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Carbon Monoxide Intoxication: Experiences from Hungary

Edit Gara

Abstract

Carbon monoxide (CO) is odorless, colorless, tasteless, and nonirritating gas. Hence, mild CO poisoning often remains unrecognized and appears lethally. Carbon and gas systems, unfavorable architectural designs and machines may also cause intoxications. The prevalence rates in Hungary ranged from 2.37 to 3.80 cases per 100,000 people per year between 2013 and 2015; fatality rates have been decreased from 5.96 in 2013 to 3.38 in 2015. Given the vagueness and the broad spectrum of complaints, misdiagnosis of CO toxicity is common. The gold standard diagnosis is detecting the level of circulating carboxyhemoglobin (CO-Hgb). The measurement of CO-Hgb can be performed via blood-gas analyses or by spectrophotometry. Treatment protocol should follow the ACBDE rule. Administration of 100% oxygen should be performed as soon as possible. Later in-hospital management includes evaluation, treatment and prevention of further peripheral organ damage and long-term neurological complications. Fetuses and children are prone to suffer more severe intoxication due to higher oxygen demand. Though hyperbaric oxygen is the mainstay therapy, a prompt cesarean section is effective in preventing further intoxication. In conclusion, fatal CO intoxication can occur due to plain early signs and symptoms. Hyperbaric oxygen therapy should be considered in severe intoxication, in fetal and children.

Keywords: carbon monoxide poisoning, pathophysiology, differential diagnose, intoxication in pregnancy, treatment options, prevalence in Hungary

1. Introduction

Carbon monoxide (CO) is an odorless, colorless, tasteless, and nonirritating gas product of carbon and gas combustion. CO is also present in cigarette smoke and vehicle combustion

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gas. CO diffuses through general building constructions (brick and wood). Generally, atmospheric concentration of CO is low, however, in urban and industrial regions it may be elevated. Poisoning usually occurs via impaired operating heating and mechanical systems and fire emergencies.

CO enters human body via breathing and gas exchange, furthermore CO is also generated endogenously in small amounts during oxygen consumption in healthy subjects. The hemoxidase enzyme, responsible for the catabolism of hemoglobin into biliverdine, is responsible for endogenous CO production.

CO has higher affinity in binding to hemoglobin then oxygen, thus development of carboxyhemoglobin (CO-Hgb) is responsible for the major signs and symptoms, and late complications associated with CO intoxication such as cardiovascular and neurological complications due to hypoxia which are often lethal.

The aim of this chapter is to summarize the toxicokinetics, epidemiology, pathophysiology, signs and symptoms, diagnosis, differential diagnosis and treatment options of carbon monoxide poisoning. In doing so, a case report involving a whole family that suffered severe carbon monoxide intoxication will be presented including the story of a successful management of a pregnant woman (one of the family members) who underwent an urgent cesarean section to protect the baby from carbon monoxide intoxication. This will help to elaborate on treatment options to be carried out pre- and during hospitalization; and discuss the merits of hyperbaric oxygen therapy.

2. Toxicokinetics of carbon monoxide

CO is formed as a by-product of burning organic compounds; that is why poisoning by CO is common during power outages due to storms, as a result of the improper use of gasoline-powered portable generators to provide electricity and indoor use of charcoal briquettes for cooking and heating. Other sources of CO include improperly vented gas water heaters, kerosene space heaters, charcoal grills, malfunctioning or obstructed exhaust systems stoves, portable heaters, fires, cigarette smoke, and automobile exhausts.

In a healthy adult alveolar CO concentration, during one cigarette smoke is increased to 400–500 ppm. Nonsmoker individual in the same room is also exposed to CO, resulting 25–100 ppm alveolar concentration [1].

CO is absorbed and eliminated through the lungs. The amounts inhaled and exhaled are dependent on the alveolar-capillary pressure gradient of oxygen and alveolar diffusion. CO intoxication also occurs by inhalation of methylene chloride vapors, a volatile liquid found in degreasers, solvents, and paint removers. The liver metabolizes as much as one-third of inhaled methylene chloride to CO. A significant percentage of methylene chloride

is stored in the tissues, and continued release results in elevated CO levels for at least twice as long as with direct CO inhalation. The half-time of CO in a healthy adult takes 4 hours breathing on air, 1.5 hours breathing on 100% oxygen, and 20 minutes in hyperbaric oxygen circumstances [2].

