



Carbon Monoxide as a Toxic Agent in Charcoal Briquette-Related Deaths: A Systematic Literature Review

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ABSTRACT

Carbon monoxide (CO) is a colorless, odorless, and tasteless toxic gas that poses a substantial health hazard, particularly in enclosed environments. Combustion of charcoal briquettes is a frequently overlooked source of CO exposure, linked to both accidental and intentional fatalities. This review synthesizes current evidence on the toxicological role of CO in charcoal briquette-related deaths and identifies preventive strategies. A systematic literature review was conducted using PubMed, ScienceDirect, and Google Scholar to identify peer-reviewed articles published between January 2020 and April 2025. Studies in English or Indonesian reporting fatal CO poisoning cases associated with charcoal briquette combustion were included, provided they contained data on carboxyhemoglobin (COHb) levels, clinical presentation, and environmental risk factors. Ten studies met the inclusion criteria. Most fatal cases occurred in enclosed or poorly ventilated spaces, with COHb levels exceeding 30%, a threshold strongly associated with mortality. Common clinical manifestations included headache, confusion, loss of consciousness, seizures, and respiratory distress. Autopsy findings frequently revealed cherry-red discoloration of the skin and mucous membranes. Both accidental and intentional exposures were reported, with men disproportionately affected. Charcoal briquette combustion in confined environments remains a significant and preventable cause of CO-related mortality. Public awareness campaigns, mandatory installation of CO detectors, and ventilation standards are critical to reducing the incidence of such deaths.



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ABSTRAK

Karbon monoksida (CO) adalah gas beracun yang tidak berwarna, tidak berbau, dan tidak berasa yang menimbulkan bahaya kesehatan serius, terutama di lingkungan tertutup. Pembakaran briket arang merupakan sumber paparan CO yang sering diabaikan, dan telah dikaitkan dengan kasus kematian baik yang bersifat tidak disengaja maupun disengaja. Tinjauan ini merangkum bukti terkini mengenai peran toksikologis CO dalam kematian terkait penggunaan briket arang serta mengidentifikasi strategi pencegahannya. Telaah pustaka sistematis dilakukan melalui basis data PubMed, ScienceDirect, dan Google Scholar untuk mengidentifikasi artikel ilmiah yang diterbitkan antara Januari 2020 hingga April 2025. Artikel yang disertakan adalah studi berbahasa Inggris atau Indonesia yang melaporkan kasus kematian akibat keracunan CO terkait pembakaran briket arang, dengan data kadar karboksihemoglobin (COHb), manifestasi klinis, dan faktor risiko lingkungan. Sebanyak sepuluh studi memenuhi kriteria inklusi. Sebagian besar kasus fatal terjadi di ruang tertutup atau dengan ventilasi buruk, dengan kadar COHb melebihi 30% yang secara kuat berkaitan dengan risiko kematian. Manifestasi klinis yang umum meliputi sakit kepala, kebingungan, kehilangan kesadaran, kejang, dan gangguan pernapasan. Temuan otopsi sering menunjukkan perubahan warna merah ceri pada kulit dan selaput lendir. Paparan terjadi baik secara tidak disengaja maupun disengaja, dengan proporsi kasus pada laki-laki lebih tinggi. Pembakaran briket arang di ruang tertutup merupakan penyebab kematian akibat CO yang signifikan namun dapat dicegah. Edukasi masyarakat, pemasangan detektor CO secara wajib, dan penerapan standar ventilasi merupakan langkah penting untuk menurunkan angka kejadian kematian tersebut.

Kata Kunci: Karbon monoksida; Briket arang; Karboksihemoglobin; Keracunan CO; Mortalitas

1. Introduction

Charcoal briquettes are solid fuels produced by further processing charcoal into compact, uniform shapes with enhanced calorific value, density, and ease of storage and transport [1],[2]. As an alternative energy source, they have been widely promoted to address the growing scarcity of fossil fuels. Their high carbon content, energy density, and portability make them attractive for domestic heating and cooking. However, despite these advantages, charcoal briquette combustion in poorly ventilated spaces poses serious health risks due to the release of carbon monoxide (CO) [3].

