Study of Carbon Monoxide Intoxication in Fire Victimis

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Carbon monoxide poisoning is the leading cause of mortality and morbidity of toxic origin in the world. Its insidious and polymorphic symptomatology makes it difficult to diagnose. It occurs accidentally, because of non-supervised domestic fires, or in fire victims. In fire victims, in particular, the differential diagnosis between carbon monoxide gas poisoning, inhalation of other toxic products of combustion like cyanide, oxygen deprivation, thermal burns and shock due to burns as a cause of death is not an easy task. The authors examined 107 fire victims that were autopsied at the Forensic Medicine from Iasi, Romania, in the last 10 years (2007-2016). Most cases were males (69.16%), young (0-9 years) or older than 60 with a burned surface of 91-100% in 68.22% of cases. Blood samples from the cadavers were collected in all cases in order to analyse carboxyhaemoglobin concentration and haemolysis. Toxicological analysis revealed a carboxyhaemoglobin level of maximum 95% but the majority of cases (70.72%) had a concentration inferior to 50%. An inverse correlation was identified between carboxyhaemoglobin concentration and haemolysis, an indicator of heat dissociation. Our study proves that many fire victims may die because of carbon monoxide intoxication prior to the extent of burns at a lethal potential.

Keywords: carbon monoxide, intoxication, fire victims, gas poisoning, lethal

Carbon monoxide (CO) is one of the main causes of death by poisoning for all ages, worldwide [1]. Compared with carbon dioxide (CO_2), which is heavier than air and toxic just on some ratios with breathable oxygen, CO is a toxic gas, less dense than air and dispersed with ease, which is formed mainly from the incomplete combustion of hydrocarbons. To its high toxicity is added the danger of going unnoticed, as it is odourless, colourless and not irritating for mucous membranes, not announcing its lethal presence to unnoticed people exposed [1, 2]. The biochemical mechanism of the poisoning effect is very different than with other substances [3-8].

The toxicity of this gas is due to the high affinity of haemoglobin (Hb) for the CO, forming carboxy-haemoglobin (COHb), presented in figure 1, thus unable to transport O_2, and producing tissue hypoxia. The COHb levels depend on multiple factors, including the magnitude of the exposure, the degree of alveolar ventilation, blood volume and metabolic activity. A level of COHb higher than 3% in non-smokers or higher than 10% in smokers confirms exposure. Acute or chronic severe poisoning can occur when relatively small quantities of CO are present in the inspired air [9, 10]. Acute CO poisoning is a common medical emergency and a cause of intentional or accidental death, however, its recognition may be difficult, being able to go unnoticed due to the characteristics of this gas and the nonspecificity of the symptoms it produces.

Cardiovascular and neuropsychological harmful effects have been documented in presence of CO concentrations in air below 25 parts per million and at levels of COHb in blood lower than 10%. The cardiovascular alterations that have been described are arterial hypertension, appearance of arrhythmias and electrocardiographic signs of ischemia. Deficit in memory, attention, concentration and parkinsonian type movement alterations, are the neuropsychological changes most frequently associated

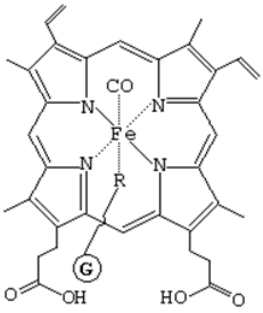

Fig. 1. Carboxyhaemoglobin formula (G - globin, R - functional group)

with chronic exposure to low levels of CO and COHb. Such clinical manifestations may mimic a variety of chronic diseases like decompensated diabetes mellitus associated with comorbidities including chronic heart failure, and neuropathy [11-16]. Biochemical markers assessment can be useful in guiding diagnosis [17]. These can be minimized or prevented after treatment with oxygen (O_2) 100% or hyperbaric therapy [9, 18].

In forensic cases, toxicological analysis should be performed, especially when the cause of death is not clear since in many cases no pathognomonic lesions are observed [19].

The aim of the current study was to analyse fire victims autopsied at the Forensic Medicine Institute from Iasi, Romania, in order to determine the contribution of CO intoxication to the lethal mechanism.

Experimental part

Material and methods

The authors analysed 107 fire victims that were autopsied at the Forensic Medicine from Iasi, Romania, in the last 10 years (2007-2016). Most cases were males (69.16%). Concerning the age distribution, 17 cases (15.89%) were younger than 10 years, and 47 (43.92%) older than 60 years. The rest of 43 cases (40.19%) were between 10-60 years old. Most victims (59 cases – 55.14%)

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deceased secondary to a household fire, 38 (35.51%) in industrial hazards, 7 in open field fires (6.54%) and 3 (2.80%) in road accidents with car burning.