3. Prevalence of carbon monoxide poisoning in Hungary

Carbon monoxide intoxication cases have been documented in Hungary from 2012. Hence, as shown in **Figure 1**, the total number of cases was, respectively, 235 in 2013, 375 in 2014, and 355 in 2015.
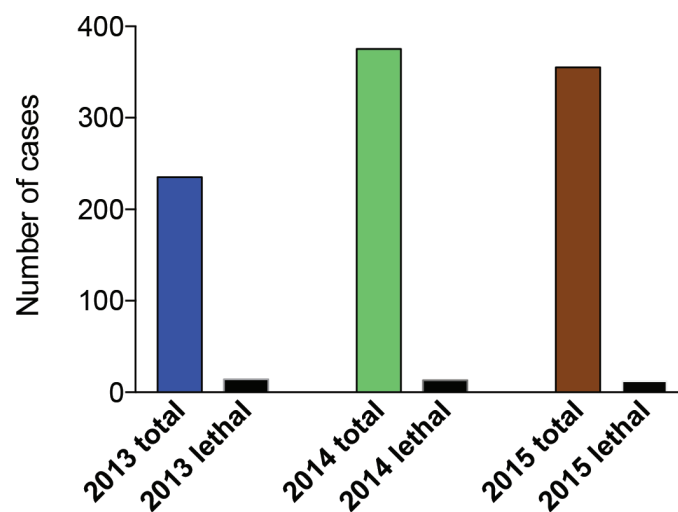

Figure 1. Prevalence of carbon monoxide poisoning in Hungary. Figure shows total number of cases and lethal cases in 2013, 2014, and 2015.

Parameters	2013	2014	2015
# Cases	235	375	355
# Population	9,910,000	9,880,000	9,860,000*
# Cases/100,000 people	2.37	3.80	3.60
# Deaths due to CO	14	13	12
Case fatality rate	5.96	3.47	3.38

*Source: Hungarian Central Statistics Office.

Table 1. Rates of CO poisoning and deaths from 2013 to 2015.

These figures translate to 2.37–3.80 cases per 100,000 people per year. Since, the number of deaths from CO poisoning were, respectively, 14 in 2013, 13 in 2014, and 12 in 2015, the case fatality rates have been decreasing from 5.96 in 2013 to 3.38 in 2015 (**Table 1**). These figures when compared to other countries such the USA, they appear to below [3].

The decrease in the case of fatality rates can be explained not only by the decreasing population, but also by the efforts of the Hungarian National Ambulance that successfully initiated a widespread campaign to increase awareness of CO intoxication. In this project, education focuses on safe handling of household heating systems, importance of regular, controlled, and authorized servicing of equipment and the use of CO level detector devices.

4. Pathophysiology

Carbon monoxide poisoning leads to impaired oxygen delivery and utilization at the cellular level. In doing so, it affects several organs including the brain, heart, and other organs with the highest oxygen requirement. It causes cellular hypoxia by impedance of oxygen delivery as it reversibly binds hemoglobin, resulting in relative functional anemia. Because it binds hemoglobin 230–270 times more avidly than oxygen, even small concentrations can result in significant levels of carboxyhemoglobin (HbCO).

Several studies have indicated that CO may cause brain lipid peroxidation and leukocyte-mediated inflammatory changes in the brain, a process that may be stopped by hyperbaric oxygen therapy. Studies have demonstrated release of nitric oxide free radicals from platelet and vascular endothelium, following exposure to CO concentrations of 100 ppm. One study suggests a direct toxicity of CO on myocardium that is separate from the effect of hypoxia [4].

It should be noted that the severity of CO poisoning depends on Ref. [5]:

- The time of the exposure.
- The concentration of inhaled CO gas.
- Alveolar-capillary diffusion parameters (e.g., general pulmonology status).
- Accompanied illnesses and general condition of the patient.