Carbon monoxide is a non-irritating, colorless, odorless, and tasteless gas generated by the incomplete combustion of carbon-containing materials [4],[5]. It is highly toxic and can be lethal when inhaled in high concentrations. Once absorbed, CO binds with hemoglobin to form carboxyhemoglobin (COHb), which has approximately 240 times greater affinity for hemoglobin than oxygen [6]. This binding significantly reduces oxygen transport to tissues, resulting in hypoxia, oxidative stress, and potentially irreversible damage to vital organs, particularly the brain and heart [5],[9]. Fatal CO poisoning cases have frequently reported COHb levels exceeding 30%, often accompanied by neurological injury, including necrosis in the globus pallidus and apoptosis in brain tissue [7].

Although CO poisoning is a recognized public health threat and a documented method of suicide—particularly among educated individuals—many cases remain underreported [8]. Globally, it is estimated that 137 per one million people experience CO poisoning each year, with approximately five fatalities. Over the past 25 years, the incidence has remained relatively stable, with men exhibiting twice the mortality rate of women. In 2022 alone, the Centers for Disease Control and Prevention (CDC) recorded 1,244 deaths from CO poisoning, underscoring its persistent relevance as a public health concern [6].

While numerous studies have examined CO toxicity and its health impacts, literature focusing specifically on fatal cases related to charcoal briquette use remains limited. Previous reports have largely concentrated on occupational exposures, environmental monitoring, or generalized CO epidemiology, with insufficient emphasis on the unique risk profile posed by briquette combustion in domestic and enclosed settings. To address this gap, the present review systematically analyzes published cases of CO-related mortality linked to charcoal briquettes, describes the associated toxicological mechanisms, and highlights evidence-based prevention strategies.

2. Methods

This review employed a systematic literature review approach to identify and analyze fatal cases of carbon monoxide (CO) poisoning associated with charcoal briquette use. The literature search was conducted using three electronic databases – PubMed, ScienceDirect, and Google Scholar – covering the period from January 2020 to April 2025. The search strategy combined relevant keywords using Boolean operators, including: “carbon monoxide”, “charcoal briquettes”, “COHb”, “carbon monoxide poisoning”, “death”, and “prevention”.

The inclusion criteria for this review were: (1) articles published between January 2020 and April 2025; (2) written in English or Indonesian; (3) peer-reviewed journal publications; (4) studies reporting fatal CO poisoning cases directly linked to charcoal briquette combustion; and (5) availability of data on carboxyhemoglobin (COHb) levels, clinical manifestations, or environmental risk factors. The exclusion criteria were: (1) non-peer-reviewed articles, conference abstracts, or non-scientific reports; (2) studies focusing solely on environmental CO measurement without human health data; (3) articles without full-text availability; and (4) duplicate publications.

The literature selection process was conducted in three stages. During the identification stage, the initial search retrieved 78 articles. In the screening stage, removal of 15 duplicates resulted in 63 unique articles for title and abstract review; 41 were excluded for irrelevance. In the eligibility and inclusion stage, 22 full-text articles were assessed in detail, of which 12 were excluded due to absence of briquette-specific data or lack of clinical outcome information. Ultimately, 10 articles met all inclusion criteria and were included in the final synthesis.

Data extracted from each study included author and year of publication, COHb concentration, clinical signs, pathological findings, environmental context, and recommended prevention measures. The extracted data were synthesized thematically to identify patterns in toxicological mechanisms, mortality risk thresholds, and preventive strategies. The overall selection process is summarized in the PRISMA flow diagram (**Figure 1**), which enhances transparency and reproducibility of the methodology.

