CASE REPORT

The Lethal Lullaby: Sleeping into a carbon monoxide trap

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ABSTRACT

Background: Carbon monoxide (CO) poisoning remains a significant yet often under-recognized cause of accidental death worldwide, particularly during sleep when early symptoms go unnoticed. CO is a colorless, odorless gas that produces tissue hypoxia through the formation of carboxyhemoglobin and direct cellular toxicity. Combined exposure with ethanol may potentiate central nervous system depression and delay arousal, increasing lethality.

Case Presentation: We report the case of a 44-year-old male who was brought unresponsive to a tertiary care hospital in Belagavi, Karnataka. On arrival, he had no palpable pulse, unrecordable blood pressure, fixed dilated pupils, and absent spontaneous respiratory efforts. Electrocardiography revealed an isoelectric flat line, and the patient was declared dead on arrival. A medico-legal autopsy revealed no external injuries or traumatic pathology. Internal examination showed congested organs with yellowish patches on the liver surface. Toxicological analysis detected ethyl alcohol and carbon monoxide in blood and viscera. Blood alcohol concentration was 428.96 mg/100 mL, and carboxyhemoglobin saturation was 17.30%. Histopathology revealed micronodular cirrhosis of the liver, myocardial hypertrophy, and advanced coronary atherosclerosis. The final cause of death was concluded as respiratory failure due to carbon monoxide poisoning, compounded by ethanol consumption.

Conclusion: This case highlights the lethal synergy between carbon monoxide exposure and alcohol intoxication during sleep. It emphasizes the importance of toxicological screening, meticulous autopsy evaluation, and public health measures for CO exposure prevention.

Keywords: Carbon monoxide poisoning; Carboxyhemoglobin, Ethanol intoxication; Forensic autopsy; Sudden death;

How to cite this article: Stephen A, Honnungar RS, Bannur V, Koulapur VV. The Lethal Lullaby: Sleeping into a Carbon Monoxide Trap. Int J Drug Deliv Technol. 2026;16(31s):1094-1097. DOI: 10.25258/ijddt.16.31s.120

Source of support: Nil., **Conflict of interest:** None

Introduction

Carbon monoxide (CO) poisoning is a leading cause of accidental poisoning-related mortality worldwide and continues to pose a major public health challenge, particularly in low- and middle-income countries [1]. CO is a colorless, odorless, and non-irritant gas produced by incomplete combustion of carbon-containing fuels such as coal, wood, kerosene, liquefied petroleum gas, and petrol [2]. Because of its undetectable sensory properties, exposure frequently occurs without warning, especially during sleep, resulting in delayed recognition and fatal outcomes.

The toxic effects of carbon monoxide are primarily mediated through its high affinity for hemoglobin, forming carboxyhemoglobin (COHb), which impairs oxygen transport and delivery to tissues [3]. CO binds hemoglobin with an affinity approximately 200–250 times greater than oxygen, leading to functional anemia and leftward shifting of the oxyhemoglobin dissociation curve. In addition to hypoxic stress, CO exerts direct cytotoxic effects by binding to myoglobin, cytochrome oxidase, and other heme-containing enzymes, thereby impairing oxidative phosphorylation and cellular respiration [4].

Clinical manifestations of CO poisoning vary depending on exposure duration, concentration, and host factors. Early symptoms include headache, dizziness, nausea, and fatigue, while higher levels may cause confusion, syncope, seizures, coma, and death [5]. Importantly, individuals exposed during sleep may never experience prodromal symptoms, making CO poisoning particularly dangerous in enclosed residential environments. Numerous fatal cases are discovered only after prolonged exposure, often presenting as sudden unexplained deaths.

Alcohol consumption is a recognized risk factor that exacerbates the lethality of CO poisoning. Ethanol acts as a central nervous system depressant, reducing arousal responses and impairing protective reflexes that would otherwise prompt escape from a toxic environment [6].

Furthermore, alcohol may delay symptom recognition and accelerate progression to respiratory failure. Forensic investigations have repeatedly documented co-existence of alcohol intoxication in fatal CO poisoning cases, underscoring its contributory role .

From a forensic perspective, diagnosing CO poisoning requires careful correlation of scene findings, autopsy observations, and toxicological analysis. Classical cherry-red lividity is infrequently observed, and gross autopsy findings are often nonspecific, including visceral congestion and cerebral edema [7]. Measurement of blood carboxyhemoglobin levels remains the cornerstone of diagnosis, supplemented by histopathological evaluation and chemical analysis of viscera.

The present case illustrates a fatal instance of carbon monoxide poisoning occurring during sleep, complicated by significant ethanol intoxication and underlying chronic liver disease. Through comprehensive clinical documentation, medico-legal autopsy, toxicological examination, and histopathology, this report highlights the silent and synergistic lethality of carbon monoxide and alcohol exposure, reinforcing the need for heightened awareness, preventive strategies, and forensic vigilance.

