RESEARCH ARTICLE

The silent killer behind closed doors: Forensic patterns of carbon monoxide intoxication

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Introduction: Acute carbon monoxide poisoning is a common cause of death during the autumn and winter months when various heat sources – some improvised – are used. It is often classified as an accidental rather than a voluntary intoxication.

Materials and method: This retrospective study includes a group of 25 individuals whose cause of death was acute carbon monoxide poisoning. The autopsies were performed between 2021 and 2024 at the Institute of Forensic Medicine from Târgu Mureș. Demographic parameters, toxicological results, the influence of the exposure method to the causative factor, and the correlation between alcohol consumption and carboxyhemoglobin concentration were analyzed.

Results: Statistical analysis showed that 68% of the subjects were males and 32% females, with 44% originating from urban areas and 56% from rural zone. 60% of the individuals included in the study had carboxyhemoglobin concentrations over 50% at the time of death. The mean carboxyhemoglobin concentration for individuals involved in fires was 39%, compared to 52.22% for those caught in gas leaks; this represents a statistically significant difference between two mechanisms ($p = 0.0282$). The presence of ethanol in the blood was identified in 32% of the victims.

Conclusions: Individuals involved in fires presented lower carboxyhemoglobin concentrations, suggesting the implication of other factors or substances in thanatogenesis. Similarly, patients with associated comorbidities had lower carboxyhemoglobin levels. Regarding the association with alcohol, the results suggest a possible potentiating effect of ethanol but this finding did not reach statistical significance.

Keywords: carbon monoxide, intoxication, forensic medicine

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Introduction

Carbon monoxide (CO) is a toxic, colorless gas containing 57.13% oxygen and 42.83% carbon, has a molecular weight of 28.01 g/mol, and a density lower than that of air, being the result of the incomplete combustion of certain fuels [1,2]. After the inhalation of this gas, the signs and symptoms presented by the patient may be non-specific; some symptomatology can suggest other pathologies, which is the reason this intoxication is characterized as a "silent killer" [2,3]. Carbon monoxide intoxication is more frequent in houses with heating systems that are defective, improvised, or not carefully checked before use [4]. In cases victims exposed to CO, hypoxia occurs because the formation of carboxyhemoglobin (COHb), which results from CO binding to hemoglobin. The coordinative chemical bond between CO and hemoglobin is more stable compared to the coordinative chemical bond between oxygen and hemoglobin (oxyhemoglobin). In addition to binding with hemoglobin, carbon monoxide also binds to myoglobin and the tissue cytochrome system, although its highest affinity remains for hemoglobin. The affinity of carbon monoxide for hemoglobin is approximately 200–250 times greater than that of oxygen [5,6,7,8]. Blood COHb concentration can vary based on several factors. In non-smokers, a normal value is below 3%, while for smokers or occupationally exposed at non-toxic levels of CO, it is below 10%. Measuring values above these limits suggests acute CO exposure [1,6]. Clinical signs and symptoms vary depending on the blood COHb concentration. Headache is the most frequent symptoms; subsequently, other neurological, pulmonary, or cardiovascular symptoms may occur [4,6,9]. A carboxyhemoglobin concentration over 50% is considered lethal in healthy individuals without other pathologies, and for patients with comorbidities, the lethal limits is lower, ranging from 20% - 30% blood COHb concentration [6,10,11]. Following death and the circulatory arrest, blood moves under the force of gravity to the posterior and uncompressed regions of the body, forming cadaveric lividity (livor mortis). These lividities with various colors, when correlated with their response to digital pressure, can provide information regarding the time of death [12]. Specific colors may appear in particular cases like in CO intoxication, the characteristic color of the lividity is cherry-red, pink or carmine red [12,13,14].

