

CASE REPORT

Suicide by Inhalation of Carbon Monoxide of Car Exhausts Fumes

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Abstract: Background: Carbon monoxide (CO) is a major and ubiquitous component of fire atmospheres produced when organic matter is burned in an inadequate supply of oxygen. Accidental poisoning by CO is common in cold climates where fireplaces, a gas, electric or kerosene heaters or grills are used inside ill-ventilated buildings. In the Brazilian Amazon, with its hot and humid climate, there is no need for the use of heaters and accidents may occur in cases of residential fires or burning of the forests for land use in agriculture.

Objective: We present a case of CO suicide of twenty-six-year-old men.

Method: A forensic autopsy was performed to evaluate the circumstances, cause and medio-legal death etiology.

Results: Autopsy evidenced the typical but also not commonly published cherry-red color of the hypostasis, lungs and other organs, and the very fluid cherry-red blood. The cause of death was due to a massive CO inhalation.

Conclusion: While these poisonings are well recognized, and a vast number of publications on CO toxicity exist, both in an environmental and industrial context, suicide is infrequently encountered in forensic practice and the typical signs are rarely seen in the literature.

Keywords: Carbon monoxide, suicide, poisoning, forensic autopsy, ill-ventilated, residential fires.

1. INTRODUCTION

After ethanol and drugs, CO poisonings represent one of the most common intoxications in forensic routine practice [1]. CO is a “silent killer”; indeed since it is a colorless, tasteless, no irritative and an odorless gas, its presence is not recognized until the effects of poisoning are begun [2, 3]. Death from CO inhalation occur usually from accidental poisonings and since the gas can percolate considerable distances in house conflagrations, the cause of death is usually due to the inhalation of smoke rather than by thermal burns [4]. Moreover, since it is lighter than air, it is always advisable to crawl on the floors to get out of the scene than to walk or run to reduce the risk of CO inhalation. CO binds to hemoglobin (Hb), approximately 200 times more strongly than oxygen, leading to the formation of carboxyhaemoglobin (COHb), and consequently anemic hypoxia or even anoxia [2, 5]. A COHb concentration of about 50-60% is usually considered fatal, but variations exist and are easily exemplified when two or more victims have died together in

the same environment and present different COHb percentages. Indeed, in victims with previous/concomitant morbidity, namely of cardiovascular (e.g., coronary disease, myocardial dysfunction) and respiratory (e.g., obstructive airway disease) origin, or in the presence of high doses central nervous system depressants drugs such as ethanol and barbiturates, interpretation of lower COHb concentrations may be more difficult.

The CO originates from the incomplete combustion of carboniferous materials in low oxygen conditions, such as the burning of fossil fuels (e.g., firewood, charcoal and mineral coal, gasoline, kerosene, diesel and gas), heaters, thermoelectric mills, fires forest or residential areas and even the burning of tobacco [6]. In countries with cold weather, CO poisoning is more related to accidental causes, followed by suicides and rarely by homicides [2, 6]. In the Amazon Region, where the weather is hot and humid, with a predominance of annual average temperatures ranging between 22 and 28°C accordingly to the Center for Weather Forecasting and Climatic Studies (CPTEC) of the National Institute for Space Research (INPE), accidental intoxications are very rare, and when they happen, comes from inhaling smoke from urban fires or burning of the forests for land use in agriculture.

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Fig. (1). Cherry-red lividity and blood as consequence of carboxyhemoglobin formation. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

In the present case, a young man committed suicide by inhaling CO from the exhaust fumes of a motor vehicle.

2. CASE REPORT

A twenty-six-years-old man committed suicide by CO inhalation. Family members reported that when they woke up the car was parked in the garage with the engine turned on and the victim inside. The car was locked and garden hose was inserted in the car exhaust, which was sealed with a t-shirt. The other end of the hose was inserted through the window at the passenger side of the car. The hose was removed from the car exhaust and the family broke the window through which the hose was inserted. Then, the vehicle was opened and the victim was already dead.

The autopsy was performed 9 hours after death. The cadaveric stiffness was already complete, and the cherry-red hypostasis fixed on the dorsum and extremity of the lower limbs (Fig. 1). Internally, the most noticeable sign was again the color. Brain with cherry-red staining, pink lungs with normal auditory signs and highly fluid and bright pink blood due to COHb formation, were registered. In muscle was also evident the pink color due to carboxymyoglobin formation. No other relevant signs were noted in the thoracic, abdominal organs and genitals. Previous psychiatric problems were not reported from relatives and clinical records. Although none of these subjective findings can replace proper toxicological laboratory investigation, the level of COHb in the *postmortem* blood could not be quantified, since the forensic laboratory was not able to perform the necessary analysis at the time of the autopsy.

