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Case Report

From Recovery to Relapse: Delayed Neuropsychiatric Sequelae After Gas Geyser Related Carbon Monoxide Poisoning in a Thirteen-Year-Old

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Abstract

Carbon monoxide (CO) poisoning is a significant cause of toxic encephalopathy, particularly in low- and middle-income countries where gas-based appliances such as water heaters are commonly used in poorly ventilated settings. A characteristic feature is its biphasic course, with delayed neuropsychiatric sequelae (DNS) occurring after a lucid interval due to immune-mediated demyelination and oxidative injury. We report a 13-year-old previously healthy male who presented with coma following exposure to a gas water heater in a closed bathroom, requiring mechanical ventilation. After initial recovery, he remained asymptomatic for eight weeks before developing multifocal myoclonus and behavioural disturbances including irritability, aggression, and decline in scholastic performance. Neuroimaging revealed bilateral periventricular white matter changes consistent with delayed toxic leukoencephalopathy, and electroencephalography showed diffuse slowing with epileptiform discharges. A diagnosis of DNS secondary to CO poisoning was made, and he was treated with high-dose methylprednisolone and perampanel, resulting in partial clinical improvement. This case highlights the classical biphasic course with a relatively prolonged lucid interval and an atypical pediatric presentation, emphasizing the need for awareness, early recognition, and long-term follow-up in such preventable exposures.

Introduction

Carbon monoxide (CO) poisoning remains a significant global cause of toxic morbidity and hypoxic–ischemic brain injury, particularly in low- and middle-income countries where domestic fuel-based appliances are widely used [1]. CO is a colorless, odorless, and non-irritant gas produced during incomplete combustion of hydrocarbons and is often referred to as the silent killer. Gas water heaters using liquefied petroleum gas are an important but underrecognized source of CO exposure, especially in poorly ventilated environments. CO exerts toxicity by impairing oxygen

delivery and disrupting mitochondrial oxidative phosphorylation, resulting in oxidative stress and lipid peroxidation. These processes trigger delayed inflammatory cascades and immune-mediated demyelination, particularly affecting cerebral white matter [2]. A hallmark feature of CO poisoning is its biphasic course, where patients initially recover from acute exposure, followed by delayed neuropsychiatric sequelae (DNS) after a latent period [3].

DNS occurs in approximately 10–40% of survivors and is characterized by cognitive impairment, personality changes, movement disorders, and white matter involvement [2]. Although well described in adults, DNS is less frequently reported in pediatric populations and may present with variable or atypical manifestations, contributing to diagnostic delay.

We present a pediatric case demonstrating a classical biphasic trajectory with a relatively prolonged lucid interval, radiological evolution, electrophysiological correlates, and therapeutic challenges.

Case Description

A 13-year-old previously healthy male with right-handedness, normal developmental milestones, and no prior neurological or psychiatric illness was brought to the emergency department after being found unconscious in a closed bathroom following exposure to a domestic gas water heater. There was no history of preceding fever, trauma, seizure disorder, or toxin ingestion. On arrival, he was comatose and required emergent intubation and mechanical ventilation, which was continued for four days. He received supportive intensive care management along with empirical intravenous antibiotics during the acute phase. Magnetic resonance imaging (MRI) of the brain performed approximately two weeks after exposure demonstrated gyral diffusion restriction with T2/FLAIR hyperintensities, consistent with acute hypoxic-toxic encephalopathy secondary to carbon monoxide exposure. Following stabilization, he showed significant neurological recovery, regained full consciousness, and was discharged in a clinically stable condition.

Over the next two months, he remained apparently well and resumed routine daily activities, representing a lucid interval. However, approximately eight weeks after the initial exposure, he developed sudden onset involuntary jerky movements involving both upper and lower limbs. These movements were brief, lasting less than one second per episode, multifocal in distribution, and consistent with myoclonus. Concurrently, his parents noted significant behavioral changes including irritability, unprovoked aggression, emotional lability, and a marked decline in scholastic performance. There were no associated loss of consciousness, headache, vomiting, visual disturbances, autonomic symptoms, or bowel and bladder dysfunction.

On neurological examination at readmission, he was conscious, alert, and oriented with a Glasgow Coma Scale score of 15/15. Cranial nerve examination was normal. Motor examination revealed normal tone and strength in all four limbs, with preserved deep tendon reflexes graded 2+ symmetrically and bilateral flexor plantar responses. No cerebellar signs or extrapyramidal rigidity were noted. Systemic examination was unremarkable.

