Silent Combustion, Fatal Consequences: An Autopsy-Based Case Report of Carbon Monoxide Poisoning from a Smoldering Mattress in an Enclosed Room

Authors: Dr Hemalatha P[1*] , Dr. M.Muralidharan[2], Dr.M.Sreenivasan[3]

Affiliation:

1. Postgraduate Resident, ORCID: 0009-0006-7094-1596
2. Postgraduate Resident, ORCID: 0009-0003-9662-9689
3. Professor & Head

Department of Forensic Medicine and Toxicology,
Chettinad Hospital and Research Institute,
Chettinad Academy of Research and Education,
Kelambakkam 603 103, Tamil Nadu, India.

***Corresponding author:** Dr. Hemalatha P, Department of Forensic Medicine and Toxicology, Chettinad Hospital and Research Institute, Kelambakkam, Tamil Nadu, India.

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ABSTRACT

Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion and a common cause of accidental fire-related deaths. We report a 35-year-old man who, after consuming alcohol and smoking, slept in a closed, air-conditioned room and was found unresponsive the next morning with a smoldering mattress. Autopsy showed soot around the nostrils and in the larynx, trachea and bronchi, a minor burn over the lower limbs, and cherry-red discoloration of tissues and organs. Postmortem blood showed a carboxyhemoglobin (COHb) saturation of 56%. The cause of death was opined as asphyxia due to carbon monoxide poisoning, and the manner was accidental. This case shows that soot and cherry-red discoloration are useful but not

confirmatory, and that diagnosis requires scene investigation, complete autopsy and COHb estimation.

Keywords: Carbon monoxide poisoning; Soot; Smoldering mattress; Asphyxia; Autopsy

1.INTRODUCTION: Carbon monoxide (CO) is a major and ubiquitous component of fires due to incomplete combustion of organic compounds or carbon. CO is a colorless, odorless, tasteless, and nonirritating gas, which makes it difficult for those who are exposed to detect it. Because CO lacks sensory warning properties, it is commonly known as the "silent killer." [1,2] As the specific gravity of CO is 0.97, it is slightly lighter than air.[3] Carbon monoxide (CO) is produced by the incomplete combustion of carbon-containing fuels and organic materials, including gasoline, coal, wood, propane and natural gas. Common sources of occupational exposure include fuel-powered engines, such as those in motor vehicles, forklifts and generators; fuel-burning heating systems; and industrial installations, including coke ovens and blast furnaces. In domestic settings, CO exposure commonly arises from faulty, poorly maintained or inadequately ventilated gas appliances, malfunctioning heating systems, improperly ventilated vehicles, generators, grills, stoves and residential fires.

CO is a gas at normal room temperature, and is inhaled from the lungs into the blood stream. CO has approximately 200 to 250 times greater affinity for hemoglobin than oxygen, resulting in carboxyhemoglobin formation and impaired oxygen transport and delivery to tissues, inhibits mitochondrial oxidative phosphorylation, and inactivates cytochrome oxidase thereby causing cellular hypoxia.[4,5] CO also binds to myoglobin in myocardium and skeletal muscle, causing dysfunctional tissue oxygen transport. In myocardium, this results in cardiac dysfunction. CO poisoning leads to apoptosis in myocardial cells. Neurotoxicity following CO exposure involves apoptosis and intracellular oxidative stress. Tissue hypoxia due to CO potentiates vascular permeability and

