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The Toxic Face of Fire - Chemical Poisonings in Fire Conditions and its Treatment

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ABSTRACT

Fire damage is not the only cause of death in fires. Combustion releases many toxins, including carbon monoxide (CO), hydrogen cyanide (HCN), and other chemicals discussed in this article. Symptoms of exposure are often nonspecific, including headaches, dizziness, nausea, shortness of breath, and, in severe cases, loss of consciousness. Prompt intervention is essential, and oxygen therapy - especially hyperbaric oxygen therapy in severe cases - is the most effective treatment. Modern building materials, such as polyurethanes and mineral fibers, further complicate fire toxicity by releasing additional harmful substances during combustion. Fire hazards require increased public awareness. Appropriate response and treatment can reduce the mortality rate of people poisoned in fires.

Key words: carbon monoxide, poisoning, toxic substances, burns

1. INTRODUCTION

In 2024, the fire service recorded 102,882 fires in Poland. As a result of these incidents, 295 people lost their lives, and 2,052 sustained injuries (Statistics of the National Headquarters of the State Fire Service of Poland, 2024). During a fire, the flames consume much oxygen, significantly reducing oxygen concentration in the room. Respiration under such conditions can affect the functioning of body tissues, especially the heart and brain. When a fire occurs in an oxygen-poor environment, harmful byproducts of incomplete combustion, for instance, cyanides or carbon monoxide, are produced. In addition, introducing modern building technologies and new chemical compounds often complicate, if not prevent, fire smoke hazards and poisoning assessment (Duda, 2006).

Objective

This paper analyses the problem of poisoning resulting from fires and discusses effective treatment methods. We have paid particular attention to toxic substances produced by the combustion of various materials, such as carbon monoxide, hydrogen cyanide, and other chemical compounds. The work aims to

raise awareness of the dangers of inhaling toxic gases and identify best practices in medical management that can help improve treatment and reduce mortality among fire victims.

2. METHODS

This article presents a scientific review of the available literature. Available publications were analysed, focusing on clinical trials, meta-analyses, and systematic reviews containing information on toxic substances resulting from fire. The analysis covered the most common toxins responsible for poisoning and less obvious chemical compounds released during fires, including mineral fibers, polyurethane, and cellulose. The literature review included specialised textbooks and publications in the PubMed database, using keywords such as carbon monoxide, poisoning, toxic substances, and burns. The main articles reviewed in this work were published between April 2000 and September 2024, while the oldest article dates back to 1967 and concerns nitrogen compound poisoning.

3. RESULTS AND DISCUSSION

Carbon Monoxide (CO)

Carbon monoxide is a gas devoid of colour, odour and taste. Fires, malfunctioning fireplaces, vehicle exhaust fumes, gas water heaters, coal, gas, oil furnaces, and gas stoves can produce it through incomplete combustion. Fires, malfunctioning fireplaces, vehicle exhaust fumes, gas water heaters, coal, gas, oil furnaces, and gas stoves can produce it through incomplete combustion. It forms during the incomplete combustion of various fuels, including wood, gasoline, coal, natural gas, and kerosene. CO readily binds to hemoglobin (Hb) in the blood, forming carboxyhemoglobin (COHb) (Raub et al., 2000; Penney et al., 2010). Researchers estimate that carbon monoxide poisoning significantly affects 50–75% of fire-related injuries.

Epidemiology of Carbon Monoxide (CO) Poisoning

Data from the Global Health Data Exchange (GHDx) indicate the global incidence and mortality rates of carbon monoxide poisoning in 2017, estimated at 137 cases and 4.6 deaths per million, respectively. Findings indicate no significant correlation between the passage of time and the frequency of CO poisoning. Despite this, carbon monoxide (CO) poisoning mortality has shown a significant time-dependent decline, decreasing by 36% between 1992 and 2017 (Mattiuzzi & Lippi, 2020). In 2024, Polish firefighters participated in 4,329 carbon monoxide-related incidents. In 2024, firefighters in Poland intervened 4329 times in connection with carbon monoxide exposure, of which carbon monoxide fatally poisoned 52 people and firefighters rescued 1334 (Statistics of the National Headquarters of the State Fire Service of Poland, 2024).