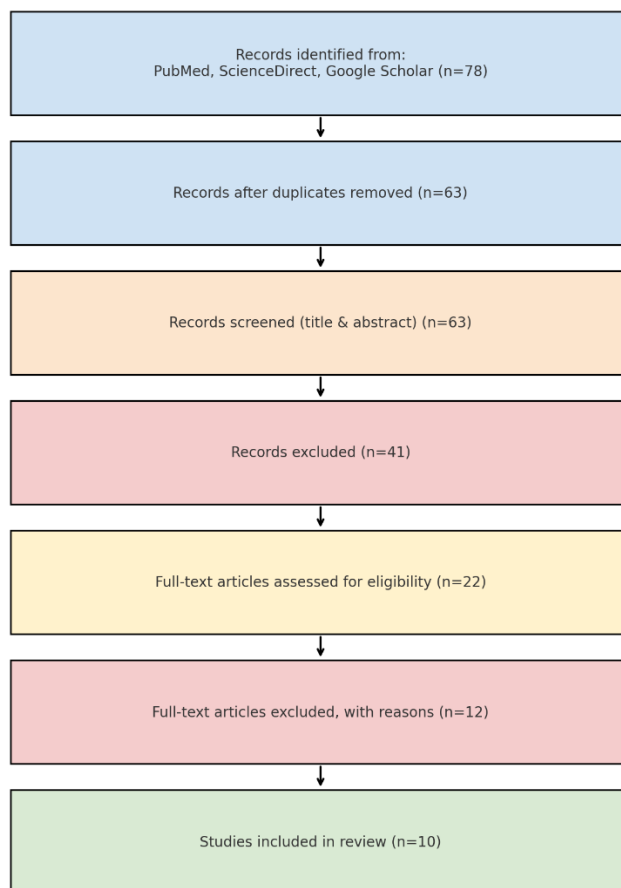


Figure 1. Figure 1. PRISMA flow diagram illustrating the literature selection process

3. Results and Discussion

Overview of Included Studies

This review included 10 peer-reviewed articles published between 2020 and 2025, encompassing both national and international sources [8],[10]-[16], [21],[22]. The studies originated from diverse geographical settings, including Asia, Europe, and the Middle East, and primarily consisted of detailed case reports describing fatal carbon monoxide (CO) poisoning events linked to charcoal briquette combustion. The cases varied in intent, with several being suicide attempts [8],[12],[15] and others representing accidental exposures in domestic settings [16],[20],[21].

Across the included literature, carboxyhemoglobin (COHb) levels in fatal cases were frequently reported to exceed 30%, aligning with thresholds associated with high mortality risk [7],[10],[11],[15]. Clinical presentations ranged from mild neurological symptoms such as headache and confusion to severe manifestations, including seizures, coma, and respiratory distress. In some cases, particularly among elderly or comorbid patients, even moderate COHb levels (~5%) were associated with significant clinical outcomes [9]. Pathological findings reported in autopsies often included cherry-red discoloration of the skin and mucous membranes, pulmonary congestion, and hypoxic injury to the globus pallidus [17],[18],[19].

Environmental risk factors were consistently linked to poorly ventilated or sealed spaces, where prolonged combustion of charcoal briquettes allowed CO to accumulate to lethal concentrations [13],[14]. Several studies also underscored the

importance of functional CO detectors in preventing fatalities [9],[21]. **Table I** summarizes the key characteristics of the included studies, including COHb levels, clinical and pathological findings, environmental conditions, and prevention implications.

Table 1. Results of Literature Study Analysis

Author (Year)	COHb Levels	Clinical Signs	Pathological Findings	Environmental Risk Factors	Prevention Implications
Aoshima et al. (2021) [11]	3.0%	Right hemiplegia, hypertension, headache	Hypodensity in left globus pallidus	Charcoal briquette use in enclosed space	Highlight importance of early neurological evaluation
Baksevic et al. (2024) [10]	28.8%	Altered consciousness, GI symptoms, Babinski sign	Bilateral white matter hypodensity, toxic leukoencephalopathy	Indoor combustion of unknown source	Early imaging critical for CNS involvement
Shah et al. (2025) [12]	20.7%	Respiratory distress, unconsciousness	Acute infarction in both globus pallidus	Generator and car engine running in garage	Ventilation monitoring and mental health surveillance
Desai & Shukla (2024) [9]	5.0%	Mild symptoms, exposure history	Not specified	CO detector disconnected, poor heater maintenance	Functional CO detectors are crucial
Kuwahara et al. (2023) [21]	4.0%	Dyspnea, high respiratory rate, O ₂ sat 90%	Suspected hypersensitivity pneumonitis	Charcoal burning in suicide attempt	Address mental health, ensure access to CO sensors
Singh et al. (2023) [19]	Not measured	Found dead, cherry red skin	Soot in trachea, lungs with pink fluid	Closed room, burned mattress	Fire safety and indoor air regulation
Salil et al. (2024) [8]	Not measured	Loss of consciousness, found with family	Cherry red skin and petechiae	Indoor use of charcoal with white powder	Awareness of CO use in group suicides needed
Jian-qiang (2023) [20]	17.5%	Found dead in outdoor tent	Rigor mortis, conjunctival hyperemia	Incomplete alarm function, sealed tent	Emphasizes role of functional CO alarms even in outdoor shelters
Hosahally et al. (2023) [16]	76.55%	Found dead in bathroom	Pink lungs, generalized rigor mortis	LPG gas geyser in poor ventilation	Mandatory ventilation systems in

					water heater installations
Ferorelli et al. (2021) [15]	91%	Found dead in sealed room	No external trauma	Airtight room, open brazier	Indicates lethality of high-concentration CO exposure in closed systems