Exogenous CO intake is regulated via alveolar-capillary diffusion rate. Alveolar-capillary diffusion is dependent on the permeability of the alveolar membrane and from the hemoglobin concentration of alveolar capillaries. Diffusion efficacy of CO in the alveoli is 80%, similarly as for oxygen. The hemoglobin binding efficacy of CO is 200–250 times greater than those of oxygen. Thus, in the presence of CO, oxygen-hemoglobin binding is impaired, however partial oxygen pressures are normal. Main pathological step behind CO intoxication and the related organ dysfunction is the severe tissue hypoxia, caused by elevated levels of CO-Hgb. The hemoglobin-oxygen dissociation curves are shifted to the left, meaning that less oxygen can bind to Hgb at the same partial oxygen pressure levels and CO-Hgb cannot deliver oxygen to the peripheral tissues effectively (**Figure 2**). Furthermore, CO also has higher binding efficacy
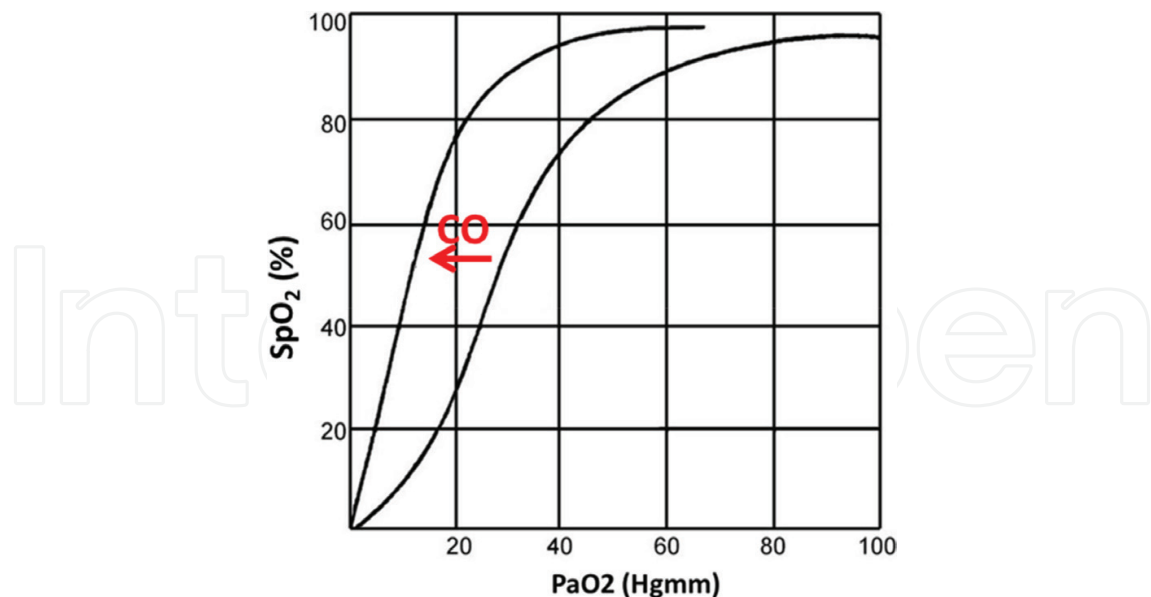
Figure 2. Effects of CO poisoning on hemoglobin-oxygen dissociation curve. In response to CO intoxication the Hgb-O_2 dissociation curve is shifted to the left, physiological sigmoid shape of the curve is impaired, and tissue O_2 delivery is decreased. CO: carbon monoxide, SpO_2%: oxygen saturation, PaO_2: partial oxygen pressure.

to myoglobin than oxygen. This results in more severe peripheral myogenic ischemia and also myocardial ischemia. Partial O_2 pressure is normal, thus forced respiratory responses to compensate tissue hypoxia are lacking. CO also binds to the cytochrome oxidase and NADPH systems at high concentration or long time exposure poisoning. This causes impaired mitochondrial activity and cellular energy development.

5. Signs and symptoms

Signs and symptoms of CO intoxication depend on the CO concentration, thus the CO-Hgb levels. **Table 2** provides a list of general symptoms depending on gas concentration and CO-Hgb levels. Generally, tissue hypoxemia dominates and causes the most severe complications in the cardiovascular and central nervous system. Physical signs of CO intoxication lack unique features. CO-Hgb may cause cherry-pink color on skin, however it only occurs in high levels of CO-Hgb, when the accompanied cyanosis usually lighten cherry-pink sign. Further organ specific signs and symptoms are discussed below.

