RESEARCH PAPER

Silent Hypoxia Unmasked: A Case Series of Suspected Acute Carbon Monoxide Poisoning Following an Enclosed Workplace Fire

Dr. Lakshmi M[1], Dr. Ajay Pawar[2*], Dr Shailesh V Udapudi [3], Dr Mahesh A Hatti[4], Dr Anas T C[5], Dr Haneena Haneef [6], Dr Mohammed Unais A[7], Dr Jishnu K S[8], Dr Sneha Joji[9], Dr Mariya Iju[10]

[1]Junior resident and Post graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010 lakshmimkrishna.364@gmail.com,

[2]Assistant Professor Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010 ajaypawardr@gmail.com,*

[3]Professor & Head of Department Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[4]Senior Resident Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[5]Junior Resident & Post graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[6]Junior Resident & Post Graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[7]Junior Resident and Post graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[8]Junior Resident and Post Graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[9]Junior Resident and Post graduate Department of Emergency Medicine, J.N Medical college ,Belagavi,KLE Academy of Higher Education and Research,Deemed to be University,Belagavi,Karnataka,India 590010

[10]CMO Alphonsa Hospital,Anakkara Idukki,Kerala

ABSTRACT

Background: Carbon monoxide (CO) poisoning is a common and potentially life-threatening consequence of enclosed-space fires. Diagnosis is frequently delayed because conventional pulse oximetry may remain normal despite significant tissue hypoxia, a phenomenon termed silent hypoxia.

Aim: To describe the clinical presentation, physiological findings, management, and short-term outcomes of patients with suspected acute CO poisoning following an enclosed workplace fire.

Methods: This descriptive case series included ten female patients presenting to a tertiary care emergency department within eight hours of smoke exposure from an enclosed workplace fire. Clinical features, pulse oximetry, arterial blood gas (ABG) parameters, radiological findings, treatment, and outcomes were analyzed.

Results: All patients presented with neurological, respiratory, or gastrointestinal symptoms, most commonly altered sensorium and breathlessness. Pulse oximetry readings were normal in all cases (96–99% on room air). ABG analysis consistently demonstrated respiratory alkalosis with low $PaCO_2$ and reduced bicarbonate, while PaO_2 remained normal. Chest radiographs and ECGs were unremarkable. All patients received 100% oxygen via non-rebreather mask, resulting in rapid symptomatic improvement within 15–30 minutes. None required ventilatory support or hyperbaric oxygen therapy.

Conclusion: Normal pulse oximetry does not exclude carbon monoxide poisoning in enclosed- space fire exposure. Recognition of silent hypoxia and early administration of high-flow oxygen are essential for prompt recovery and favorable outcomes.

Keywords: Carbon monoxide poisoning; Silent hypoxia; Enclosed-space fire; Smoke inhalation; Workplace exposure; Pulse oximetry limitation; Respiratory alkalosis; Arterial blood gas; Oxygen therapy; Emergency medicine; Inhalational injury.

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*****Author for Correspondence:* ajaypawardr@gmail.com

INTRODUCTION

Carbon monoxide (CO) poisoning is one of the most common and potentially fatal forms of toxic inhalational exposure worldwide, particularly in the context of fires occurring in enclosed or poorly ventilated spaces [1]. Carbon monoxide is a colorless, odorless, and non-irritant gas produced by incomplete combustion of carbon-containing materials. Because it lacks warning properties, exposure often goes unrecognized until clinical manifestations develop, by which time significant tissue hypoxia may already be present [2]. Occupational and workplace fires represent an important yet under-reported source of acute CO exposure, especially in low- and middle-income countries where industrial safety measures and early detection systems may be suboptimal [3].

The pathophysiology of carbon monoxide poisoning is multifactorial. CO binds to hemoglobin with an affinity approximately 200–250 times greater than that of oxygen, forming carboxyhemoglobin (COHb) and thereby reducing the oxygen-carrying capacity of blood [1]. In addition, CO shifts the oxyhemoglobin dissociation curve to the left, impairing oxygen release at the tissue level. Beyond its effects on hemoglobin, CO directly interferes with cellular respiration by inhibiting mitochondrial cytochrome oxidase, leading to impaired oxidative phosphorylation and cellular hypoxia [4]. Secondary inflammatory responses, oxidative stress, and endothelial dysfunction further contribute to tissue injury, particularly in the brain and myocardium [5].

