this case, we use a carcass blood sample. Several studies have shown that this is an

appropriate method for measuring COHb and post-mortem. But after death, the biological sample undergoes decay processes. The turbidity associated with this is a potential interference when measuring COHb. Higher values of MetHb [9] and/or SulfHb [7] are measured in carcass samples determined by CO-oximetry. In the blood of the carcass, haemoglobin values may be too low due to clotting and hemolysis [7]. If ctHb is considerably below that in the living organism, the accuracy of CO-oximetry is potentially reduced [7]. A reference method for the determination of COHb is gas chromatography (GC). Comparative analysis of the results obtained with CO-oximetry and those of GC revealed that CO-oximetry could be a reliable method for measuring COHb after death, even with the values of ctHb in the sample being 10 g /L [8].

Measuring with the Dräger X-AM 5000 provides valuable information on the composition of the air that breathed the deceased. In O₂-poor environment (measured by the Dräger value of -16.5%), the toxic effect of carbon monoxide (CO) is multiplied. There is no doubt that the cause of death is carbon monoxide poisoning (COHb 77.3%). The average background levels of CO in the air are 0.05 - 0.12 ppm [1]. CO exposure may not exceed ten ppm. In our clinical case, the concentration of CO in the air is too high, exceeding the dynamic detection limits of the detector (0 - 2000 ppm or 2290.39 mg/m³). There is a relationship between CO exposure and equilibrium concentrations of HbCO [9]. The Coburn-Forster-Kane (CFK) model predicts blood COHb levels. However, at very high COHb values, the simulation model introduces significant errors from the limited Hb binding capacity. At the measured levels of saturation of HB with CO, the concentrations of CO in the air are most likely to be above 4000 - 5000 mg/m³ [10]. The unconscious state and lethal outcome occurred rapidly, fulminant due to the combination of high concentrations of CO in the highly hypoxemic environment. The International Organization for Standard (ISO) publishes lethal exposure concentrations for CO of 12,000-16,000 ppm for 5 min and 2500 - 4,000 ppm for 30 minutes (values are based on data from animal experiments) [5].

Discussion

Haemoglobin (HB) is a chromoprotein whose primary role is the transport of oxygen and carbon dioxide to the tissues and back to the lungs. Hb in the blood of a healthy person, when leaving the lungs (when breathing) and in the arterial blood, is close to 100% oxygenated. About 95 - 98% is present as oxyhemoglobin (O₂Hb), and about 2 - 5% is present as reduced haemoglobin (HHb). In venous blood, the amount of HHb is much higher, reaching up to 30 - 40%. In addition to O₂Hb and HHb, blood usually contains traces (not more than 1 - 2%) of two more types of haemoglobin - carboxyhemoglobin (COHb) and methemoglobin (MetHb) [7].

COHb in non-smokers is up to 1.5%. This amount is present in the blood due to endogenously produced CO during heme catabolism [11]. Cigarette smoke contains carbon monoxide. Smokers are exposed to approximately 400 - 500 ppm CO. According to the daily use of cigarettes and the duration of smoking, COHb can reach 6.5 - 8% [5]. These values have no clinical manifestation, except for the occurrence of several pathophysiological processes associated with an increased risk of ischemic cardiac, cerebral and vascular accidents and carcinogenesis [12]. The first signs of CO poisoning are observed with HbCO above 12 - 15% [13]. There is an individual sensitivity to CO. The most sensitive are adolescents, women and patients with anaemia, lung and heart disease. Prenatal exposure leads to damage to the fetus.

COHb (%)	Symptoms
10	No appreciable effect, except shortness of breath on vigorous exertion, possible tightness across the forehead, dilation of cutaneous blood vessels
20	Shortness of breath on moderate exertion, occasional headache with throbbing in temples
30	Decided headache, irritable, easily fatigued, judgment disturbed, possible dizziness, dimness of vision
40 - 50	Headache, confusion, collapse, fainting on exertion
60 - 70	Unconsciousness, intermittent convulsion, respiratory failure, death if exposure is long continued
80	Rapidly fatal

Table 3: COHb-related symptoms in healthy elderly.

Source: Adapted from WHO 1999a.

Based on the results obtained from the Dräger X-AM 5000 and blood test, the effects of other toxic gases present in the air also discussed.

The air we breathe contains 21% O₂. The typical composition of the inhaled air is about 21% O₂, 78% N₂ and 2.0% CO₂. Approximately 0.5 L of air passes through each breath deep into the lungs. The atmospheric content of gases varies with altitude (altitude above 10000 m nitrogen is 86%, and oxygen is 19%) [14]. Values below 19% and above 23% are hazardous to humans [15]. The first case is severe hypoxic hypoxia. The most sensitive to oxygen starvation are the central nervous system, the retina, heart, and to a lesser extent, the cells of the parenchymal organs. At O₂ -8% values, there is a loss of consciousness up to 5 minutes. When O₂ is above 23%, it is explosive and toxic to the body. In our case, the combined effect of low O₂ values (16.5%) and high CO levels are factors that enhance the harmful influence of inhaled air. Studies in experimental animals show that the smaller the oxygen level in the air, the faster the death from CO intoxication [16].

Methane (CH₄) is formed by the decomposition of plant materials in the absence of oxygen in marshy places, mines, which is why it is also called marsh gas. Methane levels are usually low in the atmosphere (about 2.2 ppm) [17]. Methane is non-toxic but extraordinarily flammable and can explode at concentrations between 5% (lower) and 15% (upper explosive limit). The measured values in the hole (6%) are far from those of explosion. Most likely, the water in the hole (bubbling) is a methane generator. Including the explosion produced a number of gas mixtures. Methane is classified as a pure asphyxiant gas when present in high concentrations in the air. A pronounced toxic effect of CH₄ has values above 50 - 60% [18]. Methane reduces the amount of oxygen available, displacing it and can cause suffocation (at O₂ < 19%). High levels of methane can provoke mood swings, incomprehensible speech, vision problems, memory loss, nausea, vomiting, flushing, and headaches [18]. There is no particular test for blood methane levels. The most important is exposure control.

Hydrogen cyanide (HCN) is a colourless, fast-acting, poisonous gas with the characteristic odour of bitter almonds. The smell is at levels of 0.6 - 5 mg/m³. The presence of cyanide in the body leads to the inhibition of cellular respiration and hypoxia in tissues with the fastest oxygen metabolism, such as cardiac muscle and brain [19]. Maximum permissible industrial exposure concentration is up to 4 - 6 mg/m³ no more than 1 hour/day. Values of about 8 - 10 mg/m³ at exposure for 1 hour only cause a mild headache [20]. Although HCN is a highly toxic cellular poison (blocking cytochromes of the respiratory chain in cells), airborne measured values (8.3 mg/m³) could not be the cause of death. A review of human fatal cases (ATSDR 1997) indicated that exposure to HCN air concentrations at 180 - 270 mg/m³ was deadly within minutes. The measured values in the air with the Dräger X-AM 5000 are 20 - 30 times lower.

Conclusion

In the current case, the major cause of death is carbon dioxide poisoning resulting from very high levels of CO in the inhaled air. The explosion and the flue gas in an oxygen-poor environment may have helped to increase carbon monoxide many times over. The presence in the gas mixture of other toxic gases at higher concentrations (though far from deadly ones) and hypoxemia are a potent and additional factor in the fulminating course of intoxication and death of the face.

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