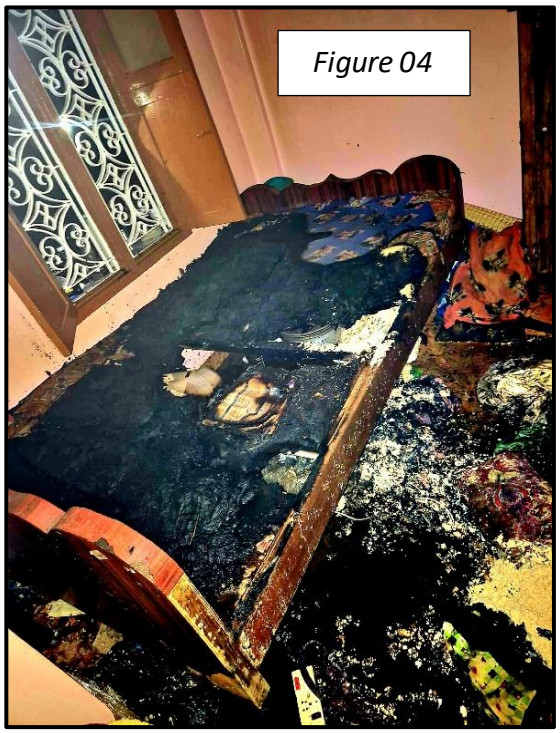
causes increased accumulation of interstitial fluid with decreased circulating blood volume (hemoconcentration) affecting multiple organs. This includes brain edema with neurological symptoms and disorders of consciousness; pulmonary edema with respiratory failure; decreased myocardial contractility, arrhythmias and heart failure; and renal failure.[6,7,8,9,10,11] Clinical manifestations vary based on percentage of CO-HB formation which in turn depends on the intensity and duration of exposure and may include headache, dizziness, weakness, nausea, confusion, disorientation and visual disturbances whereas severe exposure may progress to loss of consciousness, convulsions, coma and death. The formation of CO-Hb in blood depends on a wide variety of factors, including the concentration of inspired CO, duration of CO exposure, pulmonary ventilation, exercise and health status[12,13] It is more dangerous especially with people who are asleep, unconscious, drunk or drugged.[14] Carbon monoxide produces an estimated 40,000 emergency department visits each year.[5]

2.CASE HISTORY : As per the police inquest, the deceased, a 35-year-old man, was residing with his wife and three sons. On 14 October 2026, he reportedly consumed alcohol, returned home in the evening and went to sleep after eating food and consuming alcohol as well as smoking cigarette. The following morning, people noticed that there was smoke emanating from his residence window. Upon checking the place, he was found unresponsive inside the house. Family members reported that he had been sleeping in a closed, air-conditioned room, where a mattress was found smoldering. He was subsequently taken to the hospital, where he was declared dead. A police investigation was initiated and the body was brought for medico-legal autopsy.

3.POSTMORTEM FINDINGS:

3.1. External Examination:

The body of a 35 years male showed soot-like black particulate material around the nostrils. A minor burn injury was present over the lower limbs. Blackish, sticky, soot-like particulate material was adherent to the skin of both hands, giving them a soiled appearance.(Figure 1a,1b,1c) These findings were considered in correlation with the reported scene circumstances and the possibility of smoke exposure.





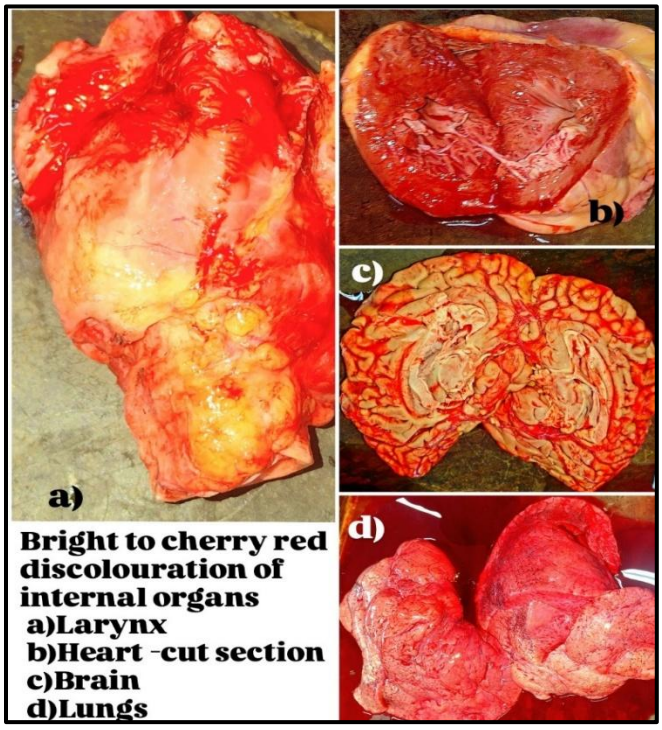
3.2. Internal Examination: A cherry-red to bright red discoloration of the mucous membranes, tissues and internal organs were seen. The lungs were congested with pink fluid blood and the trachea showed the black colored soot particles adherent to the tracheal walls(Figure3). There were no internal injuries found on the body.

