

CASE SERIES

The Silent Killer – A Case Series on Carbon Monoxide Poisoning Following a Domestic Fire

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Abstract

Introduction: Carbon monoxide (CO), the “silent killer,” is a colourless, odourless gas that binds haemoglobin with approximately 200–240 times greater affinity than oxygen, forming carboxyhaemoglobin (COHb), causing tissue hypoxia and death. Fire-related CO poisoning remains a major but under-recognized cause of mortality in poorly ventilated domestic environments.

Case report: We present a retrospective forensic case series of six fatalities from three families who died in a single domestic fire in a rented building in Shastri Park, Delhi, after a mosquito coil fell onto a mattress and ignited it. Medico-legal autopsies included external and internal examination, while blood and viscera were submitted for toxicological analysis. Carbon monoxide was detected qualitatively, and blood ethyl alcohol was quantified.

Discussion: Five of six victims tested positive for CO; quantitative COHb saturation was unavailable. Three showed co-detected ethyl alcohol (38.2–94.7 mg/100 mL). Common findings included soot on the body, clothing, tongue and nostrils; cherry-red lividity, blood and muscle; respiratory tract oedema, congestion and soot deposition; and petechial haemorrhages in the lungs and heart. Victims included a 2-year-old child, a 25-year-old woman in early pregnancy, and one adult with 15–20% total body surface area burns, the only victim with significant thermal injury.

Conclusion: Fire deaths with minimal burns may primarily result from smoke inhalation and asphyxiant gas poisoning. Quantitative COHb and cyanide analysis, alongside careful autopsy, is essential for accurate death certification. Preventive measures, including safe mosquito-coil and candle placement, functioning smoke and CO detectors, and accessible evacuation routes, are critical.

Keywords: Smoke inhalation injury; Suffocation; Carbon monoxide; Carboxyhaemoglobin

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INTRODUCTION

Carbon monoxide (CO) is a colourless, odourless, tasteless and non-irritating gas, which makes it almost impossible to detect without specialised sensors [1-4]. Often called the “silent killer,” it is generated whenever organic material undergoes incomplete combustion [5-7]. Common sources include malfunctioning or poorly ventilated gas appliances such as heaters, stoves, generators and grills, as well as accidental house fires [8, 9]. CO binds haemoglobin roughly 200–240 times more avidly than oxygen, forming carboxyhaemoglobin, which impairs oxygen delivery to tissues, disrupts cellular respiration and inhibits key

enzymes [1-4, 8]. CO poisoning predominantly affects the brain and heart, producing headache, dizziness, nausea, vomiting and dyspnoea, but frequently causes death without specific antemortem warning [1-7, 10]. Individuals who are asleep, unconscious, intoxicated or under the influence of drugs are especially vulnerable, since their capacity to recognise danger and self-rescue is impaired [5-7, 11].

In India, fatal CO poisoning shows a strong seasonal pattern, with a retrospective AIIMS New Delhi study reporting that approximately 80% of cases occurred during the winter months [12], a pattern corroborated by a more recent fifteen-year single-institution series from the same city [13]. Fire-related deaths are a particularly important

subset, since the toxic gas mixture generated by combustion is rarely CO alone. Modern furnishings, bedding foam and household plastics release hydrogen cyanide (HCN) and other asphyxiant and irritant gases on burning, and several forensic series have documented that HCN is detectable in a substantial proportion of fire fatalities and can independently contribute to death even when COHb levels are only moderately elevated [14-16]. This combined toxic burden can incapacitate occupants within minutes, well before flames or heat cause direct injury. We report a case series of six fatalities from a single domestic fire, to illustrate the autopsy and toxicological features that establish smoke inhalation and asphyxiant gas poisoning as the cause of death, and to highlight the forensic and preventive lessons this incident offers.

CASE PRESENTATION

This is a retrospective case series describing six medico-legal autopsies performed at the Department of Forensic Medicine & Toxicology, UCMS & GTBH, Delhi, following a fatal domestic fire in Shastri Park, Delhi, in March 2023.