Pathophysiology of Carbon Monoxide (CO) Poisoning

The severity of carbon monoxide poisoning stems from various environmental and individual factors. These include the concentration of CO in the air, ventilation of the environment, duration of exposure to the toxin, respiratory capacity, and the body's ability to regulate acid-base balance. CO molecules cross the alveolar-capillary barrier upon inhalation and diffuse into the capillary blood (Lippi et al., 2012). Carbon monoxide has an affinity for hemoglobin 230–300 times greater than oxygen, preferentially binding to the iron ion in the heme group. This binding affects a conformational change in hemoglobin, shifting the oxyhemoglobin dissociation curve and reducing oxygen transport capacity and its release to peripheral tissues (Rose et al., 2017).

At the cellular level, CO triggers multiple pathological processes, including intensified lymphocyte proliferation, neutrophil activation, mitochondrial dysfunction, and lipid oxidation. Additionally, the generation of reactive oxygen species, inflammatory responses, increased oxidative stress, and the initiation of apoptosis mimic the damage mechanisms observed in ischemia-reperfusion injury, making them key contributors to cellular destruction (Rose et al., 2017; Culnan et al., 2018).

Symptoms of Carbon Monoxide (CO) Poisoning

Clinicians base the diagnosis of carbon monoxide poisoning on three key elements: symptoms typical of CO exposure, confirmed or probable recent contact with the gas, and an elevated carboxyhemoglobin (COHb) level in the blood. These criteria require careful evaluation to prevent the occurrence of chronic carbon monoxide poisoning at lower concentrations (Mattiuzzi & Lippi, 2020; Hampson et al., 2012).

Common symptoms of CO poisoning include headaches, dizziness, nausea or vomiting, concentration difficulties, fatigue, chest pain, dyspnea, and loss of consciousness. Poisoned people are treated in hospital emergency departments in critical condition, making

it impossible to collect a history. Firefighters can measure ambient CO concentrations, providing relevant exposure evidence. In addition, detecting elevated levels of carboxyhemoglobin (COHb) in the blood is an important, if not crucial, element in confirming poisoning (Rose et al., 2017; Penney, 2010).

Treatment of Carbon Monoxide (CO) Poisoning

The primary treatment for CO poisoning involves administering high-concentration oxygen (90–100%) via a mask or an endotracheal tube. When breathing ambient air, the half-life of carboxyhemoglobin (COHb) ranges from 120 to 200 minutes, whereas inhaling pure oxygen reduces it to approximately 30 minutes (Duda, 2006; Rose et al., 2017). However, clinical studies have not confirmed a significantly higher efficacy of normobaric 100% oxygen therapy compared to ambient air. In cases where hyperbaric treatment is unavailable, patients should receive pure oxygen in the emergency department until COHb level drops to physiological values (≤3%) and symptoms resolve - typically within six hours. Guidelines recommend considering hyperbaric oxygen therapy for severe or acute CO poisoning. Clinicians should continue normobaric 100% oxygen therapy until they initiate hyperbaric treatment (Hampson et al., 2012).

Studies are also exploring pharmacological approaches. Studies have suggested that hydroxocobalamin and ascorbic acid may accelerate the conversion of CO to carbon dioxide (CO_2). A study performed at Virginia Commonwealth University showed that this method reduces the half-life of Hb-CO and limits CO-induced cerebral hypoxia in rodents. Unfortunately, there was no improvement in cognitive function, which is necessary for patient prognosis (Roderique et al., 2015).

Another pharmacological method is a porphyrin complex surrounded by cyclodextrin, capable of binding carbon monoxide (CO) with a 100 times greater affinity than hemoglobin. After administration to rats, it captured endogenous CO, which the body eliminated with urine. Cyclodextrin is a toxic compound. Its nephrotoxicity may significantly limit the use of this method (Kitagishi et al., 2010). A new and promising form of therapy currently is globulin proteins. Research indicates that these compounds possess a high affinity for CO, effectively acting as carbon monoxide scavengers. The elimination of CO from erythrocyte hemoglobin and tissues is accelerated, and the action is not limited to Hb-CO reduction only. Their mechanism extends beyond COHb reduction; by directly binding CO to hemoproteins such as cytochrome c oxidase (COX), they may also modulate other aspects of CO poisoning, including inflammatory responses, mitochondrial dysfunction, and ischemia-reperfusion injury (Rose et al., 2017).