Methods

The study is retrospective and based on autopsies that were conducted between 2021 and 2024 at the Institute of Forensic Medicine in Târgu Mureș. The inclusion criteria consisted of violent deaths in which the cause of death was acute carbon monoxide poisoning, regardless of the victim's sex or age. We also identified but excluded cases in which COHb was present, but in low levels, so the cause of

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death was attributed to other types of acute intoxication or a critical pathology. Additionally, victims of fires who presented severe and extensive burns with evidence of vital reaction at the histopathological examination, and who had low COHb levels were also excluded. Data was collected manualy from the autopsy reports. The demographic parameters (sex, age, and area of origin) were identified and monitored, along with the results of toxicological examinations (the concentration of carboxyhemoglobin in blood samples collected during autopsy or in hospital units in the case of individuals who survived for a period of time, as well as the presence of other toxic substances such as ethanol), and contextual aspects such as the presence of a fire with or without burns on the body surface or only gas leaks. Additionally, the existence of other victims involved in the same incident was also identified. The chronic pathologies of the deceased individuals were analyzed, as well as the presence or absence of cadaveric lividity specific to acute carbon monoxide intoxication. Blood samples collected during autopsy were obtained from the cranial cavity and subsequently analyzed in the toxicology laboratory. The method for determining carboxyhemoglobin was UV-Vis spectrophotometry. During the analyzed period, all cases of carbon monoxide poisoning were classified as accidental intoxications. For data entry and the creation of the statistical database, Microsoft Excel was used, and subsequently the GraphPad Prism application was also utilized.

Ethical statement

We note that, for the completion of this study, all ethical standards and confidentiality regulations regarding the processing of personal data were fully approved and strictly observed.

Results

During the period 2021–2024, a total of 2,981 autopsies were performed at the Institute of Forensic Medicine from Târgu Mureș, of which 25 cases (approximately 0.83%) had acute carbon monoxide intoxication as the cause of death. The group selected for this study consisted of 25 individuals who died as a result of acute carbon monoxide intoxication. From a demographic perspective, a higher proportion of males was observed, representing 68% (n=17), while females accounted for 32% (n=8). Analysis of the ages of the individuals included in this study revealed a wide distribution: the youngest was 8 years old and the oldest 90 years old, resulting in a mean age of 65.84 years for the analyzed group (Table I).

Demographic data

Statistical analysis of the study group showed that 44% (n=11) originated from urban areas, while 56% (n=14) came from rural areas. The mean COHb concentration at the time of death for individuals from rural areas was 48.25%, whereas for those from urban areas it was 47.66%. Based on these values, it can be concluded that, regardless of the area of origin, death occurs at similar COHb concentrations, with no statistically significant differences ($p = 0.91$; a p-value < 0.05 was considered statistically significant), even though there is a slightly higher predominance of individuals from rural areas. Processing the data regarding the time of year when the deaths were recorded showed that 48% (n=12) occurred in autumn, 36% (n=9) in winter, and 16% (n=4) in the early part of spring (Figure 1).

Toxicological examination

From a toxicological perspective, analysis of blood samples collected during the forensic autopsies revealed the presence of ethanol in 32% of cases (n=8). It should be noted that no psychoactive substances were detected in any individual from the analyzed group during the toxicological examination.

After calculating the Pearson correlation coefficient (r), a negative value (–0.4507) was obtained. This value may suggest that as blood alcohol concentration increases, the lethal value of carboxyhemoglobin tends to decrease. This indicates that acute ethanol intoxication may contribute to

Table I. General data on the analyzed sample

Parameters	Result
Number of cases	25
Mean age	65.84
Gender (M/F)	M: 68%; F:32%
Area of origin	U: 44%; R:56%
Positive blood alcohol conc.	32%
Associated comorbidities	92%
Specific lividity	84%
Family deaths	36%
Accident/Suicide	Accident: 100%

