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Case Report

Carbon Monoxide as a Hidden Environmental Trigger of Seizure: A Case Report

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Abstract

Introduction: This study aimed to report a case of covert carbon monoxide (CO) poisoning presenting with seizure as the sole initial symptom. It also aimed to emphasize the diagnostic difficulty caused by the nonspecific clinical manifestations of CO poisoning. Delayed diagnosis in such cases may lead to serious morbidity or mortality. Therefore, CO poisoning should be considered in patients presenting with unexplained seizures.

Case Presentation: A 32-year-old man with no contributory medical history presented to the hospital with decreased consciousness, seizures, blurred vision, impaired balance, and hearing loss after spending the night alone at home. When he arrived at the hospital, he had hearing loss and balance disturbance. Nasal oxygen therapy (4–6 L/min) was administered. Initial lab results and brain imaging were unremarkable, and a diagnosis of "seizure of unknown origin" was made. A toxicology consultation and fire department inspection revealed CO levels over 150 ppm due to a leak from the central heating system.

Conclusion: This case demonstrates that CO poisoning may present solely with acute neurological symptoms like seizures, without the classic signs of exposure. In patients with unexplained neurological findings, especially those who have spent time in enclosed spaces with central heating, CO poisoning should be considered. Training healthcare staff, increasing public awareness, and using CO detectors are crucial for prevention and timely diagnosis.

Keywords: carbonmonoxide poisoning, Seizure, Biomarkers

Introduction

Poisoning remains a significant public health concern globally. In Iran, poisoning is a leading cause of hospital admissions and death. CO is one of the major causes of poisoning, accounting for over 20% of emergency visits(1). The incidence of CO poisoning in Iran ranges from 1.9 to 37 per 100,000 population, with mortality rates up to 20% reported in western provinces. In 2016, the Iranian Legal Medicine Organization recorded 836 CO-related deaths (2-4). CO is a toxic, colorless, odorless gas produced by incomplete combustion of carbon-based fuels and organic compounds in gas heaters, motor vehicles, poorly ventilated appliances, and stoves. CO binds to hemoglobin with an affinity over 200 times greater than oxygen, reducing available hemoglobin for oxygen transport, leading to hypoxia and severe outcomes, including neurological damage and death(5). Clinical signs range from mild (headache, dizziness, and nausea) to severe (confusion, unconsciousness, hypoxia, and respiratory failure). Around 40% of cases may develop delayed neurological sequelae (DNS) from 3 to 240 days post-exposure. Statistically, young men are at greater risk(6). This article presents a case of seizure linked to covert CO exposure in a healthy young male.

Case presentation

A 32-year-old man returned home alone at 3:00 AM. By 6:00 AM, he was unresponsive to calls. At 11:00 AM, emergency services transported him with reduced consciousness, stertorous breathing, and oral frothing. He was diagnosed with seizure and hearing loss but discharged from the first hospital against medical advice. He presented to a second hospital at 10:00 PM.

History revealed that the patient regained consciousness during transfer and subsequently developed symptoms, including headache, hearing loss, blurred vision, vomiting,

and balance disturbance. No prior illnesses, allergies, or medications were recorded. Vitals: alert, blood pressure 120/70 mm Hg, heart rate 96 beats/min, respiratory rate 24 breaths/min, and peripheral oxygen saturation 98%. General examination was normal; neurological findings included reactive pupils, hearing loss, blurred vision (FC 5 m), normal reflexes, and the need for assistance for walking. Because no significant changes were observed in subsequent laboratory tests, only the first day's results are reported to avoid redundancy. The laboratory findings are presented in Table 1.

Diagnostic workups (EEG, ECG, echocardiogram, MRI, audiometry, LP, and meningitis PCR) were unremarkable. Furthermore, due to the delayed diagnosis and the time elapsed, the assessment of carboxyhemoglobin (COHb) levels could not be performed. The home lacked gas heaters or water heaters; no odor or eye irritation was reported. Central heating was used. Fire department inspection found CO levels over 150 ppm (acceptable: < 25 ppm), traced to a boiler flue leak entering through wall cracks

The patient was not seen having a seizure, but seizure evidence was apparent. Conservative treatment was administered. Two months post-exposure, the patient reported persistent insomnia, unilateral hearing and vision loss, memory impairment, balance issues, and inability to drive.