Carboxyhemoglobin (COHb) Levels and Mortality Thresholds

Carboxyhemoglobin (COHb) concentration is a critical biomarker in assessing the severity of carbon monoxide (CO) poisoning and predicting mortality risk. The reviewed studies consistently demonstrated that fatal outcomes were strongly associated with COHb levels exceeding 30% [7],[10],[11],[15]. This threshold aligns with established toxicological evidence indicating that COHb concentrations above this level significantly impair oxygen delivery to tissues, leading to hypoxia, multi-organ failure, and death [5],[9].

Several cases exemplify this relationship. For instance, Ferorelli et al. [15] reported a COHb level of 91% in a suicide case involving charcoal combustion in a sealed room, a concentration almost universally fatal. Similarly, Hosahally et al. [16] described an accidental exposure in a poorly ventilated bathroom where the victim’s COHb reached 76.55%, resulting in rapid death. In Baksevice et al. [10], a patient with 28.8% COHb developed acute toxic leukoencephalopathy and central nervous system (CNS) involvement, highlighting the potential for severe neurological damage even at borderline-lethal levels.

Notably, the data also revealed that moderate COHb levels can still be clinically significant in susceptible populations. Desai and Shukla [9] reported symptomatic CO poisoning at 5.0% COHb in an elderly patient with comorbidities, while Kuwahara et al. [21] documented 4.0% COHb in a suicide attempt, accompanied by marked respiratory distress. These findings emphasize that absolute COHb concentration should not be the sole determinant of clinical severity; patient age, comorbid conditions, and duration of exposure are important co-factors influencing outcomes [6],[9].

From a forensic perspective, COHb measurement during autopsy remains an essential diagnostic tool. Cases documented by Singh et al. [19] and Jian-qiang [20] demonstrated that, in the absence of ante-mortem measurements, post-mortem COHb levels and associated histopathological findings—such as cherry-red discoloration—provide strong evidence of CO-related mortality. This is particularly relevant in settings where CO exposure is suspected but not initially recognized, reinforcing the need for routine toxicological analysis in unexplained deaths occurring in enclosed or semi-enclosed environments [17],[18],[19].

Overall, the evidence affirms that COHb levels above 30% represent a critical mortality threshold in CO poisoning. However, lethal outcomes may still occur at lower levels depending on individual vulnerability and exposure context, underscoring the importance of early recognition and intervention regardless of the measured COHb value.

Clinical Manifestations of CO Poisoning

The clinical presentation of carbon monoxide (CO) poisoning varies widely, ranging from mild, non-specific symptoms to severe, life-threatening complications.

Across the reviewed literature, early symptoms often included headache, dizziness, nausea, vomiting, and generalized weakness symptoms that are frequently mistaken for viral or gastrointestinal illnesses [5],[9],[10]. Such non-specificity underscores the risk of delayed diagnosis, particularly in primary care or emergency settings without access to CO monitoring equipment.

Neurological manifestations were among the most consistently reported and clinically significant outcomes. Several studies documented central nervous system (CNS) involvement, including confusion, disorientation, seizures, and loss of consciousness [8],[10],[11],[12]. Imaging findings in severe cases revealed hypodensity or infarction in the globus pallidus, a brain region particularly vulnerable to hypoxic injury [10],[11],[12]. For example, Aoshima et al. [11] described a patient presenting with right hemiplegia and radiological evidence of left globus pallidus hypodensity, while Shah et al. [12] reported bilateral acute infarctions in the same region in a suicide attempt case with high CO exposure.

Respiratory manifestations were also prominent in certain cases, particularly those with rapid and intense exposure. Kuwahara et al. [21] reported a patient with dyspnea, tachypnea (respiratory rate of 32 breaths/min), and oxygen saturation of 90%, while Hosahally et al. [16] described post-mortem findings of pulmonary congestion and pink frothy fluid in the lungs – consistent with acute respiratory compromise due to CO-induced hypoxia.

Dermatological and forensic indicators, notably cherry-red discoloration of the skin and mucous membranes, were frequently observed in fatal cases [17]–[20]. This classical sign results from the bright red color of COHb and was evident in both accidental and intentional exposures, as reported by Singh et al. [19], Salil et al. [8], and Jian-qiang [20]. Although not pathognomonic, its presence in combination with environmental context strongly supports a diagnosis of CO poisoning.