5.1. Cardiovascular signs and symptoms

Co-Hgb and CO-myoglobin impair myocardial oxygen delivery. Ischemic myocardium is a severe complication in CO poisoning. Clinical features include decreased inotrope function of the left ventricle, accompanied by hypotension and hemodynamic instability. Furthermore, arrhythmogenic complication occurs in one-third of CO-intoxicated patients. Myocardial tissue hypoxia can trigger atrial and ventricular arrhythmias. Malignant ventricular arrhythmia (ventricular tachycardia or ventricular fibrillation) may occur at high CO-Hgb levels, which

CO concentration	Symptoms	CO-Hgb level
35 ppm	Mild headache, vertigo	5–20%
100 ppm	Headache, vertigo, fatigue	
200 ppm	Previous listed above + altered reflexes and cognitive alert	
400 ppm	Previous listed above + nausea, vomiting, tachycardia, tachypnea, palpitation, angina pectoris	30–50%
800 ppm	Previous listed above + arrhythmia, convulsion, respiratory insufficiency, loss of consciousness	
1600 ppm	Convulsions, loss of consciousness, lethal in 2 hours	50–66%
3200 ppm	Convulsions, loss of consciousness, lethal in 30 minutes	
6400 ppm	Loss of consciousness, lethal in 20 minutes	>66%
12,800 ppm	Loss of consciousness after 2–3 breaths, lethal in minutes	

Table 2. Symptoms of CO intoxication in relation with CO concentration and CO-Hgb levels.

may be lethal. Myocardial ischemia may also cause chest pain, angina pectoris and can trigger vegetative reflexes (nausea, vomiting, sweating, and vertigo).

5.2. Neurological signs and symptoms

Loss of consciousness and acute neurological symptoms are due to neuronal hypoxemia. However, after surviving acute intoxication, major studies reported late-onset neuropsychiatric syndrome related to CO poising [6, 7]. This includes change in personality, depressive disorders, cognitive deficits, and psychomotor imbalance. Exact signaling pathways are not yet detailed, general brain hypoxemia/anoxia tends to be responsible for long-term neurological complications.

6. CO poisoning in pregnancy

CO poisoning is rare condition in pregnancy. When occurs, it is a critical condition, the mother and the fetus may suffer severe long-term complications. Due to fetal hemoglobin transport system, the intoxication of the fetus is usually more severe than it is in the mother [8]. Fetal hemoglobin desaturation has higher levels in tissue transport, than adult hemoglobin, even more the fetal gas exchange via the placenta eliminates CO slowly [9]. CO poisoning in pregnancy can cause severe hypoxic/anoxic damage in all fetal organs and tissues. Prenatal injury of the central nervous system may lead to life-long mental and somatic dysfunction, attention deficit disorder or behavioral disorders, due to high sensitivity for hypoxic attacks. Mainly, fetal cortical region and basal ganglions suffer in case of tissue hypoxia [10].

Pathophysiology of CO intoxication of the fetus includes:

1. Maternal O_2 transport via the placenta is impaired in correlation with the elevated CO-Hgb level of the mother

2. CO enters fetal circulation via passive diffusion or facilitated diffusion through the placenta and forms fetal-CO-Hgb, further worsening fetal hypoxemia. Placental CO diffusion is dependent on gestation age and weight of the fetus. In later stage pregnancy, placental diffusion capacity increases and CO intoxication are more severe on the fetus. Many cases presented in the literature lethal, in utero CO intoxication in pregnancies close to terminus [11].

3. Fetal-Hgb binds CO 172 times more than O_2 (lower affinity than adult Hgb), albeit CO binding capacity is longer in time than in adult Hgb. Usually fetal CO-Hgb levels are 10–15% higher than maternal CO-Hgb.