Autopsy protocol: Each body underwent a standard medico-legal autopsy comprising external examination (documentation of the extent and distribution of thermal injury, soot deposition on the skin, clothing, nostrils and oral cavity, and the character of postmortem lividity) followed by systematic internal examination. The respiratory tract-nasal cavity, oropharynx, epiglottis, subglottis, trachea, bronchi and bronchioles were examined for mucosal congestion, oedema and soot deposition, and the lungs, heart and other viscera were assessed for congestion, oedema and petechial haemorrhage. In the female victim, the uterus was examined and products of conception was identified.

Toxicological analysis: Heart blood and preserved viscera were forwarded to the forensic science laboratory for toxicological analysis, following standard chain of custody procedures. In this series, carbon monoxide exposure was reported qualitatively by the laboratory (positive or negative for CO), rather than as a quantitative carboxyhaemoglobin (COHb) saturation percentage; blood ethyl alcohol was quantified (mg/100 mL). Viscera were preserved in saturated solution of common salt rather than formalin, to avoid interference with CO estimation. A full toxicological/poison panel, including cyanide/hydrogen cyanide analysis, was requested and performed in all cases; carbon monoxide was the only poison detected on this panel.

Case Series

Fatal domestic fire (mosquito-coil ignition), Shastri Park, Delhi, March 2023: six fatalities across three families, with death attributed to asphyxia due to smoke inhalation and CO poisoning. Findings are summarized in Table 1.

Common Findings

1. No antemortem injury was identified in five of the six victims; one individual sustained thermal burn involving approximately 15–20% of the total body surface area.

2. Blackish soot particles were present on the clothing, body surface, tongue and in both nostrils in all cases.

3. A distinct cherry-red discoloration was noted in postmortem lividity (Figure 1), and in the blood and skeletal muscle (Figure 2).

4. Examination of the upper and lower respiratory tract showed oedema, congestion and black soot particles throughout (Figure 3).

5. Multiple petechial haemorrhages were present in the lungs and heart, with generalized visceral congestion.

6. Toxicological analysis confirmed carboxyhaemoglobin in five of six cases, with ethyl alcohol detected in three.

DISCUSSION

Accidental CO-related deaths commonly occur during fires, or from incomplete combustion of wood, charcoal or coal in poorly ventilated domestic spaces [1-4]. Such incidents are more frequent in winter, Sikary et al. reported that roughly 80% of fatal CO cases at AIIMS New Delhi occurred during the colder months [12], and a more recent fifteen-year review from the same institution similarly found the great majority of fatalities clustering in winter, most often linked to coal burning heating appliances or structural fires [13]. A comparable seasonal pattern has been described outside India, a 15-year forensic autopsy series from Turkey found that 60% of fatal CO poisonings in older adults occurred in winter and 84% in domestic settings [17]. In the present series, the victims were found in a confined room with a burnt mattress, apparently ignited by a mosquito coil, consistent with rapid asphyxiation in an inadequately ventilated space [8, 9, 18, 19].

Soot deposition on the skin, in the nostrils and throughout the respiratory tract present in all six victims is one of the most important antemortem markers in fire deaths, since it indicates that the victim was breathing during the fire and therefore distinguishes death occurring before the fire (or early in its course) from postmortem burning of an already deceased body. Cherry-red discoloration of lividity, blood and muscle, also seen in all cases, is a classical though non universal sign of CO poisoning; it is typically evident once COHb saturation exceeds approximately 30%, and its absence does not exclude poisoning, particularly at lower saturations [20]. Pulmonary oedema, congestion and petechial haemorrhages in the lungs and heart, present in most of the present cases, reflect the hypoxic and irritant effects of smoke and CO on the cardiopulmonary system, and are supportive though again non-specific corroborative findings rather than diagnostic in isolation.

