JOURNAL OF INTERNATIONAL CRISIS AND RISK COMMUNICATION RESEARCH
ISSN: 2576-0017
2025, VOL 8, NO 1

Forensic Toxicology of Carbon Monoxide in Saudi Arabia

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Abstract
Carbon monoxide (CO) poisoning is a major global health issue, especially in the field of forensic medicine. CO is a colorless, odorless, and tasteless gas that, when inhaled in high concentrations, can lead to tissue hypoxia and death by binding to hemoglobin and reducing the blood's oxygen-carrying capacity. Every year, CO poisoning leads to a significant number of fatalities, particularly in winter when charcoal is used for heating in poorly ventilated areas. Additionally, CO is a major contributor to fire-related deaths, especially in homes where a fire is contained before it spreads, allowing the gas to accumulate indoors. Saudi Arabia's CO poisoning is notably more prevalent during the winter months due to malfunctioning heating systems and inadequate ventilation, which result in the build-up of CO in indoor spaces. Despite its relevance, there is a significant lack of case-specific forensic toxicological data on CO poisoning in Saudi Arabia. This makes the Kingdom an interesting location for studies on CO poisoning, as there is currently limited published data on the topic. This review article aims to discuss CO poisoning from a clinical perspective, exploring Sources and toxicological characteristics of CO, while also shedding light on its impact on public health in Saudi Arabia.

Keywords: Carbon Monoxide Poisoning, Forensic Toxicology, Hypoxia, Saudi Arabia Health Risks,and Air Pollution

Introduction

One of the most frequent causes of unintentional poisoning, carbon monoxide has severe forensic and health effects. CO is a tasteless, colorless, and non-irritating gas that is sometimes referred to as a "silent killer". CO has a specific gravity of 0.97, making it somewhat lighter than air. and odorless gas that is produced when carbon-containing objects do not completely burn. As a result, it is found in many homesteads and workplaces (Rose, et al. 2017) (Can, et al. 2019).

In forensic practice, a CO examination is performed in any fire-related death to ascertain whether the fire caused the victim's death or if the fire was staged to alter the crime scene and the cause of death. The presence of CO in the postmortem blood samples is a crucial factor in this assessment. A biomarker of smoke CO inhalation, carboxyhemoglobin (COHb), is thought to be a reliable indicator of when a person died, either prior to or during a fire. (Melez, et al. 2017) (Sully, et al. 2018).

In Saudi Arabia, there are multiple causes of CO poisoning. Most of the year is hot in Saudi Arabia, so heat-generating equipment is not necessary. Electrical failure or short circuits are a primary source of fire accidents in Saudi Arabia, as indicated by the Saudi General Authority for Statistics, based on data from the General Administration of the Saudi Civil Defense report covering the years 2010 to 2019.In comparison to the other 14 regions of Saudi Arabia, the Makkah Region has the greatest rate of fire incidents despite having the second-largest population, according to the survey. (Al-Asmari, et al. 2021)

Saudi Arabia, with its vast desert regions, is subjected to elevated levels of particulate matter because of dust storms and building activities. Strong winds lift sand into the atmosphere, leading to heavy aerosol loading (Khodeir, et al. 2021) (Alam, et al. 2014) (Farahat, et al. 2016). Additionally, transportation and dense vehicle traffic on the country's roads contribute to the release of greenhouse gases (Rahman, et al. 2017) and volatile organic compounds. According to (Lim, et al. 2018), aerosol long-range transport caused by oil combustion in the northeastern oil deposits of Saudi Arabia affects air quality in the western regions, including Makkah. Due to limited air pollution monitoring, most studies on air pollutants in the country focus primarily on important urban regions like Riyadh (Rushdi, et al. 2013) (Alharbi, et al. 2015), Makkah (Al-Jeelani, et al. 2009) (Othman, et al. 2010), Yanbu (Al-Jeelani, et al. 2014) (Khalil, et al. 2016), and Jeddah (Porter, et al. 2014) (Rehan, et al. 2022).