The burned surface exceeded 80% in most cases:
- less than 30% - 2 cases (1.87%);
- 31-40% - 3 cases (2.80%);
- 41-50% - 5 cases (4.67%);
- 51-60% - 5 cases (4.67%);
- 61-70% - 8 cases (7.48%);
- 71-80% - 11 cases (10.28%);
- 91-100% in 73 cases (68.22%).

69 cases (64.49%) presented soot in the trachea and lungs (Montalti sign), vital signs signifying that the victim was burned alive. In 11 cases (10.28%) the sign was negative, and in the rest of 27 cases (25.23%) it was not indicated or not significant.

Blood samples from the left ventricle of the cadavers were collected in 92 cases (85.98%) in order to analyse carboxyhaemoglobin concentration and haemolysis.

Toxicological analysis revealed a carboxyhaemoglobin level of 10-50% in 47 cases (51.09%) which also is a sign of vitality (50% is considered the minimum lethal level). In 21 cases (19.63%) the value was below 10% and in these cases corroboration with other signs is necessary in order to affirm whether the victim was alive or not during the disaster. The value exceeded 50% in only 14 cases (13.08%) thus being the direct cause of death. COHb was negative in 10 cases (10.87%), the victim being already dead when the fire started. Thus, the majority of cases (70.72%) had a concentration inferior to 50%. An inverse correlation was identified between carboxyhaemoglobin concentration and haemolysis, an indicator of heat dissociation.

With regards to drugs in general, it was found that in 52 cases (48.6%) of bodies, the drug analysis was not requested. Among the rest, 8 (14.55%) were positive for alcohol, benzodiazepines or other psychoactive substances. However, it is important to highlight that although very few cases resulted positive, alcohol levels found in four cases exceeded the 250 mg%, which allows us to suppose that these individuals were in stupor or even ethyl coma in the moment of death.

Concerning autopsic findings, cadaveric lividities were usually fixed, pinkish, associated to pink foam in the respiratory orifices and absence of cadaveric rigidity. The muscle structures had a strong rosy color and the blood was very fluid, with no clots, in victims with >50% COHb. Pulmonary oedema was also discovered in 37 cases (34.58%). The outer surface of the brain appeared with an intense meningeal congestion and on the cut there was also evident, vascular congestion.

Results and discussions

In the case of fire victims, it is a routine procedure for the forensic toxicologist to examine blood samples for determining levels of COHb, since saturation levels greater than 50% indicate that one of the main cause of death was by CO intoxication, levels between 10-50% indicate that the deceased inhaled smoke which could have contributed to his death, concentrations between these levels indicating that the deceased was alive when the fire started. Levels of COHb below 10% raise the suspicion that the individual died before the fire started. This determination is very important in civilian litigation cases and in criminal investigation [20].

CO affinity for Hb is about 250-300 times higher than Hb affinity for oxygen, so a concentration of 50% can be achieved with inspired CO levels of 0.08% [20]. This situation leads to a decrease in transport of oxygen to the tissues and anoxia, in addition to a deviation to the left of the dissociation curve of hemoglobin [21]. After one hour of exposure to a CO concentration of 0.1%, COHb can reach a concentration of 80% which would cause seizures, coma and death [22]. Environmental concentrations higher than 1/20 would produce death in a fulminating way [23].

Cases have been cited in which relatively low levels of COHb (<10%) can trigger a clinical picture of angina in subjects with arteriosclerosis [24, 25]. Levels between 10 and 20% of COHb can cause visual alterations, vertigo, abdominal pain and nausea. Concentrations between 20 and 40% can cause dyspnea, arrhythmia, syncope and vomiting. Values greater than 60% induce seizures and coma [26], leading to death in 70-80% of cases. Some authors [27] consider COHb values of less than 2% rather harmless, and values >40% potentially fatal due to suffocation. A carboxyhemoglobinemia of 2.5%, secondary to an exposure for 90 min at an approximate concentration of 50 ppm of CO, can produce a deterioration of the temporal orientation; when the concentration approaches 5%, there is degradation of the psychomotor faculties and above 5% cardiovascular alterations can occur. It is necessary to take into account the endogenous production of CO. Healthy subjects present a carboxyhaemoglobin saturation of 0.4-0.7%, reaching 5% in large smokers [28].