Ethyl alcohol was detected in three of the six victims (38.2–94.7 mg/100 mL). Alcohol raises the arousal threshold during sleep, impairs judgement and slows escape response, and is a recognized contributory factor in fatal domestic fires; it likely compounded the effect of CO in these cases, alongside the fact that the fire occurred at night while residents were asleep. The series also included two particularly vulnerable victims, a 2-year-old child and a 25-

Table 1. Summary of demographic, autopsy and toxicological findings in the six fatalities

Case	Age/Sex	External findings	Internal findings	Toxicology	Special notes
1	2 y, M (paediatric)	Soot on body/clothes and tongue; cherry-red lividity, blood, muscles	Congestion/oedema of epiglottis, subglottis, trachea, oesophagus, stomach; lungs oedematous/congested with soot to bronchi/bronchioles	COHb positive	Youngest petechial identified victim; no haemorrhages
2	26 y, M	Soot on body, clothes, tongue; cherry-red lividity, blood, muscles	Forehead abrasion, no burns; soot and mucus in airway with congestion/oedema; lungs with petechial haemorrhages, soot to terminal bronchioles	COHb positive; ethyl Alcohol alcohol 79.4 mg/100 mL (~0.079%)	intoxication
3	47 y, M	Same pattern as Case 2	Congestion/oedema of epiglottis, subglottis, trachea, oesophagus, stomach; lungs oedematous/congested with soot to bronchi/bronchioles; petechial haemorrhages on heart	COHb positive; ethyl Alcohol alcohol 94.7 mg/100 mL series (~0.095%)	Highest alcohol level in series (~0.095%)
4	50 y, M	Same pattern	Same pattern as above	COHb positive	No alcohol detected
5	25 y, F (pregnant)	Same pattern; striae gravidarum on abdomen	Same pattern	COHb positive	Uterus contained products of conception, ~1-month gestation (early pregnancy)
6	48 y, M	Superficial thermal burns, 15–20% TBSA; cherry-red lividity; soot on tongue	Same airway pattern as other cases	Ethyl alcohol 38.2 mg/100 mL; COHb not specified as positive in this case	Only victim with burns, likely closest to fire origin; alcohol present but lower than Cases 2 and 3

TBSA: total body surface area; COHb: carboxyhaemoglobin



Figure 1. Cherry-red postmortem lividity

year-old woman in early pregnancy underscoring that children and pregnant women are disproportionately susceptible to CO related hypoxic injury because of higher metabolic oxygen demand and, in the fetus, the leftward shifted oxygen dissociation curve of fetal haemoglobin.

Only one victim (Case 6) sustained significant thermal burns (15–20% TBSA), consistent with proximity to the fire's origin; the remaining five victims, on the upper floors, showed no significant burns despite fatal smoke and CO

exposure. This pattern illustrates a point of broader forensic importance: in structure fires, asphyxiant gas poisoning and smoke inhalation rather than thermal injury are frequently the actual cause of death, and inexperienced examiners may under recognize this when external burns are minimal or absent. This is further complicated by cases of low saturation CO poisoning, in which classical postmortem



Figure 2. Cherry-red discolouration of skeletal muscle

signs are attenuated but death is nonetheless attributable to CO exposure [20].