Carbon monoxide enters the bloodstream and forms carboxyhemoglobin (COHb), which has a higher affinity than oxygen and hemoglobin. This reduces the oxygen carrying capacity of hemoglobin, leading to tissue and organ hypoxia (Ashcroft. 2019). Blood vessel compensatory dilation and extravasation occur, and ATP is consumed in an oxygen-free environment, causing sodium ion accumulation in cells and tissue edema. Severe hypoxia can lead to pathophysiological changes, and CO poisoning can ultimately cause death. The measurement of %COHb in the corpse is a crucial reference index for determining death, but identifying the cause of death in CO poisoning cases has become more challenging due to factors like postmortem time, temperature, corruption, body pathology, and individual differences. (Jian-qiang. 2023).

CO poisoning is a critical condition that requires rapid recognition and intervention. Due to its silent nature, widespread public education and preventive measures are vital in minimizing the risk of exposure. Therefore, this review article seeks to examine carbon monoxide (CO) poisoning from a clinical standpoint, focusing on Sources and toxicological effects of CO. Additionally, it highlights the public health implications of CO poisoning, particularly in the context of Saudi Arabia.

Literature Review

1. Sources of CO

CO, a greenhouse gas, is formed from incomplete combustion of organic compounds. It is primarily found in house fires, fuel combustion, improper heating or cooking equipment, exhaust gas from internal combustion engines, and industrial accidents. The air near a home fire has a maximum CO content of about 5%. Inhaling diesel engine exhaust, which contains 0.01–0.06% CO, does not result in lethal CO poisoning. Since three-way catalytic converters were introduced, the amount of CO in gasoline engine exhaust has dropped. (Kinoshita, et al. 2020)

In contrast to sources in the western world, the primary causes of CO poisoning in Saudi Arabia are determined to be motor fuel and charcoal. nations (automobiles, smoke from fumes or fires, or from malfunctioning heating systems), and lack of fresh air. (Al-Asmari et al. 2021) discovered in their antemortem forensic analysis that CO emissions from broken household equipment continued to be the most prevalent in Jeddah, especially during the colder months when household heating appliances are used often. The significant contribution of local sources to CO poisoning in Dammam has also been highlighted by (Aldossary et al. 2015), highlighting the regional risk of both poorly maintained and inappropriately fitted appliances. In Alqurayyate, Car exhaust was most frequently cited as the source of CO exposure among carbon monoxide poisoning (COP) deaths 36.8%, followed by charcoal heaters 31.6% (Attaia, et al. 2020). In other places, industrial gas releases and vehicle exhaust are the main causes of poisoning. Similar studies were conducted in Greece by (Stefanidou et al. 2012) and Portugal by (Costa et al. 2019). discovered that vehicle emissions in metropolitan areas were the primary source of CO exposure in these locations. Furthermore, (Ruas et al. 2014) emphasized the risks connected to the use of charcoal, particularly in areas with inadequate ventilation, as is the case in Saudi Arabia and other states. In the culture, some households cook, heat, and stir inside using charcoal, which increases the danger of carbon monoxide exposure. It has gotten worse in Saudi Arabia because of cultural customs that require these areas to be enclosed within homes.

2. The toxic action of carbon monoxide

The toxic action of carbon monoxide (CO) is primarily due to its ability to bind with hemoglobin, forming carboxyhemoglobin (COHb), which reduces the capacity of blood to carry oxygen. Here's a breakdown of how CO affects the body:

Binding to Hemoglobin: CO has a much higher affinity for hemoglobin than oxygen (about 200-250 times greater). When CO is inhaled, it forms carboxyhemoglobin (COHb) when it binds to hemoglobin, which prevents oxygen from binding to hemoglobin and reduces the oxygen-carrying capacity of blood. This leads to tissue hypoxia (lack of oxygen), as oxygen is not efficiently delivered to tissues and organs.

Impairment of Cellular Respiration: CO also interferes with mitochondrial cytochrome c oxidase, an enzyme essential for oxidative phosphorylation in cells. This further impairs cellular energy production, especially in tissues with high oxygen demands, like the brain and heart. The inhibition of cytochrome c oxidase can lead to the accumulation of metabolic byproducts such as lactate, causing lactic acidosis.

Effects on the Cardiovascular and Nervous Systems: Due to reduced oxygen delivery, vital organs, particularly the brain and heart, are severely affected. Symptoms of CO poisoning can include headache, dizziness, confusion, chest pain, and in severe cases, loss of consciousness, seizures, or death. The brain, being highly sensitive to oxygen deprivation, is particularly vulnerable, leading to neurological effects like memory loss, cognitive dysfunction, and in extreme cases, irreversible damage.