Case Presentation

A 44-year-old male was brought unresponsive to the emergency department of KLES Dr. Prabhakar Kore Hospital & MRC, Belagavi, on 16 April 2025. According to available records, the patient was brought unconscious with no signs of life. On examination, carotid pulse was not palpable, blood pressure was unrecordable, pupils were fixed and dilated, and there were no spontaneous respiratory efforts. Electrocardiography demonstrated an isoelectric flat line. The patient was declared dead on arrival at 4:13 PM. As the death was sudden and unexplained, the case was registered as a medico-legal death, and the body was sent for post-mortem examination. External examination revealed a well-built and nourished male body measuring approximately 170.6 cm in length, with post-mortem staining present. No external injuries, trauma, or signs of violence were noted. Bluish nail discoloration was observed. Internal examination revealed intact scalp, skull, brain, spinal cord, neck structures, thoracic cage, and abdominal cavity. Both lungs were intact and congested, with frothy fluid noted in airways. The heart was intact but showed left ventricular hypertrophy. The liver was enlarged, congested, and showed yellowish patches over the surface. Other organs were congested but structurally intact.Viscera and blood samples were preserved and forwarded to the Regional Forensic Science Laboratory, Belagavi. Toxicological analysis detected ethyl alcohol and carbon monoxide in blood and viscera samples. Blood alcohol concentration was 428.96 mg/100 mL, and carboxyhemoglobin saturation was 17.30%. Histopathological examination revealed micronodular cirrhosis with fatty change and ballooning degeneration of hepatocytes. Cardiac examination showed myocardial hypertrophy with advanced coronary atherosclerosis. Based on autopsy, toxicology, and histopathology findings, the final cause of death was opined as respiratory failure due to carbon monoxide poisoning, compounded by ethanol consumption.

Discussion

Carbon monoxide poisoning is a well-recognized but frequently underdiagnosed cause of sudden death, particularly when exposure occurs during sleep. The present case exemplifies the classic forensic challenge posed by CO poisoning, where external findings are minimal and diagnosis relies heavily on toxicological confirmation and contextual interpretation.Hampson and Weaver emphasized that CO poisoning continues to be one of the leading causes of unintentional poisoning deaths globally, with many victims succumbing during sleep due to lack of sensory warning and rapid hypoxic collapse [8]. In the present case, the victim was found unresponsive with no prodromal symptoms reported, supporting this mechanism.The detected carboxyhemoglobin level of 17.30% is lower than levels traditionally associated with fatality; however, death can occur at lower concentrations in the presence of contributing factors such as alcohol intoxication, anemia, or underlying cardiovascular disease [9]. Ernst and Zibrak [10] highlighted that COHb levels do not always correlate directly with severity of clinical outcome, particularly when exposure is prolonged or combined with other depressant substances.Alcohol intoxication plays a critical synergistic role in CO-related deaths. Levine et al. [11]demonstrated that ethanol significantly increases the risk of fatal CO poisoning by impairing consciousness, delaying escape, and worsening hypoxic injury. In the present case, the blood alcohol concentration of 428.96 mg/100 mL represents severe intoxication, sufficient to cause profound CNS depression. This level likely prevented arousal during early CO exposure, facilitating prolonged inhalation and respiratory failure.

Underlying pathological conditions further compounded vulnerability. Histopathological evidence of micronodular cirrhosis suggests chronic alcohol use, which may impair hepatic metabolism and systemic resilience to hypoxia. Additionally, myocardial hypertrophy and significant coronary atherosclerosis, as observed in this case, are known to reduce tolerance to hypoxic stress. Weaver [12] reported that individuals with pre-existing cardiovascular disease are at increased risk of sudden death even at modest COHb levels.

Autopsy findings in CO poisoning are often nonspecific. Knight and Saukko [13] described visceral congestion, pulmonary edema, and organ congestion as common but non-diagnostic features, all of which were observed in the present case. The absence of trauma and natural disease sufficient to cause death further strengthened the conclusion of toxicological causation.Forensic toxicology remains the definitive diagnostic modality. Detection of CO and ethanol in blood and viscera, along with quantification of COHb, provided objective evidence supporting the cause of death. The presence of frothy fluid in airways and pulmonary congestion is consistent with hypoxic respiratory failure secondary to CO exposure [14].This case underscores the importance of considering carbon monoxide poisoning in sudden unexplained deaths, especially when alcohol intoxication is present. It also highlights the need for public health interventions such as CO detectors, safe fuel usage, and awareness campaigns targeting high-risk populations. From a medico-legal standpoint, meticulous autopsy, comprehensive toxicological screening, and histopathological correlation are essential to avoid misclassification of such deaths.

Conclusion

This case demonstrates the silent lethality of carbon monoxide exposure during sleep and its dangerous interaction with alcohol intoxication. Even moderate carboxyhemoglobin levels can prove fatal when compounded by ethanol-induced central nervous system depression and underlying cardiovascular pathology. Comprehensive forensic evaluation is critical for accurate diagnosis, while preventive strategies remain the cornerstone for reducing carbon monoxide–related mortality.

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