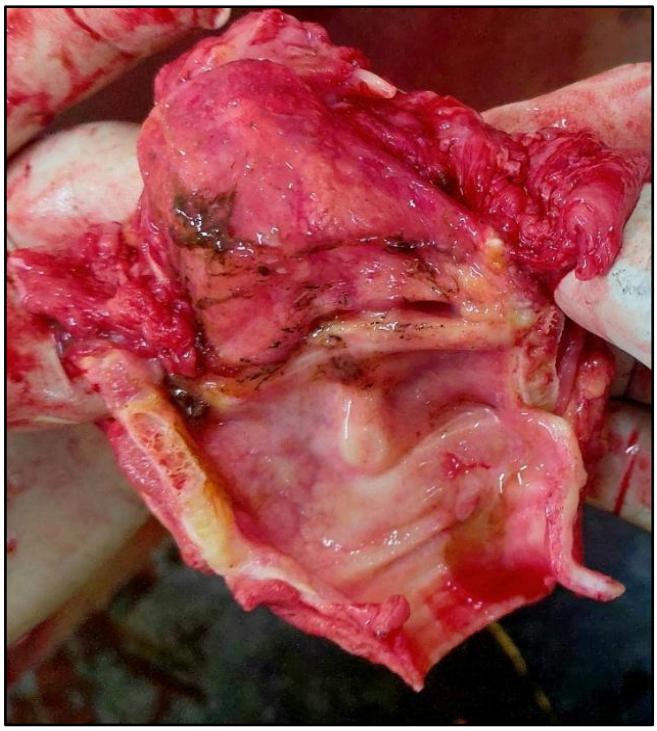
Larynx (Figure 2a): The laryngeal region shows prominent red discoloration of the exposed tissues. On dissection, blackish soot particles seen and traced up to the terminal bronchiole.

Heart (Figure 2b): On cut section, the myocardium exhibits prominent reddish to bright red discoloration.

Brain (Figure 2c): The cut surface of the brain shows prominent red discoloration of the cerebral tissue and visible blood vessels.

Lungs (Figure 2d): The lungs appear reddish-pink, with a mottled appearance over the pleural surfaces.





3.3. Scene correlation: The scene photograph (Figure 4)shows a mattress with visible burn damage and charred areas, consistent with exposure to smoldering combustion. This finding correlates with the reported circumstances of the deceased being found in a closed, air-conditioned room.

3.4 Toxicological Analysis: Blood was taken from the heart and preserved under a layer of liquid paraffin and sent for the estimation of the COHb concentration. Carboxyhemoglobin (COHb) was detected in the blood sample (Exhibit A) at a concentration of 56% saturation. After consideration of the autopsy findings, scene investigation and blood COHb concentrations, the cause of death was

opined as asphyxia due to carbon monoxide poisoning. The manner of death was accidental.

4)DISCUSSION:

4.1. Pathogenesis: Carbon monoxide toxicity is multifactorial and extends beyond simple displacement of oxygen from hemoglobin. After inhalation, CO diffuses across the alveolar-capillary membrane and binds avidly to hemoglobin, producing functional anemia and a leftward shift of the oxyhemoglobin dissociation curve. It also binds to other heme-containing proteins, such as myoglobin and mitochondrial cytochrome c oxidase, causing direct cellular toxicity, oxidative stress and inflammatory injury. The resulting tissue hypoxia affects the brain and heart most severely because of their high oxygen demand. The principal mechanisms are described system-wise below.

Hb-Specific Effects: CO binds to hemoglobin (Hb) in the blood with high affinity, forming COHb. CO competes with oxygen for binding to Hb and, by displacement of oxygen, reduces oxygen carrying capacity. CO binding to Hb also stabilizes the relaxed, high-affinity quaternary state of Hb (known as R-state), increasing the affinity for oxygen of other sites within the Hb tetramer, and further reducing oxygen release and delivery. Neither the clinical severity nor the clinical improvement of CO-poisoned patients directly correlates with the blood COHb level or COHb clearance. In addition to Hb, CO binds to other heme-containing proteins, including myoglobin in heart and skeletal muscle, mitochondrial cytochrome c oxidase (COX; complex IV), and others. COHb levels of 2% or greater in nonsmokers and 10% or greater in smokers are considered abnormal and may produce symptoms.[5,15,16]

Mitochondrial Inhibition and Free Radical Generation: CO inhibits mitochondrial respiration by binding the ferrous heme a_3 in the active site of COX, effectively shutting down oxidative phosphorylation, similar to the effects

of cyanide and nitric oxide (NO). With COX inhibited, oxidative phosphorylation slows down, decreasing ATP production in tissues, such as the brain or heart. Other complexes in the electron transport chain continue to shuttle electrons, generating superoxide, leading to further damage of cells and tissues.[17,18,19]