Shifting the Oxygen-Hemoglobin Dissociation Curve: The presence of CO also causes a leftward shift in the oxygen-hemoglobin dissociation curve, making it harder for oxygen to be released from hemoglobin to tissues, exacerbating hypoxia in tissues despite normal oxygen levels in the lungs. (Baksevice, et al. 2024)

3. Toxicokinetic of CO

CO enters the bloodstream through the lungs and is a gas at room temperature. Because of its 230–270 times stronger affinity for hemoglobin (Hb) than for oxygen, it forms CO-Hb in erythrocytes. Concentration, exposure length, lung ventilation, exercise, and health state are some of the variables that affect the production of CO-Hb. After inhalation, less than 0.1% of CO is transformed into carbon dioxide, leaving it nearly entirely unoxidized. Myoglobin in the heart and skeletal muscle are bound by CO, which has a strong affinity for other hemeproteins like myoglobin and cytochrome c oxidase. Tissues absorb up to 15% of the body's total CO, which permits CO to flow from organs into the blood as CO-Hb saturation falls. Depending on the disease condition, CO-Hb saturation might range from less than 2% in healthy, nonsmoking participants to 4-6% in hemolytic anemia cases and almost 10% in other situations. When methylene chloride, a solvent used to remove paint or varnish, is converted to CO, it can cause acute CO poisoning with CO-Hb saturation as high as 50%. With a half-life of roughly 4-5 hours under room air ventilation at sea level, 80 minutes breathing 100% oxygen at norm baric pressure, and 23 minutes breathing oxygen at 3 atmospheres absolute, oxygen administration can lower CO-Hb saturation. (Kinoshita, et al. 2020)

4. Toxicity and pathophysiology of CO poisoning

Acute CO poisoning leads to tissue hypoxia, a toxic effect caused by the formation of CO-Hb. This decreases oxygen transport capacity, resulting in insufficient oxygenation at the tissue level. CO binds to hemoglobin subunits, increasing the affinity for oxygen molecules, which shifts the oxygen-hemoglobin dissociation curve to the left, inhibiting oxygen dissociation in the low-oxygen region and causing tissue hypoxia. The bond between CO and hemoglobin is strong, but it can be broken reversibly. CO also binds to myoglobin in myocardium and skeletal muscle, causing dysfunctional tissue oxygen transport and cardiac dysfunction. CO poisoning may also impair cardiac and neurological functions. Apoptosis is a key factor in heart failure, and CO poisoning leads to apoptosis in myocardial cells. Neurotoxicity following CO exposure involves apoptosis and intracellular oxidative stress. erythropoietin, resveratrol, and hyperbaric oxygen can reduce dysfunction in the myocardium and brain by suppressing apoptosis or through other pathways. CO-induced tissue hypoxia increases vascular permeability and decreases circulating blood volume, affecting multiple organs, including brain edema, respiratory failure, decreased myocardial contractility, arrhythmias, heart failure, and renal failure. (Kinoshita, et al. 2020)

5. Symptoms and management of CO poisoning

Carbon monoxide (CO) exposure can lead to a few difficulties, such as major cardiovascular problems and brain issues that could possibly be fatal. According to estimates, between 30 and 40 % of CO poisoning patients typically pass away before arriving at the emergency room. To improve the results and prognosis of the impacted patients, the care of these patients is therefore essential. (Zein, et al. 2021)

Healthy, nonsmoking subjects have a CO-Hb concentration of less than 2%, while smokers have a concentration of less than 15%. While neurological symptoms including headache, nausea, and dizziness are seen with CO-Hb levels above 10%, levels below 10% do not produce noticeable symptoms. CO-Hb values between 30 and 50 percent are associated with syncope, motor paralysis, disorientation, and increases in heart and respiratory rates. If CO-Hb values are more than 50%, CO poisoning is deemed potentially fatal. Due to the lack of specificity in the symptoms, behavioral illnesses may need differential diagnoses. In situations of CO poisoning, motor function is impaired before awareness is impaired. Because of things like CO-Hb saturation and heme protein affinity, clinical symptoms do not always match CO-Hb values at admission. Patient management entails airway control, prompt evacuation, intravenous access, cardiac monitoring, and the use of an endotracheal tube or facemask to administer 100% oxygen. (Table. 1) (Kinoshita, et al. 2020)

Table 1. Levels of carboxyhemoglobin (CO-Hb) saturation (%) and symptoms.