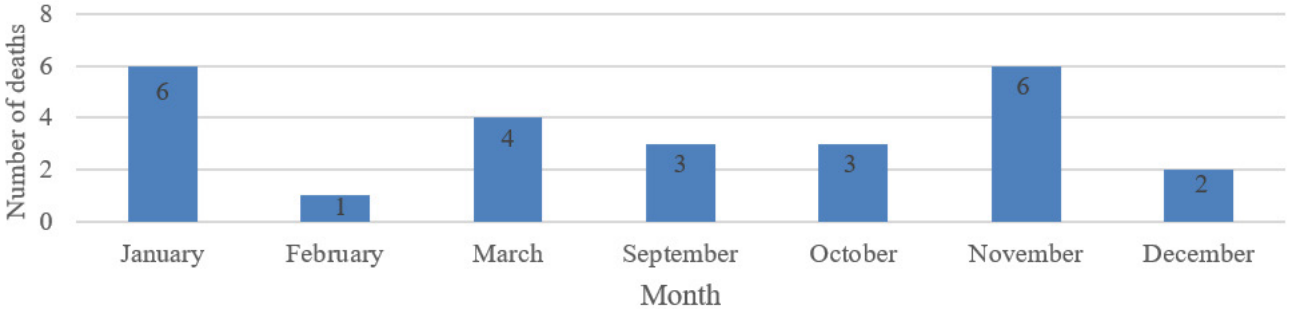

Fig. 1. Number of deaths by month.

the onset of death at lower carboxyhemoglobin concentrations. In this sub-group, the absence of statistical significance (p = 0.2624) may be explained by the small sample size in which ethanol was detected.

Associated comorbidities

Of the total 25 individuals in the studied group, 60% (n=15) had carboxyhemoglobin levels over 50%, a lethal limit even in individuals without other associated chronic pathologies. In the remaining cases, 40% (n=10), death occurred at carboxyhemoglobin concentrations below 50%, with all victims presenting chronic pathology. The most frequent comorbidities identified among these 40% of cases included: myocardiosclerosis, hypertrophic or dilated cardiomyopathy, pulmonary emphysema, bronchopneumonia, subendocardial myocardial ischemia, stenosing coronary atherosclerosis, and aortic atherosclerosis.

Livor mortis

Lividity typical of carbon monoxide intoxication, characterized by a carmine-red or brick-red coloration, was observed in the majority of cases included in the study, accounting for 84% (n=21). In the remaining 16% of cases (n=4), lividity appeared violaceous or pale violaceous; however, based on these findings alone, acute carbon monoxide intoxication cannot be excluded. The gold standard for diagnosis remains the determination of carboxyhemoglobin levels in blood samples collected during the autopsy.

Gas leaks vs fire

Statistical analysis regarding gas leaks and their association with fires showed that in 68% of cases (n=17), only gas leaks were involved, while in 32% of cases (n=8), the individuals were exposed to domestic fires. In these fire-related cases, forensic autopsies revealed burns of varying degrees, affecting 15–80% of body surface area, including burns to the airways.

Mean COHb values were calculated for both groups. Individuals involved in fires had a mean COHb concentration of 39%, whereas those exposed to gas leaks had a mean concentration of 52.22%. This resulted in an absolute difference of 13.22% between the two mechanisms, which was statistically significant (p = 0.0282, Welch's t-test). The lower mean COHb in fire victims suggests the contribution of additional factors (such as combustion products, burns, and heat exposure) in thanatogenesis (Figure 2; Table II).

Family deaths

Data were also analyzed regarding the involvement of other family members in the same incident, resulting in 36% of cases (n=9) where the individuals belonged to the same family.

Discussions

Involvement of other related factors or substances besides carbon monoxide in fires. In cases of individuals involved in fires, lethal COHb concentrations were found to be lower, as other substances—such as hydrogen cyanide, sulfur dioxide, and other toxic products generated during combustion—had a synergistic effect, resulting in death at lower carboxyhemoglobin levels [15]. In the analyzed group, the mean COHb concentration in fire-related cases was indeed lower compared to individuals exposed to gas leaks. The mechanism can be attributed to the inhalation of additional toxic substances (cyanides, nitrogen oxides, acrolein, sulfur dioxide) and soot particles released from burning various materials, causing severe hypoxia more rapidly. At