7. Diagnosis and differential diagnosis

Given the vagueness and the broad spectrum of complaints, misdiagnosis of carbon monoxide (CO) toxicity is common. Hence, it is necessary to specifically inquire about possible exposures when considering the diagnosis. In some nonfire-related incidents, the most common symptoms were headaches (37%) followed by dizziness (18%), and nausea (17%) [10]. When a patient has a history compatible with CO exposure and when more than one patient in a group or household presents with similar complaints, the following symptoms have been noted: malaise, flu-like symptoms, fatigue, dyspnoea on exertion, chest pain, palpitations, lethargy, confusion, hallucination, agitation, visual disturbance, syncope, seizure, and neurological symptoms [5].

It should be noted that chronic exposure to CO may produce the above symptoms; but what is more common is the gradual-onset neuropsychiatric symptoms such as memory disturbance including retrograde and anterograde amnesia, impaired judgment and psychosis. Some patients may develop delayed neuropsychiatric symptoms, often after severe intoxications associated with coma. After recovery from the initial incident, patients present several days to weeks later with neuropsychiatric symptoms such as those just described. Two-third of patients eventually recover completely.

The gold standard diagnosis of CO poisoning can be accomplished only by detecting the level of circulating carboxyhemoglobin (CO-Hgb). The measurement of CO-Hgb can be performed via blood-gas analyses or by spectrophotometry by performing SpO_2 measurements. However, general SpO_2 sensors are not able to detect CO-Hgb, a special equipment is required. Either arterial or venous blood can be used for testing. The analysis of CO-Hgb requires direct spectrophotometric measurement in specific blood-gas analyzers. It is noteworthy that the latest emergency defibrillator-monitor systems are equipped with CO-Hgb sensors. Equally, it is important to underline that even in case of severe CO-Hgb intoxication, normal SpO_2 levels can be sensed, due to similar spectrophotometry waveform of oxy- and CO-Hgb. Thus, diagnosis in emergency pre-hospital care may be challenging. Even

more, differential diagnose of CO poisoning includes wide range of acute and chronic medical conditions. Early signs and symptoms are atypical, thus it is extremely important to be aware and keep in mind the possibility of CO intoxication, when the circumstances of the scenario suggest. In severe cases, every medical condition that may cause loss of consciousness should be ruled out.

The main differential diagnoses considerations include ruling out:

- other intoxications such as by ethyl- or methyl-alcohol, cyanide, drugs of abuse, or medications;

- infective neurological disorders such as encephalitis or meningitis;

- metabolic disorders such as hypoglycemia, hepatic coma, hypothyroid coma, diabetic keto-acidosis, and lactic acidosis;

- psychiatric conditions.

During in-hospital diagnostics several nonspecific tests can be performed, which help clinicians to determine and quantify the severity of organ specific tissue damage. In this concept, cardiac biomarkers can estimate myocardial ischemia: for example, troponin I, T, and creatine kinase. Peripheral myogenic injury may be estimated via myoglobin urine levels. NT-pro-BNP is able to mirror elevated left ventricular filling pressure and heart failure, especially in the case of severe myocardial ischemia and related acute heart decompensation. Further laboratory tests are able to estimate renal (BUN, creatinine) and hepatic function (liver transaminase). Blood count, ion levels, and glucose levels are also measured routinely. To follow-up metabolic status, blood-lactate should be investigated. Elevated lactate levels suggest more severe CO poisoning and are the predictor of worse outcome [8].

In the most severe cases, radiology scanning of the central nervous system is required to estimate damages. Hence computer tomography (CT), magnetic resonance imaging (MRI), or positron emission tomography (PET) scans are advisable to detect organic neurological complications, mainly bilateral ischemic focus in basal ganglions and subcortical white matter damages. Rarely, haemorrhagic complication can also occur if there is a severe central nervous hypoxia/anoxia in high metabolic demand areas of the brain.

8. Treatment options

Treatment protocol should follow the ACBDE rule, as for every emergency scenario. Airway, breathing, circulation, disability, and exposure should be assessed and treated in respective order. In short, it means that the clinical team whether first responders or people first attending to the patient, must ensure that the airways are clear from any obstruction, that the breathing is present or is restored and efficient, that blood circulation is assured; that further complications and disability are prevented, and that the victim is removed or shielded from further exposure. In sequence, the treatment options include pre-hospital care and hospital care in emergency departments.