Hydrogen Cyanide (HCN)

Fire smoke contains many toxic substances; hydrogen cyanide (HCN) is crucial alongside carbon monoxide. HCN is generated from the combustion of natural materials like wool and silk and the thermal decomposition of synthetics such as polyurethane, polyacrylonitrile, and polyamide (Tabian et al., 2021). Hydrogen cyanide is a highly flammable compound. Furthermore, when exposed to fire, it releases suffocating and potentially toxic vapors. Its interaction with air can also form explosive mixtures, presenting a significant hazard in fire environments (Økland et al., 2020).

Pathophysiology of Hydrogen Cyanide (HCN) Poisoning

Hydrogen cyanide and other cyanogenic compounds impair cellular oxygen utilisation by binding to complex IV of the mitochondrial electron transport chain. This inhibition causes cells to shift towards anaerobic metabolism, which impacts the surplus production of lactic acid. Moreover, this impairment occurs even though lungs-tissue oxygen transport remains unaffected. Clinically, cyanide poisoning is characterised by persistently elevated lactic acid levels and delayed recovery, even providing adequate oxygen therapy (Økland et al., 2020).

Symptoms of Hydrogen Cyanide (HCN) Poisoning

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Treatment of Hydrogen Cyanide (HCN) Poisoning

Given the absence of readily available on-site cyanide detection tests during fire incidents, emergency responders and medical personnel are compelled to base their decision to administer an antidote primarily on patient history, clinical presentation (including observed symptoms), and other indicative signs of poisoning (Sanders et al., 2022). FDA-approved pharmacological treatments for cyanide poisoning fall into three main categories: methemoglobin inducers and nitric oxide donors (sodium nitrite, amyl nitrite, and dimethylaminophenol), sulfur donors (sodium thiosulfate and glutathione), cyanide-binding agents (hydroxocobalamin and dicobalt edetate) (Hendry-Hofer et al., 2019).

One therapeutic strategy involves inducing methemoglobinemia, which unblocks cytochrome oxidase by diverting cyanide to bind with hemoglobin instead. However, this approach is highly controversial, primarily because methemoglobinemia may worsen the toxic effects of elevated carboxyhemoglobin (COHb) levels. Alternatively, cobalt-based agents such as dicobalt edetate directly react with cyanide, restoring cytochrome oxidase activity, though they may negatively impact cardiac function. Hydroxocobalamin, on the other hand, converts into cyanocobalamin (vitamin B12), effectively neutralizing cyanide toxicity (Duda, 2006).

Other Toxic Compounds in Fire Smoke

Besides carbon monoxide and hydrogen cyanide, fire smoke contains a complex mixture of poisonous substances. This complexity amplifies advancements in construction technology and the growing demand for modern building materials, which release various toxic substances when burned. These include aromatic hydrocarbons, benzene, acrolein, sulfur oxides, benzaldehyde, and other irritants (Duda, 2006; Navarro et al., 2021). These chemicals can cause severe respiratory irritation, systemic toxicity, and long-term health effects, including carcinogenic and mutagenic risks. The composition of toxic emissions depends on the materials involved in combustion, making each fire potentially unique in its hazardous exposure profile.

Mineral Fibers

These include non-glass materials. These include semi-crystalline wool and synthetic glass fibers. The category of glass fibers includes: glass wool, rock (stone) wool, slag wool, alkaline earth silicate wool, and aluminosilicate wool. These materials, commonly employed as construction insulators, are typically composed of silicates and various mineral oxides, although their exact composition varies with application (Wienen et al., 2023). To ensure insulation, they melt at high temperatures, typically 1000 to 1500°C. However, their combustion releases various toxic substances, including hydrogen cyanide, carbon monoxide, nitrogen oxides (NO_2), sulfur oxides (SO_2), acrolein, benzene, various hydrocarbons, and harmful particulate matter. Collectively, these compounds present serious health hazards (Duda, 2006).

Polyurethane (PU)

Polyurethane is a polymer that can be modified depending on various requirements. It has high tensile strength, abrasion resistance, and durability in repeated stresses. At the same time, it maintains flexibility at very low temperatures (Wienen et al., 2023). At 250°C, it decomposes by releasing isocyanate monomers, amines, carbon monoxide, and hydrogen chloride. Fine oil particles heavily saturate the smoke generated from combustion.