A major diagnostic challenge in carbon monoxide poisoning is the phenomenon of so-called "silent hypoxia." Conventional pulse oximeters measure the percentage of hemoglobin saturated with oxygen but are unable to differentiate oxyhemoglobin from carboxyhemoglobin [6]. As a result, patients with significant CO exposure may demonstrate apparently normal or near-normal oxygen saturation values despite profound tissue hypoxia. This misleading reassurance can delay diagnosis and treatment, increasing the risk of acute complications and delayed neurological sequelae [1,7]. Symptoms of CO poisoning are often nonspecific and may include headache, dizziness, nausea, vomiting, dyspnea, confusion, and altered sensorium, all of which can be mistakenly attributed to anxiety, heat exposure, or other inhalational irritants encountered during fires [1].

Arterial blood gas (ABG) analysis may provide additional physiological clues in suspected cases. Although PaO_2 is often normal, patients may exhibit patterns of hyperventilation, including respiratory alkalosis with low $PaCO_2$, reflecting a compensatory response to tissue hypoxia and stress [8]. However, definitive diagnosis relies on measurement of COHb levels using co-oximetry. Importantly, COHb concentrations decline rapidly once high-flow oxygen therapy is initiated, and delayed sampling may yield falsely low or normal values. Therefore, a high index of clinical suspicion remains crucial, particularly in cluster presentations following enclosed-space fires [9].

Early recognition and prompt administration of 100% oxygen are the cornerstones of management. High-concentration oxygen significantly reduces the half-life of COHb, enhances dissociation of CO from hemoglobin, and improves tissue oxygen delivery [1]. While hyperbaric oxygen therapy may be indicated in selected severe cases, timely normobaric oxygen alone is often sufficient for patients with mild to moderate poisoning and can lead to rapid symptomatic improvement [10].

This case series describes a cluster of patients presenting to the emergency department following an enclosed workplace fire, all of whom exhibited clinical features suggestive of acute carbon monoxide poisoning despite normal pulse oximetry readings. By highlighting the presentation, physiological patterns, and response to treatment, this series aims to emphasize the importance of clinical vigilance, early oxygen therapy, and recognition of silent hypoxia in similar occupational exposure scenarios.

AIMS AND OBJECTIVES

AIM:

To describe the clinical presentation and early physiological features of suspected acute carbon monoxide poisoning in patients exposed to an enclosed workplace fire despite normal pulse oximetry readings.

OBJECTIVE:

To assess the immediate clinical response to high-flow oxygen therapy in patients with suspected carbon monoxide poisoning following an enclosed-space workplace fire.

CASE PRESENTATIONS

Exposure Context: 10 patients were female workers exposed to smoke following a fire in an enclosed workplace with poor ventilation. The patients presented sequentially to the emergency department within an eight-hour window. None had significant past medical comorbidities, smoking history, or prior neurological or cardiopulmonary disease. There was no history of trauma, burns, or loss of consciousness at the scene. All patients were evaluated using a standardized emergency protocol.

Case 1

Patient Profile and Time of Presentation: A 36-year-old female presented to the emergency department at 14:30 hours following smoke exposure in an enclosed workplace fire.

Clinical Presentation: She complained of acute breathlessness associated with altered sensorium. There was no history of loss of consciousness, seizures, chest pain, or trauma. Co- workers reported confusion and inappropriate responses.

Examination Findings: On examination, the patient was hemodynamically stable with mild tachypnea.

Oxygen saturation on room air was 98%. Cardiovascular, respiratory, and neurological examinations revealed no focal abnormalities.
Investigations: Arterial blood gas analysis demonstrated respiratory alkalosis with low $PaCO_2$ and reduced bicarbonate, with normal PaO_2. Chest radiograph and electrocardiogram were normal.
Management and Outcome: She was treated with 100% oxygen via a non-rebreather mask, resulting in rapid improvement in sensorium and respiratory symptoms within 20 minutes.

Case 2

Patient Profile and Time of Presentation: A 22-year-old female presented at 15:10 hours after smoke inhalation at the workplace.
Clinical Presentation: She reported breathlessness and nausea without headache, dizziness, or altered consciousness.
Examination Findings: Vital signs were stable with mild tachypnea. Oxygen saturation was 97% on room air. Systemic examination was unremarkable.

Investigations: ABG analysis showed respiratory alkalosis with low $PaCO_2$. Chest radiograph and ECG were normal.
Management and Outcome: High-flow oxygen therapy was initiated, leading to complete resolution of symptoms within 15 minutes.