CO-Hb %	clinical Symptoms
< 1	normal range (due to endogenous production)
< 10	smoker's blood (no symptom)
10–20	headache, fatigue, ear ringing
20–30	headache, weakness, nausea, vomitting
30–40	severe headache, dizziness, nausea, vomitting
40–50	syncope, confusion, increased respiration and heart rate
50k 60	coma, convulsions, depressed respiration
60–70	coma, convulsions, cardiopulmonary depression, often fatal
70 <	respiratory failure, death

6. Diagnosis of CO poisoning

those presenting with flu-like symptoms during cold weather, particularly if other people in the same environment, including pets, show similar symptoms, and patients experiencing unexplained confusion or lactic acidosis. The medical history provided by emergency personnel or ambient CO readings taken by fire departments may suggest CO exposure. However, it's important to note that the ambient CO levels measured by fire departments are often lower than what the patient may have been exposed to, as emergency operators often instruct individuals to ventilate their environment by opening windows.

To confirm CO poisoning, it's essential to measure carboxyhemoglobin (COHb) levels through co-oximetry, which involves drawing a blood sample. For stable patients, venous blood samples are generally accurate. Non-smokers typically have baseline COHb levels up to 3%, whereas smokers can have levels as high as 10% (Hopper, et al. 2021). Levels above these thresholds are indicative of CO poisoning.

COHb Levels and Severity: While COHb levels are critical for confirming exposure, they do not always correlate directly with the severity of poisoning. This discrepancy is particularly notable when significant time has passed since exposure, as CO naturally clears from the body. Elevated COHb levels that correlate with severe symptoms like unconsciousness, confusion, or heart-related issues should be treated as severe cases. In this case, it is the clinical presentation, not the COHb level, that should guide treatment decisions.

Pulse Oximetry Limitations: Standard pulse oximeters, which measure oxygen saturation, cannot effectively detect CO exposure as they do not differentiate between COHb and oxyhemoglobin. There are more advanced pulse oximeters that can measure both COHb and methemoglobin, but these are not yet accurate enough to replace blood co-oximetry. They can be useful, though, for initial screenings. (Tobin, et al. 2017)

Interference with COHb Measurement by Hydroxocobalamin: The administration of hydroxocobalamin (used in cyanide poisoning) can interfere with COHb measurements, causing inaccurate results. Both falsely low and elevated COHb levels have been reported in such cases. In suspected cases of cyanide exposure, healthcare providers should inquire whether hydroxocobalamin was given. Regardless of its use, CO poisoning should be diagnosed based on the patient's exposure history, and treatment should be initiated promptly with oxygen or hyperbaric oxygen therapy if needed. If possible, it is best to collect blood samples before administering hydroxocobalamin for more accurate COHb readings. (Hopper, et al. 2021)

7. Treatment of CO poisoning

After removing the sufferer from the CO source, high-flow oxygen (100 percent normobaric oxygen) is administered using a non-rebreathing mask or, if the victim is unconscious, on a ventilator. Other potential concomitant toxicities, such as cyanide in home fires, are also treated. With a half-life of 250–320 minutes while breathing only air, CO takes too long to disperse; even with high-flow supplemental oxygen, this time only increases to 75–90 minutes. The CO half-life can be further shortened to 30 minutes by adding oxygen in a hyperbaric room at 2.5–3 atmospheres, which also dissolves significant amounts of oxygen into plasma. When COHb is 25–40% or higher, when lactic acid accumulation causes tissues to be severely oxygen-depleted, when there is unconsciousness, or when organs are failing, hyperbaric oxygen is useful as an emergency treatment. A hyperbaric chamber might not be easily accessible, though, and issues with transfer delays, the requirement for precise decompression, and thorough monitoring within the chamber will arise. As was the case with Richard Byrd, recovery after poisoning is frequently drawn out, and the neuropsychiatric effects may be irreversible. Pregnant women and newborns require more rigorous oxygen management because carbon monoxide bound to fetal hemoglobin has a considerably longer half-life than typical. (Rosove. 2024)