Table1. Demonstration of laboratory finding of the patient

Test	Result	Reference value	Unit
Blood gas			
PH	7.41	7.35–7.45	
PCO_2	32.6	35–45	mm Hg
BE	−2.7	−2–+2	mEq/L
BEecf	−3.7		mEq/L
HCO_3	21.0	22–26	mEq/L
PO_2	28	80–100	mm Hg
O2 sat	54.8		
Biochemistry			
Blood sugar	115	70–140	mg/dL
Urea	58	13–43	mg/dL
Creatinine	1.6	0.9–1.3	mg/dL
Calcium	9.5	8.6–10.3	mg/dL
Phosphorous	3.5	2.4–4.4	mg/dL
Na	137		mEq/L
K	4.1	3.5–5.2	mEq/L
SGOT	90	< 40	IU/L
SGPT	48	< 45	IU/L
Alkaline phosphatase	225	80–306	IU/L
Magnesium	2.07	1.8–2.6	mg/dL
CRP quantity	128.6	< 6	mg/dL
CBC & Diff			
WBC	9.1	3.5–10.5	$\times 10^3/\mu L$
RBC	49	4.3–5.7	$\times 10^3/\mu L$
Hb	16.3	13.5–17.5	g/dL
Hct	44.1	35–46	%
MCV	90	81–95	fL
MCH	33	27–34	pg
MCHC	37	32–37	g/dL
Platelet	281	145–420	$\times 10^3/\mu L$
RDW	13.0	11.6–14.6	%
PDW	10.7	10–17	fL
MPV	8.8	9.4–12.4	fL
P-LCR	16.3	17–45	%
ESR1st hr.	59	< 10	mm/hr
Differential • Neutrophils: 80.0% • Lymphocytes: 18.0% • Mixed cells: 2.0%			
Serology			
Wright	Negative	Negative	
2ME	Negative	Negative	
VDRL	Non-reactive	Non-reactive	
Urine / Toxicology			
Amphetamine and methamphetamine	Negative	Negative	
Opium alkaloids (morphine)	Negative	Negative	

Discussion

CO poisoning can cause serious neurological complications. The most fatal outcomes stem from delayed neurological sequelae (7, 8).

A 2020 retrospective study in China reviewed 20 patients with delayed encephalopathy post-CO exposure. Clinical signs included memory loss, personality changes, cognitive/executive dysfunction, mood disorders, Parkinsonism, dystonia, and akinetic mutism. MRI revealed bilateral white matter or basal ganglia lesions. Most sequelae improved, particularly cognitive deficits (from 95% to 25%) and psychiatric symptoms (from 95% to 55%) at 6-month follow-up (9).

Studies associate DNS with damage to basal ganglia and hippocampus, seizure-related regions. A cohort study of 8,264 CO-exposed vs. 41,320 unexposed individuals in China revealed CO exposure increased epilepsy risk. Adjusted hazard ratio showed patients aged 20–39 had nearly triple the risk compared to those over 65. Risk was similar between sexes(4, 10).

With greater CO exposure, severe acute neurological signs emerge: headache, dizziness, syncope, seizure, stroke, and unconsciousness(11).

Early diagnosis is hindered by nonspecific symptoms that mimic viral infections or food poisoning. Variability in individual sensitivity due to age, comorbidities, and hypoxia tolerance complicates recognition. Chronic or intermittent exposure may go unnoticed, especially in workplaces or residences with poor ventilation.

Addressing CO poisoning challenges requires multiple solutions. Education of the public and medical professionals enhances diagnosis. Porfig, sensitive diagnostic tools and environmental CO detectors in high-risk settings enable early alerts(12, 13).

Conclusion

This report illustrates that CO can act as a hidden environmental factor causing acute neurological symptoms like seizures, unconsciousness, balance disturbance, visual impairment, and hearing loss, even in the absence of classical exposure signs. The initial diagnosis was "seizure of unknown cause," but environmental investigation confirmed high CO levels from a hidden boiler flue leak.

This emphasizes the importance of evaluating living environments, especially poorly ventilated homes in winter. CO poisoning's neurological impact—including white matter damage and increased epilepsy risk—is well documented. Given the high incidence and over 800 annual CO-related deaths in Iran, CO poisoning should be considered in patients with unexplained neurological symptoms, particularly in urban settings with central heating.

Specialized training for healthcare professionals, increased public awareness, and use of home CO detectors are essential for effective prevention and early detection.

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Competing Interests

None.

Ethical Approval

All patient-identifing information has been anonymized. Informed consent was obtained from all participants included in the study.

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