3. DISCUSSION

Due to its physical properties, CO has been used to commit suicide, believing that such intoxication will cause a non-painful death [7]. What explains the severity of CO poisoning is that Hb in red blood cells has a much greater affinity for CO than O₂. CO combines reversibly with the iron moiety of the heme group of Hb to form COHb [8]. This complex is more stable than oxyhemoglobin (O₂Hb) decreasing the O₂-carrying capacity of blood and by an allosteric effect also causes a shift to the left of the O₂-Hb dissociation curve preventing the O₂ release from O₂Hb to the cells leading to anemic hypoxia [8, 9]. Observations in several animal

studies suggest that the direct effect of CO on cells is more important than decreasing the O₂ carrying capacity of Hb. Indeed, CO impairs the normal respiratory function of cells, as it irreversibly binds to hemoproteins (e.g., cytochrome a₃ and myoglobin), resulting in mitochondrial dysfunction in several organs and systems such as central nervous system and heart [2].

Particularly, autopsy findings in CO deaths are characteristic and can be important forensic clues to the cause of death. We illustrated these findings by reporting a case of CO poisoning (Fig. 1). Normal lividity (or *livor mortis* or hypostases) is blue-purplish due to venous blood color but this is highly dependent upon the state of oxygenation at about the time of death. In CO poisoning, lividity can exhibit a cherry-red or bright-pink color in Caucasians due to COHb formation. As mentioned above, this discoloration may be lost if COHb levels fall during resuscitation. Noteworthy, similar lividity will develop in other circumstances, namely: i) death due to hypothermia (e.g., exposure to intense cold or drowning) and following the morgue's refrigeration of a recently deceased body, due to reduced metabolism and left shifting of the O₂Hb dissociation curve that leads to oxygen retention in blood. If the body is brought out from a cold environment to the normal room temperature, typical zonal segmentation of hypostases may be seen, the color is dark blue in the re-warmed areas and red in cold areas; ii) cyanide or fluorocetate poisonings as consequence of an excessively oxygenated blood due to the inhibition of cytochrome oxidase; and iii) if the area of the cadaver was covered by wet clothing. In racially pigmented victims, especially Negroid, the discoloration may be masked, but may still be seen and is prominent in the conjunctivae, nailbeds, tongue and mucosa of the lips, and palms and soles of the hands and feet. On the other hand, when methemoglobinemia develops, as occurs in exposure to lethal concentrations of potassium chlorate, nitrates, and aniline, lividity tends to be a dark, chocolate-like brown color. In cases of hydrogen sulphide poisonings, a cherry-red or pink lividity (mimicking CO intoxications) and green patches in the skin may be formed [10, 11]. Moreover, an accentuation of the greenish color of the gray matter of the brain may develop probably due to the formation of sulphamoglobin [4].

Coma blisters or coma *bullae* in comatose patients as a consequence of CO poisoning, were also previously re-

ported [12-15]. Nevertheless, as CO deaths are relatively rapid, such blisters are most frequently absent.

A highly fluid and bright pink blood due to COHb formation was also observed in this case. Although, toxicological analyses were not performed, it should be mentioned that the classical cherry-red color of COHb is usually evident if the saturation of the blood exceeds about 40% and below these levels, poisonings are rarely fatal. In anemic victims with lower Hb blood concentrations, the color may be faint or even absent because insufficient Hb is present to display the color and death can occur at lower COHb percentages. The same is true for infants since the higher respiration rate in comparison to adults, allows more rapid absorption. Finally, as the major effect is the impairment of O₂ transport to cells, it is the percentage saturation of the total available Hb that is important rather than an absolute concentration of COHb in the blood [16]. If the color is not easily evident, it can be enhanced by diluting the blood with water against a white background. Another suggestive finding of CO intoxication is the non-quickly discoloration of the pink color in comparison to normal tissues when these are placed in a formol saline solution for histological analysis [16].

CONCLUSION

This work reports a CO intoxication, which diagnosis was based on the expertise of the crime scene investigation and highly suggestive findings in autopsy. The quantification of the percentage of COHb in blood would be useful for further interpretations, but the analytical technique was not available at the time of autopsy. Typically, spectrophotometry methods have been used either in *antemortem* and *post-mortem* cases [17]. The medico-legal etiology was suicide due to the deliberate running the car engine in a closed garage.