Importantly, several cases highlighted the influence of patient-specific factors on symptom severity. Older adults, individuals with pre-existing cardiovascular or respiratory disease, and those with prolonged exposure appeared more susceptible to severe manifestations at relatively lower COHb levels [9],[21]. This suggests that vulnerability is influenced not only by the concentration of CO inhaled but also by the individual's baseline health status and the duration of exposure [6],[9].

Collectively, these findings demonstrate that CO poisoning should be considered in any patient presenting with unexplained neurological or respiratory symptoms, especially when there is a history of potential exposure to combustion sources in enclosed or poorly ventilated environments. Early recognition and intervention remain critical to preventing irreversible neurological damage and mortality.

Pathological Findings in Fatal Cases

Post-mortem examinations in fatal carbon monoxide (CO) poisoning cases consistently reveal a set of characteristic pathological findings that provide strong diagnostic evidence. One of the most notable and frequently reported signs is the cherry-red discoloration of the skin, mucous membranes, and internal organs, which results from the bright red hue of carboxyhemoglobin (COHb) [17]–[20]. This discoloration, while not pathognomonic, is considered a classical forensic indicator of CO poisoning and was observed in both accidental and intentional exposures. Singh et al. [19] documented cherry-red post-mortem staining on the victim's body and mucous membranes, while Salil et al. [8] described intense discoloration accompanied by petechial hemorrhages on the conjunctiva, face, and neck.

Neuropathological changes are also well documented. The globus pallidus has emerged as a primary target of CO-induced hypoxic injury due to its high metabolic demand and vulnerability to reduced oxygen delivery [10]–[12]. Radiological and histopathological findings frequently demonstrate necrosis, infarction, or hypodensity in this brain region, as exemplified in cases reported by Aoshima et al. [11] and Shah et al. [12]. These lesions correlate with severe neurological symptoms, including hemiplegia and cognitive impairment, and are considered hallmark features of prolonged CO exposure.

Pulmonary pathology is another significant finding in fatal cases. Autopsies often reveal pulmonary congestion and the presence of pink frothy fluid within the airways, indicative of acute pulmonary edema secondary to hypoxia [16],[19]. Hosahally et al. [16] reported pink lungs in a victim of accidental CO poisoning, while Singh et al. [19] noted tracheal soot deposition alongside lung congestion in a case involving charcoal combustion in an enclosed space.

Other systemic findings can include generalized rigor mortis, conjunctival hyperemia, and tissue hypoxia across multiple organs [20]. In certain cases, soot particles were identified in the respiratory tract, suggesting concurrent smoke inhalation from incomplete combustion [19]. These findings are particularly relevant in distinguishing CO poisoning from other causes of hypoxic death.

From a forensic and diagnostic perspective, integrating gross pathological findings with toxicological analysis (COHb measurement) substantially strengthens the determination of CO poisoning as the cause of death. This is especially crucial in cases where ante-mortem clinical data are unavailable or exposure was not witnessed [17]–[20]. Such integration ensures accurate cause-of-death certification, guides public health surveillance, and informs preventive strategies targeting environmental and behavioral risk factors.

Environmental and Behavioral Risk Factors

Environmental conditions played a decisive role in the occurrence of fatal carbon monoxide (CO) poisoning across the reviewed cases. The vast majority of incidents took place in enclosed or poorly ventilated environments, where combustion of charcoal briquettes allowed CO to accumulate to lethal concentrations [13],[14]. Common scenarios included residential bedrooms, bathrooms, and sealed shelters, as well as garages where combustion sources such as generators or vehicles were operated alongside briquette burning [12],[15],[16]. In some cases, the intentional sealing of ventilation outlets – such as taping window and door edges – was documented as part of planned suicide attempts [15].

The risk was particularly elevated during night-time or prolonged indoor exposure, when occupants were asleep or unaware of rising CO levels. Several suicide cases involved individuals deliberately choosing such conditions to maximize exposure duration and prevent intervention [8],[12],[15]. In group suicide incidents, as reported by Salil et al. [8], environmental risk was compounded by the number of people sharing the enclosed space, thereby increasing the rate of oxygen depletion and CO build-up.

Behavioral factors also significantly influenced risk. Disabling or absence of CO detectors was reported in several accidental cases, limiting the possibility of early warning [9], [20]. In Jian-qiang's [20] case, the CO alarm present in the tent was rendered non-functional due to missing batteries. Similarly, Desai and Shukla [9] described an incident where a domestic CO detector had been intentionally disconnected during heater repair, which subsequently contributed to undetected CO accumulation.