Platelet and Inflammatory Effects: Excess CO activates platelets by displacement of NO from platelet surface hemoproteins. The inflammatory cascade driven by NO and ROS contributes to neurological and cardiac injuries from CO poisoning[20]

Mechanisms of Brain Ischemia: CO-mediated reductions in oxygen delivery and mitochondrial oxidative phosphorylation produce ischemic and anoxic brain injury, leading to cognitive deficits in survivors. Brain injury from ischemia can occur from excitotoxicity, acidosis, ionic imbalance and depolarization, oxidative stress, nitrative stress, inflammation, and apoptosis.[21]

Neurological and affective sequelae: Symptoms include impaired memory, cognitive dysfunction, depression, anxiety, and/or vestibular and motor deficits.[22,23] The severity of initial symptoms does not necessarily correlate with the development of longer-term neurological issues. Low-level, chronic exposure can also lead to neurological and cognitive deficits that do not resolve after removal from the CO source, suggesting neurological damage even at low levels of COHb and environmental CO.

Cardiovascular effects: CO-induced mitochondrial inhibition could cause a stunned myocardium–like syndrome (with hypokinesia in the setting of unobstructed coronary arteries).[24]CO poisoning increases risk of developing an arrhythmia. Clinical manifestations of carbon monoxide poisoning vary according to blood carboxyhemoglobin (COHb) levels. Levels of approximately 5–10% may be associated with subtle cognitive impairment, while levels of 10–20% may cause forehead tightness and headache. Levels of 20–30% may produce

headache with throbbing in the temples, whereas levels of 30–40% may be associated with severe headache, weakness, dizziness, visual disturbances, nausea, vomiting and collapse. At levels of approximately 40–60%, the likelihood of collapse, unconsciousness and coma increases. These ranges are approximate, and clinical severity may vary depending on the duration of exposure and individual susceptibility.

4.2. Significance of soot particles in respiratory tract:

The presence of soot particles within the tracheal lumen indicates the entry of smoke-containing material into the respiratory tract and supports the possibility of antemortem smoke inhalation. Soot deposition in the lower respiratory tract, particularly within the trachea and bronchi, supports active inhalation during the fire, although it is not absolute proof of vitality in isolation. In the present case, soot observed in the nostrils and larynx, when correlated with the burnt mattress and the enclosed-room circumstances, supports exposure to smoke generated by combustion. However, soot consists of particulate combustion products and does not independently establish carbon monoxide exposure, its concentration, or the cause of death. Therefore, the diagnosis of carbon monoxide poisoning requires correlation with postmortem carboxyhemoglobin (COHb) estimation, scene investigation and other relevant autopsy findings.

4.3. Differential diagnosis & Toxicological confirmation:

The differential diagnoses included smoke inhalation injury, cyanide poisoning, oxygen-depletion asphyxia, fatal thermal injury, acute alcohol intoxication and natural causes of sudden death. CO poisoning was suspected based on the scene circumstances; however, confirmation requires correlation with postmortem COHb estimation, complete autopsy findings and toxicological investigations.

Exposure to levels as low as 10 ppm of CO can lead to detectable COHb levels of approximately 2% . Normal Levels typically below 3% in non-smokers, and

roughly 5%–10% in regular or heavy tobacco smokers. Toxic/Lethal Levels exceeding 40%–50% are generally considered severe or fatal, though susceptibility varies based on age and pre-existing heart conditions[5,25]. Previous studies also show that the main cause of fatal CO poisoning is due to the collection of this gas in an ill ventilated room. Homicidal deaths are very uncommon. In the present case too, the deceased was found in an ill-ventilated room, after consuming alcohol, with a partially burnt mattress that probably ignited from a cigarette.

5. CONCLUSION: Carbon monoxide poisoning is the most common human poisoning, with no available antidotal therapy. Though CO poisoning remains common but it is often the unrecognized cause of accidental death, particularly in enclosed, poorly ventilated spaces. In the present case, a smoldering mattress, likely ignited by a cigarette in an intoxicated individual, led to a fatal COHb saturation of 56%. Cherry-red discoloration of tissues and organs and soot in the airways are useful pointers, but neither confirms CO exposure on its own. The diagnosis requires correlation of scene findings, autopsy findings and postmortem COHb estimation. Proper collection and preservation of blood samples is essential for medico-legal confirmation. Public awareness of fire safety, safe smoking habits, adequate ventilation and CO/smoke detectors is key to preventing such deaths.

6. CONFLICTS OF INTEREST: The authors declare that there is no conflict of interest.

7. SOURCE OF FUNDING: None.

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