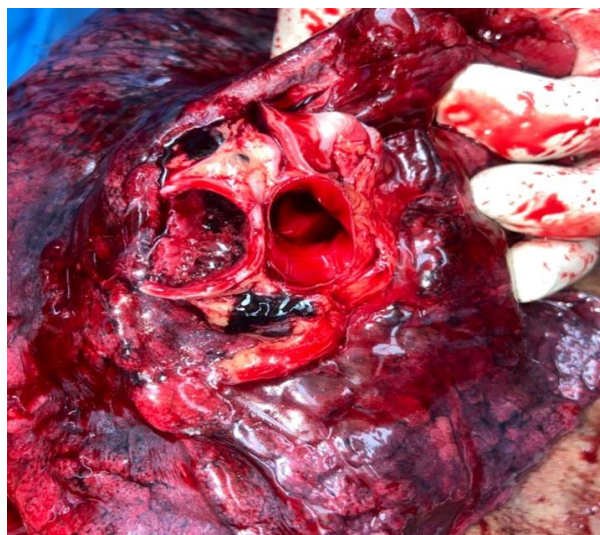


Figure 3. Blackish soot particles within the bronchus

Importantly, fire deaths are rarely attributable to CO in isolation. Combustion of synthetic bedding, foam, wool, silk and household plastics releases hydrogen cyanide (HCN) alongside CO, and HCN is now recognized as an independent and potentially lethal contributor to fire deaths rather than a minor co-toxicant [14-16]. A case report evaluating both HCN and COHb in a fire fatality found toxic to lethal concentrations of both agents and concluded that HCN made an additive contribution to death alongside CO [14]. Using cardiac blood cyanide measurement combined with pharmacokinetic modelling, a Japanese autopsy series of 29 fire deaths detected cyanide in over three quarters of cases, with several cases implying extremely high, rapidly lethal HCN exposure [15]. Conversely, a retrospective review of 42 indoor fire autopsies in Istanbul found that cyanide testing was omitted in a third of cases and, even when requested, was positive in only a minority despite carboxyhaemoglobin being positive in every case highlighting how inconsistently cyanide is screened for in routine forensic practice, and the medico-legal risk this creates when cyanide is in fact a contributory or principal cause of death [16]. Taken together, this literature indicates that COHb estimation, cyanide analysis and, ideally, a multidisciplinary reconstruction of the scene are together needed to reliably establish cause and manner of death in fire fatalities [14-16, 21].

The present series has one key limitation that these findings highlight. CO exposure was reported by the laboratory only qualitatively (positive/negative), without a quantitative COHb saturation value; this precluded correlating COHb level with the severity of hypoxic organ change, and limited our ability to identify “low-saturation”

cases in which CO alone may not fully explain death. A full poison panel, including cyanide/HCN analysis, was performed in all six victims and was negative for cyanide, effectively excluding a contributory or primary role for HCN despite the fire involving a foam mattress and other synthetic household material in a confined space; CO was the only asphyxiant detected. We therefore note the absence of quantitative COHb estimation, rather than untested cyanide, as the main gap in this series, and recommend routine quantitative COHb testing in future fire fatality autopsies alongside the standard poison panel.

From a public-health standpoint, this case illustrates how a single preventable ignition source an unattended mosquito coil combined with inadequate ventilation and the absence of early warning devices, was sufficient to cause six deaths across three families. Effective prevention requires an integrated approach: fire-safety education, safe placement of mosquito coils and candles away from bedding and combustible material, functioning smoke detectors, and given how frequently CO rather than flame is the actual killer, wider deployment of CO detectors, particularly in densely populated, poorly ventilated urban housing.

CONCLUSION

This case series reinforces the central forensic principle that, in fire-related deaths, careful autopsy examination, assessing soot distribution, cherry-red discolouration, and airway and pulmonary changes combined with directed toxicological analysis is essential to correctly establish cause of death. Because CO is rarely the only asphyxiant present, this analysis should ideally include a full poison panel with cyanide/HCN testing, as was performed in the present series, alongside quantitative COHb estimation rather than a qualitative CO report alone; without quantitative COHb data, the severity of hypoxic exposure cannot be fully correlated with the autopsy findings, even where, as here, other asphyxiants such as hydrogen cyanide have been actively excluded [14-16]. This is particularly important when external burns are minor or absent, since asphyxiant gas poisoning is easily overlooked by inexperienced examiners and its clinical and postmortem features are frequently non-specific. Accurate diagnosis is equally important for victim identification and case reconstruction in mass fatality fire incidents. Beyond the mortuary, this tragedy underscores concrete preventive priorities: regular maintenance of electrical systems, safe placement and supervision of mosquito coils and candles, clear and accessible evacuation routes, and routine fire safety inspections in densely occupied residential buildings. Installation of interlinked smoke and CO detectors can provide early warning particularly at night, when occupants are asleep and least able to detect danger and, together with prompt coordination between emergency services, has the potential to prevent similar mass-casualty domestic fires in the future.

Ethical Considerations

All examinations were conducted as part of routine

medicolegal investigation under the applicable statutory framework governing unnatural deaths in India, with due maintenance of chain of custody. Identifying details of the deceased have been withheld, and clinical photographs are presented without identifiable features, to preserve confidentiality. No additional consent beyond the statutory medico legal process was required for this retrospective case series report.

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