8.1. Pre-hospital care

Pre-hospital care includes the following:

- Promptly removing the patient from continued exposure and immediately institute oxygen therapy with a nonrebreather mask.

- Performing intubation for the comatose patient for airway protection, or if necessary, for ventilatory support and provide 100% oxygen therapy.

- Instituting cardiac monitoring.

- Alerting the emergency department of the up-coming comatose or unstable patients because rapid or direct transfer to a hyperbaric center may be indicated.

- Drawing early blood samples for accurate correlation between CO-Hgb measurements and clinical status.

- Obtaining an estimate of exposure time, if possible.

- Avoid exertion to limit tissue oxygen demand.

In CO intoxication, the most important approach is to remove the patient from CO-intoxicated gas area as soon as safely possible and provide high flow 100% oxygen inhale by nonrebreathing masks. The respiratory, hemodynamic status, and consciousness level defines whether the patient needs further respiratory (mechanical ventilation) or hemodynamic support. Controlled hyperventilation provides faster CO elimination. These should be administered as soon as possible, even in pre-hospital first medical contact. For those patients, who suffered CO intoxication in fire event, care should be taken on burns and burn-syndrome-related secondary SIRS (systemic inflammatory response syndrome).

First medical contact

- Detecting CO
- Assessing all possible intoxicated patients
- 100% oxygen ASAP to all poisoned
- Assessing the severity of signs and symptoms
- Assessing special needs: children, pregnancy

In-hospital treatment

- Evaluating CO-Hgb levels and severity
- Assessing peripheral organ dysfunction
- Assessing central nervous complication
- Preventing further hypoxic tissue damage
- Considering special treatment options
- Neuropsychiatric follow-up

Textbox 1. Summarizes key elements of first medical contact and in-hospital treatment.

8.2. Hospital care

In-hospital management requires thorough assessment and support of all damaged organs. Pre- and intra-hospital care should take careful consideration on myocardial ischemia. ECG recording should be carefully analyzed and actions should be taken to decrease myocardial injury and prevent further myocardial ischemia. High-flow oxygen therapy, is needed to prevent myocardial and central nervous ischemic injury, to support renal function, and to compensate metabolic disturbances. The administration of hyperbaric oxygen therapy is the mainstay therapy for CO intoxication. Hyperbaric oxygen therapy initiates CO-Hgb elimination from the human body, and increase partial oxygen pressure and oxygen delivery of circulating blood. During hyperbaric oxygen therapy, the Hgb–oxygen dissociation curve is shifted to the right, thus oxygen binding of Hgb and tissue oxygen delivery is increased. Myoglobin and cytochrome systems bind CO in lower rate. Every patient with CO intoxication may benefit from hyperbaric oxygen therapy, however availability and costs tailor patient population for this treatment. Patients should be selected via the following criteria [9, 12]:

- CO-Hgb level above 25%.

- Loss of consciousness.

- Evidence of organ ischemic function (e.g., renal or myocardial injury).

- Severe metabolic imbalance (lactatemia).

- Pregnancy.

Hyperbaric oxygen therapy is proven to decrease late neurological complications and deficit in CO intoxication. Neuropsychiatric tests are able to estimate loss of function, these may help finding those patients who benefit the most from rarely available and expensive therapy [13, 14]. Hyperbaric oxygen therapy should be administered as soon as possible, but at least in 6 hours to exposure time. It is cautioned here that, although some studies have reported major reductions in delayed neurologic sequelae, cerebral oedema, pathologic central nervous system (CNS) changes, and reduced cytochrome oxidase impairment as a result of hyperbaric oxygen therapy, some systematic reviews have not revealed a clear benefit of HBO, so no clear guidelines for its use have been determined. [15, 16].

8.3. CO intoxication treatment in pregnancy

The major cornerstone of treating a pregnant woman with CO intoxication is to decrease fetal CO-Hgb level. Fetal oxygenation must be provided; noting that in pregnancy, hyperbaric oxygen therapy is superior to urgent cesarian section [15]. However, hyperbaric oxygen therapy is rarely available, thus in many cases, urgent cesarean section is the only option to support fetal oxygenation. Fetal CO-Hgb levels cannot be directly measured but can be estimated. Moreover, fetal ultrasound or MRI scan, if available, can be used to check for central nervous system damage and guide further therapy for the new-born if a cesarian section was performed [16].