8. Measurement of CO

Based on autopsy results and blood CO-Hb saturation, a toxicological assessment of CO poisoning is conducted. In forensic practice, CO-Hb saturation is the fundamental evaluation point. To check for CO, a variety of techniques are used, such as oximetry, gas chromatography, detection tubes, and spectrophotometric techniques. The most popular technique is the spectrophotometric method, which uses variations in the absorption spectra to identify the presence of CO-Hb. In gas chromatography, CO is released from Hb and detected by a variety of detectors, including flame ionization detectors, barrier discharge ionization detectors, and thermal conductivity detectors. Due of its mobility and ease of handling, sensor gas chromatography—gas chromatography using a semiconductor detector—has been used in forensic practice. An aspirating pump, a separation tube, and a detection tube make up another detector tube technique. Ferricyanide-coated silica gel particles are put in the CO separation tube, and potassium palladium sulfite-coated silica gel particles are packed in the detecting tube. After the sample is injected into the CO-separator tube, CO is released from the blood, and the CO-detector tube picks up the CO gas that has been released. Clinical labs and everyday forensic practice both make extensive use of oximeters, which use numerous wavelengths to determine different hemoglobin species, including CO-Hb. The AVOX 4000 equipment has some benefits for on-site testing and makes sample handling simple. Without refrigeration, CO-Hb can remain relatively stable for up to 4 weeks and for up to 24 months at 4°C.

9. Forensic toxicology's function in verifying the cause of death or harm

A key technique for identifying CO poisoning is forensic toxicology, which focuses on the levels of carboxyhemoglobin (COHB). (Al-Asmari et al. 2021). state that a COHb value of more than 50% denotes lethal poisoning. Since it offers a more comprehensive picture of the dosages that entered the body, measuring total blood carbon monoxide (TBCO) is also advised to improve exposure diagnostic accuracy. Advanced methods such as gas chromatography and spectrophotometry have been developed to increase the effectiveness and dependability of toxicological testing. Determining if CO exposure is accidental or suicidal is clearly a function of forensic toxicology. For instance, (Nielsen.2014) argued that even if charcoal burning may be the result of homicide or suicide, forensic examination is still necessary in these circumstances. In terms of the time and place of CO poisoning, delayed neurological sequelae are a clinical reality with forensic implications. When it comes to the etiology and timeframe of CO poisoning, delayed neurological sequelae have forensic importance that may hinder diagnosis (Zhang et al., 2021; Kitamoto et al., 2020).

10. Carbon monoxide poisoning and correlation with carboxyhemoglobins saturation levels

In Jeddah (Fakiha, B. 2025), fire, burning, and fireless sources such as charcoal briquettes and vehicle exhaust are the main causes of CO poisoning. COHb levels exceeding 50% were found in most fire-related accidents, and these levels were present in [60%] of fatalities. Higher COHb concentrations were also a result of burning occurrences; [63%] of cases had levels above [50%]. Although they were less common overall, non-fire-related deaths nevertheless posed serious hazards; in 59 of these cases, the COHb level was more than [50%]. Male cases were greater in winter and summer due to outside exposure and contact with heating systems, while female cases had higher COHb percentages in spring and fall, according to the study, which also revealed a cyclical trend with minor rises in COHb concentration in autumn and summer. (Fakiha, B. 2025). COHb values in Alqurayyate (Attaia, et al. 2020) autopsied cases between 2004 and 2018. Over the course of the study, 461 cases were examined via autopsy. Of them, COHb values greater than 10% were present in 54 cases [11.7%]. Males and those over 30 and older had significantly higher carboxyhemoglobin levels. in Dammam (Aldossary et al. 2015), the severity of COHb levels varies from 15% to 77%, contingent on exposure length, concentration, and personal health. Ten to thirty percent of COHb induce headaches and mild exertional dyspnea, while less than ten percent causes no symptoms at all. COHb levels above 40% are dangerous, and even lower amounts in heart failure patients can be lethal. The median level of COHb in the postmortem blood samples was 56%, and CO intoxication accounted for just over half (58%) of the deaths. (Al-Asmari, et al. 2021)