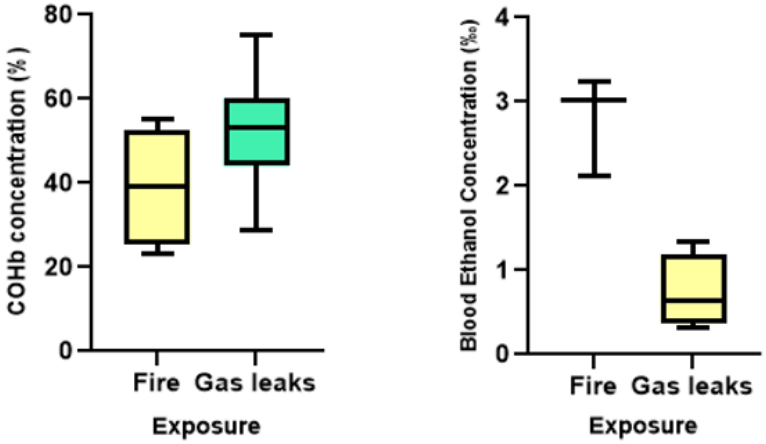

Fig. 2. Mean COHb concentration and mean blood alcohol concentration according to the exposure mechanism.

Table II. Comparative analysis of parameters by exposure mechanism.

Analyzed parameter	Fires – 32% (n=8)	Gas leaks – 68% (n=17)	Result
Mean COHb conc.	39%	52.22%	p=0.0282
Mean age	60.37	68.41	p=0.4080
Mean blood alcohol conc.	2.80‰	0.748‰	p=0.01
Deaths with COHb <50 %	75% (n=6)	23.5% (n=4)	-
Associated comorbidities	87.5% (n=7)	94.11%(n=16)	-

the same time, high temperatures and flames during a fire, act as contributing factors both through inhalation of hot air and by causing burns to the airways, leading to glottic edema and bronchospasm, which can further exacerbate asphyxia [15]. Zheng Liu et al., in the study entitled *"Acute carbon monoxide poisoning with low saturation of carboxyhemoglobin: a forensic retrospective study in Shanghai, China"* (2021), obtained similar results, in individuals involved in fires, death occurred at lower COHb concentrations, suggesting the involvement of additional factors and substances.

Individual susceptibility, age, and associated comorbidities. In the analyzed group, the mean age of individuals who had chronic pathology and a COHb concentration below 50% was 63 years, with cardiovascular and pulmonary diseases being predominant. These associated pathologies increase the vulnerability of this subgroup, as the depletion of the body's physiological reserves occurs much faster than in healthy individuals, causing death from acute carbon monoxide intoxication at lower carboxyhemoglobin concentrations. The affinity of carbon monoxide for hemoglobin is much higher than that of oxygen, leading rapidly to the formation of carboxyhemoglobin and a systemic impact on the body that is faster and more severe—especially in individuals with hypertrophic or dilated cardiomegaly, coronary or aortic atherosclerosis, bronchopneumonia, or pulmonary emphysema. In these cases, even small variations in oxygen concentration are quickly felt and have a rapid, significant impact on the affected individuals.

Association between alcohol consumption and carboxyhemoglobin levels at the time of death. Zheng Liu et al., in the study entitled *"Acute carbon monoxide poisoning with low saturation of carboxyhemoglobin: a forensic retrospective study in Shanghai, China"* (2021), found that in some cases COHb concentration levels increased significantly in individuals who had consumed alcohol; however, no significant linear correlation was observed between COHb saturation and blood alcohol concentration. This contrasts with the 2023 study *"Analyses of physical factors that contribute to the blood carboxyhemoglobin saturation in autopsy cases of house fire fatalities"* by Yukari Toyofuku et al., in which low COHb concentrations were associated with ethanol intoxication or severe coronary stenosis [15,16].

Analysis of the toxicological results showed that ethanol was present in 32% of cases. Statistical analysis did not yield a significant result ($p = 0.2624$), but the negative Pearson correlation coefficient ($r = -0.4507$) suggests a negative trend. This indicates that higher blood alcohol levels may contribute to death at lower COHb concentrations compared to individuals in whom alcohol was absent. For statistical relevance, however, testing on a larger sample would be necessary. Finally, it should be noted that carboxyhemoglobin concentration should not always be evaluated in isolation but interpreted with the patient's age and medical history.