Cellulose Fibers

Cellulose is a principal structural component of plant cell walls, making it an ideal polymer for eco-friendly applications requiring superior resilience. Due to its macro- and nanostructural organization, cellulose is well-suited for producing lightweight, resilient structures and advanced functional materials used in everyday life. During combustion, cellulose releases furan, glucose, acrolein, and other irritants, which may pose health risks (Duda, 2006; Antlauf et al., 2021).

Smoke Inhalation Poisoning Treatment

The most commonly employed approach is symptomatic treatment. Clinicians should provide oxygen administration and respiratory support in acute cough, bronchospasm, hypoxia, and respiratory failure. Healthcare providers use glucocorticoids to prevent toxic pulmonary edema, as they exhibit rapid, potent, but nonspecific anti-inflammatory effects. Its action involves blocking inflammatory mediator release by activating neutrophils, reducing capillary permeability, and promoting alveolar macrophage differentiation (Duda, 2006; Lu et al., 2021). In case of organophosphate poisoning, guidelines recommend the use of atropine. As a selective antagonist of

postganglionic muscarinic receptors, atropine counteracts salivation, lacrimation, nausea, vomiting, and gastrointestinal disturbances. Mitigating peripheral symptoms and preventing acute respiratory failure is crucial (Aroniadou-Anderjaska et al., 2020).

Poisoning with nitrates and other nitrogen compounds can lead to methemoglobinemia. Administering methylene blue to patients can reverse this condition. At low concentrations, methylene blue facilitates the rapid conversion of methemoglobin back to hemoglobin, acting as a non-enzymatic catalyst for the reduced form of nicotinamide adenine dinucleotide phosphate (NADPH) and playing a key role in regulating methemoglobin reductase activity (Duda, 2006; Prys-Roberts, 1967; Kazmi et al., 2024). The key findings of this article are presented in Table 1.

Table 1 Key findings in poisoning from toxic compounds present in fire smoke

Aspect	**Key findings**
Main Toxic Agents	Carbon Monoxide (CO) Hydrogen Cyanide (HCN) Other combustion-derived toxicants (benzene, acrolein, sulfur oxides, etc.)
Sources of Toxicants	Incomplete combustion of fuels and synthetic materials Modern building materials: polyurethane, mineral fibers, cellulose
Symptoms	CO: Headache, dizziness, nausea, dyspnea, unconsciousness HCN: Lactic acidosis, altered mental status, delayed recovery, increased methemoglobin level
Diagnostic Criteria	Clinical symptoms + elevated COHb (for CO) Clinical suspicion + high lactate (for HCN)
Treatment	CO: High-flow oxygen, hyperbaric oxygen therapy HCN: Antidotes (hydroxocobalamin, sodium thiosulfate, etc.)
Emerging Therapies	Pharmacologic CO scavengers (globulin-based, porphyrin-cyclodextrin complexes) Experimental antioxidant therapies

4. SUMMARY

Fire smoke poisoning is a serious problem that may lead to death or permanent disability. Fires severely reduce air quality, affecting body functions, particularly the heart and brain. Fire smoke contains several toxic substances, the most widely recognised of which are carbon monoxide (CO) and hydrogen cyanide (HCN). Carbon monoxide poisoning usually causes symptoms such as headache, nausea, shortness of breath, and even loss of consciousness. HCN, on the other hand, causes lactic acidosis and severe metabolic disorders. Modern construction materials further increase the toxicity of fire-related smoke by releasing other pollutants, such as aromatic hydrocarbons and nitrogenous compounds. Treatment of fire-related poisoning is symptomatic primarily, including oxygen therapy, corticosteroids, and specific antidotes according to the substance concerned.

5. CONCLUSIONS

The article concludes that exposure to fire smoke can lead to long-term health effects, especially respiratory diseases. Rapid and appropriate medical response is crucial to manage the affected individuals and minimise the damage. Public education and awareness of fire prevention can significantly reduce the occurrence of poisoning and its complications. Attention should also be paid to using safer materials in construction to reduce the harmful effects of smoke. It is essential that appropriate safety procedures are followed and that medical staff are trained to deal with rapid and effective responses in such emergencies.

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