According to other researchers [29], blood levels of COHb between 15-25% produce symptoms consisting of headaches and nausea. Levels around 30-40% produce headaches, vomiting, shortness of breath and weakness in the lower limbs and mental confusion, which prevents victims from escaping the contaminated environment. When the concentration is higher than 45% it appears coma, metabolic acidosis of lactic origin, due to anaerobic glycolysis, hypokalemia, hypotension, seizures, respiratory depression, pulmonary edema, alterations in the ECG (depression of the ST segment, pathological T waves, tachycardia, ventricular fibrillation). The death of the individual usually occurs when COHb levels > 60% [30, 31]. In table 1 these symptoms are summarized.

With regard to the causes of death of the victims we have reviewed for the present study, the data obtained evidenced that the causes of death was *Carbonization* in 82 (76.63%) of cases, *CO poisoning in* 12 cases (11.21%), the combination of the previous ones in 6 cases (5.60%), *Grade II and III burns* in 5 cases (4.67%) and other causes in 2 cases (1.87%). Other authors consider that most deaths occurring in fire are due to toxic gas intoxication and not to

COHb (%)	Signs and symptoms
<10%	Asymptomatic
10-20%	Headaches and vasodilatation
20-30%	Headaches, dyspnoea, angina
30-40%	Intense headaches, nausea, vomiting, vision and mental alteration
40-50%	Syncope, tachycardia, tachypneea
50-60%	Coma, convulsions, irregular respiration rate
>60%	Cardiorespiratory arrest, death

Table 1
SYMPTOMS OF CO INTOXICATION ACCORDING TO COHb CONCENTRATION

burns *per se*. The discrepancies could be due to the fact that we did not consider COHb concentration equal to or greater than 50% as an *a priori* cause of death but as a potential one. In 17 cases, toxicological analysis was not requested, and given the macroscopic findings of carbonization it was assumed that this was the death cause. Occasionally, opinions were issued prior to toxicological results, thus not including an eventual intoxication in the causes of death although the reported levels of COHb are elevated. In other cases, COHb levels were inconsistent with the macroscopic findings thus leading to affirming *Carbonization* as the death cause.

Tissue samples should be collected for COHb analysis in all fire victims in order to determine whether or not there was lethal CO poisoning before the thermal trauma.

In the United States, approximately 4000 deaths by fire are reported each year and of these, 90% occur at home and are caused by cigarettes, electrical defects and defects in heating installation or combustion of clothes [32]. In other countries like Korea, deaths due to thermal burns represent approximately 2.6% of the traumatic deaths [33, 34]. The majority of these deaths occur in fires caused by short circuits, carelessness of smokers who leave cigarettes lit, children who are playing with matches or traffic accidents, in which after a violent collision occurs an explosion.

Fires in buildings are usually accidental, however the possibility that a fire has been provoked in order to murder or hide bodies that were killed previously should be investigated and in this case COHb quantification proves useful.

Suicidal death by fire is less frequent, but represents a significant proportion in countries where mental illness (mainly depression) represents an increasing public health problem.

The death of victims with thermal burns, can occur before, during and after fire [35, 36].

When the death has occurred before burning, vitality signs are absent but their absence does not necessarily indicate that the victim was dead when the fire started. Toxicological analysis is mandatory to identify a potential drug or alcohol overdose. Any body found at a fire scene should be considered the victim of inflicted violence until proven contrary [37].

Deaths during fire are due to:

- burns affecting a significant percentage of the body surface (hypovolemia, toxemia, hypotension, shock, hemoconcentration, hydroelectrolytic disorders);
- intoxication secondary to gas inhalation (more than 300 toxic substances can be released during a fire and contribute to or cause death);
- asphyxia secondary to inhalation of hot gas causing oedema, local irritation, burns, glottis spasm and finally airway obstruction;
- suffocation due to lack of oxygen;
- physical trauma (crushing, jumping from buildings in fire);
- other pathologies induced by shock (myocardial infarction, stroke) [30, 37].

Deaths after fire are also called deferred deaths occuring in victims who are rescued and usually undergo resuscitation manoeuvres and benefit from treatments before dying hours, days or weeks after exposure. These deaths are due to multiple factors that affect different organs and tissues (burns, sepsis, fluid and electrolytes disorders, shock, toxaemia, respiratory distress).

Conclusions

In conclusion, fire victims die before, during or after the disaster. In the first two instances, a careful forensic analysis with toxicological determination of COHb levels is mandatory in all fire victims in order to affirm the death cause and establish the sequence of events leading to death.

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