11. The impact of carbon monoxide (CO) poisoning on public health in Saudi Arabia

The impact of carbon monoxide (CO) poisoning on public health in Saudi Arabia is significant, particularly due to seasonal fluctuations and the widespread use of heating systems in closed environments during the colder months. Here are several key points illustrating the public health effects:

1. Seasonal Peaks and Risk Factors

In Saudi Arabia, CO poisoning cases tend to rise during the winter months when people use alternative heating sources like charcoal, kerosene, and gas heaters, often in poorly ventilated indoor spaces. The use of these heating methods, combined with insufficient ventilation, creates a dangerous environment for CO buildup. The incidence is also higher in remote areas where heating practices might be less regulated. Additionally, during this period, people may unknowingly be exposed to CO while in their homes, leading to significant health risks.

2. Vulnerable Populations

Certain groups in Saudi Arabia are more vulnerable to CO poisoning. Children, the elderly, pregnant women, and people with pre-existing health conditions, such as heart disease or respiratory disorders, are at higher risk. For instance, children may be more susceptible to the neurotoxic effects of CO, and pregnant women may experience complications that affect both maternal and fetal health. Moreover, people working in confined spaces or those who engage in indoor cooking with traditional methods are at higher risk of exposure.

3. Public Health Burden

While CO poisoning is often preventable, it remains a leading cause of unintentional poisoning in Saudi Arabia, resulting in hospital admissions, long-term neurological effects, and, in severe cases, death. The symptoms of CO poisoning, such as headache, dizziness, confusion, and nausea, are often mistaken for flu-like illnesses, leading to delayed diagnosis and treatment. This delay increases the chances of severe complications, including permanent brain damage, cardiac issues, or death. Hospitals and emergency departments often deal with the consequences of this delayed recognition, placing additional strain on healthcare systems.

4. Economic Impact

The economic burden of CO poisoning in Saudi Arabia includes medical costs for treatment, hospitalization, and rehabilitation, as well as the lost productivity of individuals who are affected by severe poisoning or who require long-term care due to permanent damage. The financial burden on the public health system could be substantial, particularly if the number of cases increases without significant public health interventions.

5. Preventive Measures and Public Awareness

To mitigate CO poisoning, public health initiatives are crucial. There is a growing need for increased public awareness about the risks of CO exposure, particularly during winter months. Promoting the use of CO detectors in homes, improving the ventilation of indoor spaces, and educating people about the dangers of using fuel-based heaters in closed areas can reduce the number of poisoning cases. Additionally, training healthcare professionals to recognize CO poisoning symptoms early can help in timely diagnosis and treatment.

6. Regulation and Safety Standards

The Saudi government has started taking steps to address CO poisoning by regulating heating appliances and promoting safer alternatives for indoor heating. However, much more can be done, particularly in rural and underdeveloped areas where awareness of CO poisoning is low, and the use of hazardous heating practices persists.

Recommendations

- The public awareness regarding COP and its sources is still deficient and more awareness programs should be planned and implemented, mainly before and during the cold months.
- • In autopsy instances, COHb values should be regularly evaluated, particularly if the history is suspicious.
- Mostly postmortem signs as cherry red colour and retinal hemorrhages are not found in all cases with high COHb levels. This should not mislead the forensic examiner, and he should ask for COHb levels in COP suspicious cases
- There is a continued call for better CO poisoning prevention, such as Improvement of forensic procedures in CO deaths, reporting of CO cases, and public health measures, especially in the cold season, that can help to reduce needless loss of life and incidences of the condition.

Conclusion

Death is a possible outcome of acute carbon monoxide intoxication. According to the research, CO poisoning is most frequently observed in unintentional cases, occasionally in suicide cases, and infrequently in homicidal cases. In Saudi Arabia, carbon monoxide poisoning is still frequent and primarily caused by burning and fire-related factors, as well as non-fire ones such vehicle exhaust and cooking with charcoal briquettes. The study's authors noted that exposure to them varied seasonally, with greater levels of COHb primarily seen in the fall and summer, perhaps because of climatic and cultural factors. Among the cases, fire-related conditions had the greatest amounts of COHb, suggesting that exposure during a fire can be fatal.

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