CONSENT FOR PUBLICATION

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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REFERENCES

- [1] Alves EA, Brandao P, Magalhaes T, Carvalho F, Dinis-Oliveira RJ. Fatal intoxications in the North of Portugal: 12 Years of retrospective analysis. *Curr Drug Saf* 2017; 12(1): 39-45. [http://dx.doi.org/10.2174/1574886311666160724212407] [PMID: 27457768]
- [2] Gozubuyuk AA, Dag H, Kacar A, Karakurt Y, Arica V. Epidemiology, pathophysiology, clinical evaluation, and treatment of carbon monoxide poisoning in child, infant, and fetus. *North Clin Istanbul* 2017; 4(1): 100-7. [PMID: 28752154]
- [3] Byard RW. Carbon monoxide - the silent killer. *Forensic Sci Med Pathol* 2019; 15(1): 1-2. [http://dx.doi.org/10.1007/s12024-018-0040-5] [PMID: 30390280]
- [4] Dinis-Oliveira RJ, Carvalho F, Moreira R, *et al.* Clinical and forensic signs related to chemical burns, A mechanistic approach. *Burns* 2015; 41(4): 658-79. [http://dx.doi.org/10.1016/j.burns.2014.09.002] [PMID: 25280586]
- [5] Dinis-Oliveira RJ, Carvalho F, Magalhães T, Santos A. Postmortem changes in carbon monoxide poisoning. *Clin Toxicol (Phila)* 2010; 48(7): 762-3. [http://dx.doi.org/10.3109/15563650.2010.484394] [PMID: 20849333]
- [6] Swain R, Behera C, Kishore S, Krishna K, Gupta SK. Suicidal asphyxiation by carbon monoxide within a polythene bag. *Med Leg J* 2017; 85(1): 35-7. [http://dx.doi.org/10.1177/0025817216669286] [PMID: 27620849]
- [7] Blässer K, Tatschner T, Bohnert M. Suicidal carbon monoxide poisoning using a gas-powered generator. *Forensic Sci Int* 2014; 236: e19-21. [http://dx.doi.org/10.1016/j.forsciint.2013.12.016] [PMID: 24418208]
- [8] Varlet V, De Crouette EL, Augsburger M, Mangin P. A new approach for the carbon monoxide (CO) exposure diagnosis: Measurement of total CO in human blood versus carboxyhemoglobin (hbco). *J Forensic Sci* 2013; 58(4): 1041-6. [http://dx.doi.org/10.1111/1556-4029.12130] [PMID: 23692308]
- [9] França GVD. *Medicina Legal*. 10th Edition. Brasil. Guanabara Koogan, 2015.
- [10] Milroy C, Parai J. Hydrogen sulphide discoloration of the brain. *Forensic Sci Med Pathol* 2011; 7(2): 225-6. [http://dx.doi.org/10.1007/s12024-010-9210-9] [PMID: 21161701]
- [11] Sams RN, Carver HW II, Catanese C, Gilson T. Suicide with hydrogen sulfide. *Am J Forensic Med Pathol* 2013; 34(2): 81-2. [http://dx.doi.org/10.1097/PAF.0b013e3182886d35] [PMID: 23574866]
- [12] Larrey DJ. *Memoires de chirurgie militaire et campagnes*: Smith and Buisson 1812.
- [13] Myers RA, Snyder SK, Majerus TC. Cutaneous blisters and carbon monoxide poisoning. *Ann Emerg Med* 1985; 14(6): 603-6. [http://dx.doi.org/10.1016/S0196-0644(85)80792-7] [PMID: 3994090]
- [14] He H, Gao Y, Li C, *et al.* Cutaneous blistering secondary to acute carbon-monoxide intoxication. *Clin Exp Dermatol* 2007; 32(1): 129-31. [PMID: 16879450]
- [15] Dinis-Oliveira RJ. Drug overdose-induced coma blisters, pathophysiology and clinical and forensic diagnosis. *Curr Drug Abuse Rev* 2019; 11(1): 21-25. [http://dx.doi.org/10.2174/1874473711666180730102343] [PMID: 30058500]
- [16] Saukko P, Knight B. *Knight's forensic pathology*. Boca Raton: CRC Press, Taylor & Francis Group, 2016.
- [17] Luchini PD, Leyton JF, Strombeck MdeL, Ponce JC, Jesus Md, Leyton V. Validation of a spectrophotometric method for quantification of carboxyhemoglobin. *J Anal Toxicol* 2009; 33(8): 540-4. [http://dx.doi.org/10.1093/jat/33.8.540] [PMID: 19874665]