Cultural and socio-economic factors may also play an indirect role, particularly in regions where charcoal briquettes remain a low-cost, accessible fuel source for heating and cooking [3]. In low-resource settings, the use of combustion appliances in enclosed living areas is often driven by economic necessity and lack of awareness regarding ventilation requirements. Moreover, the portability and discreet nature of charcoal briquettes make them a commonly selected method for suicide in some populations [8],[12],[15].

From a public health perspective, these findings underscore the need for targeted risk mitigation strategies, including mandatory installation and maintenance of CO detectors in residential and commercial properties, education on safe fuel use, and stricter enforcement of building ventilation standards [13],[14],[21]. Addressing behavioral components, such as disabling safety devices or neglecting maintenance, is equally critical, as technical safeguards can only be effective when properly utilized.

Prevention and Public Health Implications

The evidence from this review indicates that fatal carbon monoxide (CO) poisoning associated with charcoal briquette use is largely preventable through a combination of engineering controls, public health interventions, and behavioral modifications. The most effective preventive measure is ensuring adequate ventilation in any environment where combustion occurs. Adequate air exchange prevents CO accumulation to hazardous concentrations, reducing the risk of both accidental and intentional exposure [13],[14],[21].

Installation of carbon monoxide detectors in residential and commercial settings is strongly recommended. Functional detectors can provide early warning, allowing individuals to evacuate and seek medical attention before CO levels reach toxic thresholds. Several cases in this review demonstrated that the absence, malfunction, or deliberate disabling of such devices contributed to fatalities [9],[20]. Public health policies mandating the installation and maintenance of CO detection systems—particularly in buildings using solid fuel or gas appliances—have been shown to significantly reduce mortality in other high-income countries, suggesting applicability in broader contexts [5],[21].

Public education campaigns are equally critical. Awareness programs should focus on the silent and non-specific nature of CO poisoning symptoms, the dangers of burning charcoal indoors, and the importance of proper ventilation. Educational materials must target vulnerable populations, including low-income households reliant on charcoal for heating and cooking, as well as communities with higher suicide rates involving combustion methods [3],[8],[15].

Mental health interventions represent another vital prevention pillar, particularly for suicide-related cases. Studies in this review show that charcoal burning is a common method of suicide in certain populations [8],[12],[15]. Integrating CO poisoning awareness into mental health screening, counseling, and crisis intervention programs could help reduce intentional exposures.

From a policy standpoint, governments and regulatory bodies should adopt a multi-layered strategy that combines building code requirements, mandatory safety device installation, targeted public education, and accessible mental health services. Collaborative efforts between public health authorities, emergency medical services, and community organizations are essential to ensure broad implementation and sustained impact.

Ultimately, preventing CO-related deaths from charcoal briquette use requires a comprehensive, evidence-based approach that addresses environmental, behavioral,

and psychosocial risk factors simultaneously. By implementing these measures, the burden of CO poisoning can be significantly reduced, safeguarding public health and saving lives.

This review is limited by the small number of included studies (n = 10), most of which were case reports or case series with limited generalizability. Geographical representation was uneven, potentially underrepresenting regions where CO poisoning cases are underreported. Publication bias may exist, as severe cases are more likely to be published than non-fatal or mild exposures. Differences in COHb measurement methods and autopsy protocols across studies could also affect data comparability. Despite these limitations, the findings consistently highlight key risk factors and mortality patterns relevant for prevention strategies.

4. Conclusion

Fatal carbon monoxide (CO) poisoning from charcoal briquette combustion remains a significant yet preventable public health problem, with most deaths occurring in enclosed or poorly ventilated spaces and carboxyhemoglobin (COHb) levels commonly exceeding 30%, a threshold strongly associated with mortality. Clinical manifestations range from mild neurological symptoms to severe hypoxic injury, with characteristic pathological findings including cherry-red discoloration and globus pallidus damage. To reduce the burden of CO-related mortality, key measures include mandating the installation of functional CO detectors in buildings using combustion appliances, enforcing ventilation standards through building codes, conducting targeted public education on the risks of indoor charcoal burning, integrating CO poisoning prevention into suicide prevention programs, and strengthening surveillance and reporting systems. Implementing these strategies could substantially lower the incidence of CO-related deaths.

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Conflict of Interest:

The authors declare no conflict of interest related to this study.

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