Case 3

Patient Profile and Time of Presentation: A 26-year-old female presented to the emergency department at 15:45 hours following exposure to dense smoke during an enclosed workplace fire.
Clinical Presentation: The patient complained of dizziness, a sense of lightheadedness, and progressive difficulty in concentrating. Co-workers reported abnormal behavior, including slowed responses and inappropriate answers, prior to hospital arrival. There was no history of loss of consciousness, seizures, chest pain, or visual disturbances.
Examination Findings: On examination, the patient was confused but arousable. She was afebrile and hemodynamically stable. Respiratory rate was mildly elevated. Oxygen saturation on room air was 99%. Cardiovascular and respiratory system examinations were normal. Neurological examination revealed altered sensorium without focal neurological deficits.
Investigations: Arterial blood gas analysis revealed respiratory alkalosis characterized by low $PaCO_2$ and reduced bicarbonate levels, with normal PaO_2. Chest radiograph and electrocardiogram did not reveal any abnormalities.
Management and Outcome: The patient was immediately administered 100% oxygen via a non-rebreather mask. Within 20 minutes, her sensorium improved significantly, and she became fully oriented. She remained asymptomatic during observation and was discharged in stable condition.

Case 4

Patient Profile and Time of Presentation: A 34-year-old female presented at 16:20 hours after smoke exposure at her workplace.
Clinical Presentation: She complained of breathlessness associated with nausea and a feeling of chest discomfort. The patient also described anxiety and restlessness but denied vomiting, headache, or altered consciousness.
Examination Findings: She was alert and oriented, with stable vital signs and mild tachypnea. Oxygen saturation on room air was 96%. Physical examination of the cardiovascular and respiratory systems was unremarkable, and neurological examination was normal.
Investigations: ABG analysis showed respiratory alkalosis with low $PaCO_2$ and reduced bicarbonate. Chest radiograph and ECG were normal.
Management and Outcome: High-flow oxygen therapy was initiated promptly. The patient showed significant symptomatic improvement within 30 minutes, with resolution of breathlessness and nausea. She was kept under observation and discharged without complications.

Case 5

Patient Profile and Time of Presentation: A 29-year-old female presented at 17:00 hours following smoke inhalation.
Clinical Presentation: The patient presented primarily with altered sensorium. Relatives reported disorientation, confusion, and reduced attention span. There were no respiratory complaints, headache, nausea, or vomiting.
Examination Findings: On examination, she was confused but obeying commands. Vital signs were stable, and oxygen saturation was 98% on room air. Neurological examination revealed altered mental status without focal deficits. Other systemic examinations were normal.
Investigations: Arterial blood gas analysis revealed hyperventilation physiology with low $PaCO_2$ and reduced bicarbonate. Chest radiograph and ECG were within normal limits.
Management and Outcome: She was treated with 100% oxygen via non-rebreather mask. Complete normalization of sensorium occurred within 20 minutes. She remained clinically stable during observation.

Case 6

Patient Profile and Time of Presentation: A 30-year-old female presented at 17:40 hours.

Clinical Presentation: She complained of acute onset breathlessness and chest tightness following prolonged exposure to smoke. There was no associated chest pain, palpitations, dizziness, or syncope.
Examination Findings: The patient was anxious but alert. Vital signs were stable, with mild tachypnea. Oxygen saturation on room air was 97%. Cardiovascular and respiratory examinations were unremarkable.

Investigations: ABG analysis demonstrated respiratory alkalosis. Chest radiograph and ECG showed no abnormalities.
Management and Outcome: High-flow oxygen therapy was initiated, leading to rapid relief of symptoms within 15–20 minutes. She was observed and discharged in stable condition.

Case 7

Patient Profile and Time of Presentation: A 29-year-old female presented at 18:30 hours after smoke exposure.
Clinical Presentation: She reported nausea, dizziness, generalized weakness, and a feeling of impending faintness. There was no history of vomiting, headache, or altered sensorium.
Examination Findings: On examination, she was conscious and oriented with stable hemodynamics. Oxygen saturation was 99% on room air. Systemic and neurological examinations were normal.
Investigations: Arterial blood gas analysis revealed respiratory alkalosis with low $PaCO_2$. Chest radiograph and ECG were normal.
Management and Outcome: Following administration of 100% oxygen, her symptoms resolved completely. She remained asymptomatic during observation.

Case 8

Patient Profile and Time of Presentation: A 29-year-old female presented at 19:15 hours.