Repeat MRI brain revealed bilateral periventricular white matter hyperintensities without evidence of acute infarction or haemorrhage, suggestive of delayed toxic leukoencephalopathy. Electroencephalography demonstrated diffuse background slowing with multifocal epileptiform discharges, correlating with the clinical myoclonus and global cortical dysfunction. Cerebrospinal fluid analysis including an autoimmune encephalitis panel was negative. Routine hematological and biochemical investigations were within normal limits.

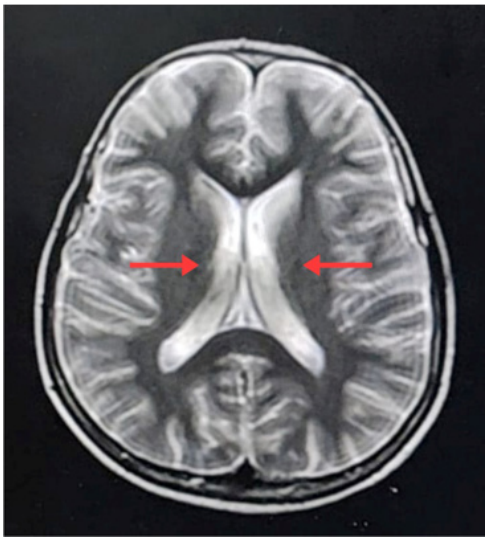

Figure 1: Axial T2-weighted Magnetic Resonance Imaging demonstrates extensive, symmetric periventricular white matter hyperintensities as pointed by red arrows and blurring of the grey-white matter interface.

A diagnosis of delayed neuropsychiatric sequelae secondary to carbon monoxide poisoning was made. The child was managed with high-dose intravenous methylprednisolone pulse therapy along with antiepileptic medication including perampanel for myoclonus. Gradual reduction in the frequency of myoclonic jerks and partial improvement in behavioural symptoms was observed over the subsequent weeks (Figure 1).

Discussion

Gas geysers are a popular choice for water heating because of their quick action and affordability, particularly in settings with inconsistent electricity supply. They use liquefied petroleum gas (LPG), mainly propane and butane, which on complete combustion produce carbon dioxide and water. However, many households lack proper flues or ventilation and often have compact bathroom spaces with inadequate airflow, leading to incomplete combustion and release of harmful gases such as carbon monoxide (CO). CO is a toxic, colorless, and odorless gas that binds to haemoglobin with high affinity, impairing oxygen delivery and causing tissue hypoxia. In addition, it disrupts mitochondrial oxidative phosphorylation, resulting in oxidative stress, lipid peroxidation, and delayed inflammatory responses that contribute to demyelination [2]. Clinical presentation varies widely, with acute exposure causing headache, dizziness, and confusion, while prolonged exposure may lead to seizures or coma. Delayed neuropsychiatric sequelae (DNS) occur due to immune-mediated processes triggered by CO-induced oxidative injury, rather than persistent hypoxia alone [3,4]. Although DNS is well described, it is less frequently reported in pediatric populations and may present with variable or atypical manifestations, contributing to its under recognition in global literature especially in developing countries.

The present case demonstrates the classical biphasic course of CO poisoning, characterized by an initial recovery followed by delayed neurological deterioration. The lucid interval of eight weeks observed here is relatively longer than the commonly reported duration of 2–40 days, indicating heterogeneity in disease progression [3]. The predominance of multifocal myoclonus and behavioral disturbances without significant motor deficits or parkinsonian features represents a less typical phenotype, particularly in children where such presentations are infrequently documented. The presence of bilateral periventricular white matter hyperintensities on MRI is consistent with delayed toxic leukoencephalopathy, reflecting demyelination, and supports previous evidence that early and follow-up neuroimaging can aid in predicting DNS with moderate sensitivity and high specificity [5]. Severe initial presentation with coma and requirement for intensive care, as seen in this case, has been identified as an important risk factor for the development of DNS in pediatric populations [6,7]. Management remains largely supportive, while hyperbaric oxygen therapy has shown variable benefit and remains limited in availability, corticosteroid therapy is increasingly utilized based on the inflammatory basis of DNS [2,7]. The use of perampanel is supported by glutamate-mediated excitotoxicity following CO exposure, providing symptomatic control of myoclonus. This case highlights that gas geyser associated CO poisoning, although preventable, can present with delayed and atypical neurological manifestations in children, emphasizing the need for increased awareness, early recognition, and long-term follow-up.

Conclusion

Gas geyser associated carbon monoxide poisoning is a preventable cause of significant neurological morbidity, with the potential for delayed and atypical neuropsychiatric sequelae, particularly in pediatric patients. This case underscores the importance of early recognition, awareness of biphasic presentation, and the need for long-term follow-up even after clinical recovery period.

Conflict of Interest

The authors declare no conflict of interest and no funding was received for the study.

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