Conclusion

The study highlighted a statistically significant difference between the average concentrations of carboxyhemoglobin depending on the mode of exposure, and also showed that those involved in fires were in an advanced state of acute intoxication with ethanol, with their reaction capacity greatly diminished. The interaction between alcohol consumption and COHb concentration showed a negative, statistically non-significant correlation, but it may indicate that alcohol consumption could be a contributing factor. Each person's biological status, through the presence of associated comorbidities, influences thanatogenesis by increasing the body's sensitivity to triggers, resulting in a significant proportion of cases who died at lower carboxyhemoglobin concentrations.

Authors' contributions

GD (Writing – original draft; Data curation; Formal analysis; Investigation)
ȘAL (Data curation; Formal analysis; Investigation)
TH (Methodology; Visualization; Software)
CC (Conceptualization; Supervision; Writing – review & editing)
CCR (Supervision; Writing – review & editing; Validation)

Conflict of interest

None to declare.

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References

1. Karapirli M, Kandemir E, Akyol S, et al. Forensic and clinical carbon monoxide (CO) poisonings in Turkey: A detailed analysis. J Forensic Leg Med. 2013 Feb;20(2):95-101.
2. Mureşan CO, Zăvoi RE, Dumache RO, et al. Co-morbidities in the multiple victims of the silent killer in carbon monoxide poisoning. Rom J Morphol Embryol. 2019;60(1):125-131.
3. Henry JA. Carbon monoxide: not gone, not to be forgotten. J Accid Emerg Med. 1999 Mar;16(2):91-92.
4. Dan Perju-Dumbravă – Medicină Legală (suport de specialitate). UMF "Iuliu Haţieganu", Cluj-Napoca – Chişinău, 2015, pg. 72-82.
5. Jiang M, Yu CH, Xu Z, Qin Z. Binding of Carbon Monoxide to Hemoglobin in an Oxygen Environment: Force Field Development for Molecular Dynamics. J Chem Theory Comput. 2024 May 28;20(10):4229-4238.
6. Palmeri R, Gupta V. Carboxyhemoglobin Toxicity. 2023 Apr 17. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://pubmed.ncbi.nlm.nih.gov/32491811/
7. Lee M, Kim HJ, Lee D, et al. Forensic utility of carboxyhemoglobin levels in postmortem spleen specimens in South Korea. Forensic Sci Int. 2024 Aug; 361:112107.
8. Jason Payne-James, Richard Jones, Steven B Karch, John Manlove – Simpson's Forensic Medicine 13th Edition, 2011, pg. 210-215.
9. Dr Tom Moore - IAEM Clinical Guideline – Acute Carbon Monoxide Poisoning, Version 1.0 June, 2023. Available from: https://iaem.ie/wp-content/uploads/2023/06/Acute-CO-poisoning-V1.0.pdf
10. Wilbur S, Williams M, Williams R, et al. Toxicological Profile for Carbon Monoxide. Atlanta (GA): Agency for Toxic Substances and Disease Registry (US); 2012 Jun.
11. Mori, K., Yoshida, J., Morioka, F. et al. Quantification of carbon monoxide (CO) in postmortem human brain tissues after CO poisoning. Sci Rep 15, 32072 (2025).

12. Shedge R, Krishan K, Warrier V, et al. Postmortem Changes. [Updated 2023 Jul 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539741.
13. Cohen PR, Moss RJ Jr, Prahlow JA. Livor Mortis and Forensic Dermatology: A Review of Death-Related Gravity-Dependent Lividity and Postmortem Hypostasis. Cureus. 2025 Aug 22;17(8).
14. Radu Carmen Corina, Caraşca Cosmin, Hogea Timur – Forensic medicine. Specialty support, Editura University Press, 2021.
15. Liu Z, Meng H, Huang J,et al. Acute carbon monoxide poisoning with low saturation of carboxyhaemoglobin: a forensic retrospective study in Shanghai, China. Sci Rep. 2021 Sep 17;11(1):18554.
16. Yukari Toyofuku, Kazuhiko Kibayashi, Ryo Shimada et al. Analyses of physical factors that contribute to the blood carboxyhemoglobin saturation in autopsy cases of house fire fatalities. Legal Medicine, Volume 62, May 2023.