Clinical Presentation: The patient presented with breathlessness and altered sensorium. She appeared disoriented and complained of severe air hunger. There was no seizure activity or loss of consciousness.
Examination Findings: Vital signs were stable with mild tachypnea. Oxygen saturation was 96% on room air. Neurological examination revealed confusion without focal neurological deficits.
Investigations: ABG analysis showed respiratory alkalosis with low $PaCO_2$ and reduced bicarbonate. Chest radiograph and ECG were normal.

Management and Outcome: High-flow oxygen therapy resulted in rapid improvement in mental status and respiratory symptoms within 25 minutes.

Case 9

Patient Profile and Time of Presentation: A 20-year-old female presented at 20:30 hours.

Clinical Presentation: She complained of repeated vomiting, headache, and breathlessness following smoke exposure. She also reported dizziness and nausea prior to vomiting.
Examination Findings: She was alert and oriented. Vital signs were stable, and oxygen saturation on room air was 98%. No focal neurological deficits were identified.
Investigations: ABG analysis demonstrated respiratory alkalosis. Chest radiograph and ECG were unremarkable.
Management and Outcome: The patient showed significant symptomatic improvement within 30 minutes of oxygen therapy and antiemetic support.

Case 10

Patient Profile and Time of Presentation: A 19-year-old female presented at 22:00 hours following prolonged exposure to smoke in the workplace.
Clinical Presentation: She complained of nausea, headache, breathlessness, and generalized weakness. Symptoms had progressively worsened over several hours after the incident.
Examination Findings: She was conscious and oriented with stable vital signs. Oxygen saturation was 97% on room air. Systemic and neurological examinations were normal.
Investigations: Arterial blood gas analysis showed hyperventilation physiology with low $PaCO_2$ and reduced bicarbonate. Chest radiograph and ECG were normal.
Management and Outcome: Administration of 100% oxygen resulted in complete resolution of symptoms. The patient remained stable during observation and was discharged without complications.

Table 1: Summary of Clinical Characteristics, Investigations, and Outcomes of All Cases

Case No.	Age (years)	Time of Presentation	Predominant Symptoms	SpO_2 on Room Air (%)	ABG Pattern	Chest X-ray / ECG	Treatment Given	Clinical Outcome
1	36	14:30	Altered sensorium, breathlessness	98	Respiratory alkalosis, low $PaCO_2$	Normal	100% O_2 via NRBM	Rapid improvement within 20 min
2	22	15:10	Breathlessness, nausea	97	Respiratory alkalosis	Normal	100% O_2 via NRBM	Complete symptom resolution
3	26	15:45	Dizziness, altered sensorium	99	Respiratory alkalosis, low $PaCO_2$	Normal	100% O_2 via NRBM	Sensorium normalized

4	34	16:20	Breathlessness, nausea	96	Respiratory alkalosis	Normal	100% O_2 via NRBM	Symptom relief within 30 min
5	29	17:00	Altered sensorium	98	Hyperventilation physiology	Normal	100% O_2 via NRBM	Complete recovery
6	30	17:40	Breathlessness, chest tightness	97	Respiratory alkalosis	Normal	100% O_2 via NRBM	Rapid symptom resolution
7	29	18:30	Nausea, dizziness	99	Respiratory alkalosis	Normal	100% O_2 via NRBM	Asymptomatic post-treatment
8	29	19:15	Altered sensorium, breathlessness	96	Respiratory alkalosis	Normal	100% O_2 via NRBM	Marked clinical improvement
9	20	20:30	Vomiting, headache, breathlessness	98	Respiratory alkalosis	Normal	100% O_2 via NRBM	Symptoms resolved
10	19	22:00	Nausea, headache, breathlessness	97	Hyperventilation physiology	Normal	100% O_2 via NRBM	Complete recovery

DISCUSSION

This case series highlights the diagnostic and clinical challenges associated with acute carbon monoxide (CO) poisoning following enclosed workplace fires, particularly in the presence of normal pulse oximetry readings. All ten patients in this series presented with nonspecific neurological, respiratory, and gastrointestinal symptoms after smoke exposure, yet demonstrated apparently reassuring oxygen saturation values on room air. This phenomenon

of "silent hypoxia" represents a critical pitfall in emergency settings and underscores the importance of maintaining a high index of clinical suspicion in similar exposure scenarios.

Carbon monoxide exerts its toxic effects primarily through high-affinity binding to hemoglobin, forming carboxyhemoglobin (COHb) and reducing effective oxygen transport. In addition to impairing oxygen delivery, CO shifts the oxyhemoglobin dissociation curve to the left, limiting oxygen release at the tissue level [1]. Furthermore, CO disrupts mitochondrial oxidative phosphorylation by inhibiting cytochrome oxidase, leading to cellular hypoxia independent of hemoglobin binding [11]. These combined mechanisms explain why patients may develop significant symptoms despite preserved PaO_2 and normal pulse oximetry readings, as conventional oximeters are unable to differentiate oxyhemoglobin from COHb [12].