9. Case report

This case report is provided from the Hungarian National Pediatric Ambulance Service. The ambulance got a call from a family member who said that their 6-year-old child suffered from convulsions while in the bathroom; but regained consciousness spontaneously, though he still had impaired mental function and could not answer simple questions. It is important to note that convulsion in a 6-year-old child may result from several conditions; hence a skillful questioning is necessary to establish the most likely cause.

Fortunately, the Hungarian pediatric ambulance services are equipped with the latest emergency care machines including CO detectors. When the ambulance arrived at the scene, the CO detectors alarmed a high concentration of CO (335 ppm). Beside the 6-year-old child, there were also five adults in the flat. The child was conscious, with altered mental status. All people were immediately evacuated from the flat to fresh air and the fire service was called on scene, to detect source of CO. The pediatric ambulance with its LifePak®15 ECG-defibrillator system could measure SpCO via spectrophotometry detectors. The SpCO measurements showed more than 30% CO-Hgb levels in all people who were in the flat. Hence, high-flow oxygen therapy using nonrebreathing oxygen masks was provided to all victims. All of them were in a stable condition based on the vital signs (heart rate, blood pressure, ECG monitor, Neurological evaluation (Glasgow Come Scale)), though adults had moderate symptoms of fatigue, dizziness, and vertigo. With the use of the removal from further exposure and the administration of oxygen, the mental status of the child also improved and he did not experience any longer convulsions.

With regard to the pregnant woman among the victims, it should be noted that she was one of the adult patients was pregnant woman. She was 26-year old and in the 39th week of her pregnancy. Her initial SpCO assessment showed a 28% CO-Hgb level, a severe CO intoxication. Her management was continued in the hospital, her CO-Hgb levels had decreased to 14.5% at the time of hospital admission. Given the high level of CO-Hgb levels, an emergency cesarean section was carried out in order to maintain fetal oxygenation. This was performed within 1 hour after hospital admission. This procedure was needed because there was only one hyperbaric oxygen chamber available in Hungary, so organizing an emergency hyperbaric oxygen therapy would have been difficult and time-consuming lost time. The fetal CO-Hgb level was 5% at the time of birth, which is the level of a smoking adult. The new-born was otherwise healthy and received high-flow oxygen support until total elimination of CO-Hgb. The mother also eliminated her remaining CO-Hgb in the following 1.5 hours. The mother and the baby were discharged from the hospital being in good health.

The lessons learnt from the above case were that ambulance services with latest diagnosis equipment and devices are critical in recognizing a diagnosis of CO quickly and facilitation medical care secondly, the administration of oxygen must be performed as soon as possible; thirdly, that an efficient triage should be performed by the providers who first see the victims in order to identify those who are in critical situations and who should be prioritized such as children and pregnant women; finally, the performance of an urgent cesarean section has been effective in preventing accumulation of CO in fetal and thus saved the life of the baby.

10. Concluding remarks

The key learning points from this chapter include that CO is silent killer; fatal CO intoxication can occur and present with plain, nonspecific signs and symptoms. Prevention is a key to avoid severe intoxication: education, safe heating systems, and use of CO detectors. Furthermore, the first medical contact has an important role in recognizing CO intoxication with the use of diagnosing devices and sometimes, based on the signs and symptoms. It is important to perform a differential diagnosis based on information gathered from patients and relatives. With regard to actual management of CO intoxication, the administration of high flow, 100% oxygen therapy must be administered as soon as possible in pre-hospital care. Later in-hospital management should include the evaluation, treatment, and prevention of further organ damage and the neurological deficits. More importantly, prompt care and interventions are required for pregnant women because children and fetus are prone to suffer more severe CO intoxication. Therefore, hyperbaric oxygen therapy should be initiated as soon as possible when available or an emergency cesarean section should be performed to save the fetus from CO intoxication.

Acknowledgements

Author details

Edit Gara

Address all correspondence to: gara.editgara@gmail.com

Semmelweis University, Budapest, Hungary

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