A notable finding in this case series was the consistent arterial blood gas pattern demonstrating respiratory alkalosis with low $PaCO_2$ and reduced bicarbonate levels. Although PaO_2 values remained within normal limits, the presence of hyperventilation physiology likely reflects an early compensatory response to tissue hypoxia and systemic stress. Similar ABG patterns have been described in early or mild CO poisoning, where hypoxia-induced stimulation of central and peripheral chemoreceptors leads to increased respiratory drive [2]. In settings where co- oximetry is unavailable or delayed, recognition of this ABG pattern in the appropriate clinical context may serve as a useful adjunctive clue to prompt early treatment.

The normal carboxyhemoglobin levels observed in this cohort should be interpreted with caution. In all cases, COHb estimation was performed after initiation of high-flow oxygen therapy, which rapidly reduces the half-life of COHb from approximately 4–5 hours on room air to less than one hour with 100% oxygen. Consequently, a normal or low COHb level obtained after oxygen administration does not exclude preceding CO exposure. This emphasizes a key practical lesson: treatment should never be delayed for confirmatory testing, and blood sampling for COHb, if feasible, should ideally be performed prior to oxygen therapy without compromising patient safety.

All patients in this series demonstrated rapid and complete symptomatic improvement following administration of 100% oxygen via non-rebreather mask. High-flow oxygen remains the cornerstone of treatment in acute CO poisoning, as it accelerates dissociation of CO from hemoglobin, improves tissue oxygenation, and mitigates ongoing cellular injury. None of the patients required ventilatory support or hyperbaric oxygen therapy. While hyperbaric oxygen has been advocated in selected cases—such as severe neurological impairment, cardiovascular

involvement, severe metabolic acidosis, pregnancy, or markedly elevated COHb levels—its role remains debated, and careful patient selection is essential. The favorable outcomes observed in this cohort suggest that early recognition and timely normobaric oxygen therapy may be sufficient in mild to moderate cases.

The cluster nature of these presentations also highlights the importance of situational awareness in emergency departments. Multiple patients presenting from the same enclosed environment with similar nonspecific symptoms should immediately raise suspicion of a shared toxic exposure. Early identification of such patterns allows for rapid triage, initiation of appropriate therapy, and implementation of public health and occupational safety measures to prevent further exposure.

This case series has several limitations. The absence of pre-oxygenation COHb measurements limits definitive biochemical confirmation of CO poisoning. The sample

size is small and derived from a single center, restricting generalizability. Additionally, long-term follow-up for delayed neurological sequelae was not systematically performed. Despite these limitations, the consistent clinical pattern and uniform response to oxygen therapy strengthen the likelihood of CO exposure as the underlying etiology.

In conclusion, this case series reinforces that normal pulse oximetry does not exclude carbon monoxide poisoning in patients exposed to smoke from enclosed-space fires. Recognition of silent hypoxia, attention to subtle physiological cues such as respiratory alkalosis on arterial blood gas analysis, and prompt administration of high-flow oxygen are critical to achieving favorable outcomes. Heightened clinical vigilance remains the most important tool in preventing missed or delayed diagnosis of this potentially life-threatening condition [13].

CONCLUSION

This case series highlights the diagnostic challenge posed by acute carbon monoxide poisoning following enclosed workplace fires, particularly in the presence of normal pulse oximetry readings. All patients presented with nonspecific neurological, respiratory, and gastrointestinal symptoms, emphasizing the deceptive nature of "silent hypoxia" associated with carbon monoxide exposure. A consistent arterial blood gas pattern of respiratory alkalosis with low $PaCO_2$ was observed, which may serve as an important early physiological clue in the appropriate clinical context. Prompt administration of 100% oxygen via non-rebreather mask
resulted in rapid and complete symptomatic improvement in all cases, underscoring the effectiveness of early oxygen therapy. Normal carboxyhemoglobin levels obtained after oxygen administration should not be interpreted as exclusion of prior exposure. These findings reinforce the need for high clinical suspicion, early intervention, and awareness of the limitations of pulse oximetry in smoke-exposed patients. Timely recognition and treatment remain critical in preventing morbidity and ensuring favorable outcomes in suspected carbon monoxide poisoning.

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