

Carbon Monoxide Poisoning Secondary to Gas Geyser

Sanjay Jaiswal¹, Amit Batra², Ankur Verma³, Meghna Haldar³, Wasil Rasool Sheikh⁴

From ¹Principal Consultant and Head of Department, Department of Emergency Medicine, ²Senior Consultant, Department of Neurology, ³Consultant, ⁴Associate Consultant, Department of Emergency Medicine, Max Super Speciality Hospital, Patparganj, New Delhi, India.

Correspondence to: Dr. Ankur Verma; House 25, First Floor, Cedar Crest, Nirvana Country, Sector 50, Gurgaon, Haryana, India.
E-mail: anksv25@gmail.com.

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ABSTRACT

Carbon monoxide (CO) is a non-irritating, colorless and odorless gas produced by the incomplete burning of carbon-containing fossil fuels. CO poisoning remains a leading cause of unintentional poisoning worldwide. During winters, accidental CO poisoning has been reported mostly due to incomplete burning of coal. Here, we report the case of a young female patient who presented with hypoxic brain injury due to CO poisoning while using gas geyser in bathroom. The diagnosis of CO poisoning was made on the basis of history, vital signs, blood gas, and MRI scan. The patient recovered well after high-flow normobaric oxygen therapy and showed significant resolution on follow up MRI. A high index of suspicion, early recognition of symptoms by emergency physicians followed by careful history taking can help in making a clinical diagnosis of CO toxicity.

Keywords: CO poisoning, Gas geyser, Intoxication.

Carbon monoxide (CO) is a non-irritating, colorless and odorless gas produced by the incomplete burning of carbon-containing fossil fuels such as charcoal, wood, kerosene, or natural gas. CO was used by the humans for smelting iron and metallic ores since prehistoric times, Greek and Roman executions and was first described in the 11th century by the Spanish doctor Arnaldus se Villa [1]. CO poisoning still remains a leading cause of unintentional poisoning worldwide [2]. In the United States, CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually [3] but in India, the incidence of CO poisoning is remarkably low. Only a few reports on fatal carbon monoxide poisoning due to motor vehicle exhaust in India have been reported [4-7].

A high index of suspicion, early recognition of symptoms by emergency physicians followed by a careful history taking can help in making a clinical diagnosis of CO toxicity. This, in turn, would aid physicians in providing early care to such patients leading to a favorable outcome. We aim that our report would help emergency physicians to be more prudent in recognizing CO poisoning. Here, we present the case of CO poisoning in a young female due to gas geyser.

CASE REPORT

A 34-year-old, non-hypertensive, non-diabetic female was brought to the emergency department by her husband in a drowsy condition. The patient was found unconscious and unresponsive in her bathroom by her family.

The patient presented in tachycardia with a pulse of 124/min, blood pressure of 100/70 mm Hg, saturation of 74% on room air and a respiratory rate of 21/min. The patient was afebrile and had a normal sugar level of 82mg/dL. On auscultations, her chest was clear with good bilateral air entry and the patient was drowsy but arousable, appeared confused, slow in her responses and had slurred speech. She had no obvious motor deficit. There was no tongue bite or smell of any substance of abuse. The rest of the systemic examination was unremarkable.

Initial blood work showed a pO₂ of 28% (room air) on blood gases, total leucocyte count of 34,500/mm [3] and neutrophils

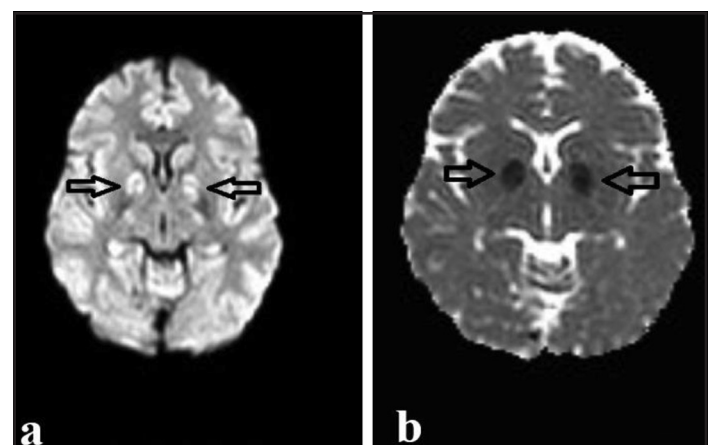


Figure 1: Magnetic resonance imaging (MRI) of the brain revealed (a) symmetric hyperintense foci on DWI in bilateral globus pallidus and (b) Corresponding hypointensity on ADC map suggesting restricted diffusion

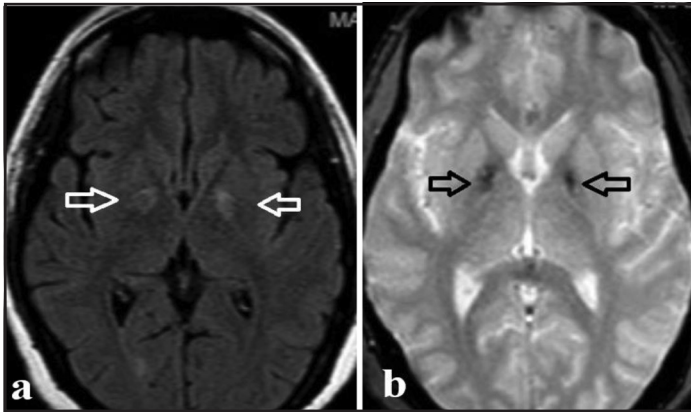


Figure 2: A 2 weeks MRI brain showed significant resolution of patient's lesion

of 85%. Remaining blood work was unremarkable. X-ray chest was unremarkable showing no focal lung parenchymal changes. The patient was started on high flow oxygen via face mask which brought the saturation up to 100%. The initial differentials included a seizure with the post-ictal state, some unknown poisoning or a vascular event (stroke).

The patient underwent Magnetic resonance imaging (MRI) of the brain which revealed symmetric hyperintense foci on Diffusion-weighted images (DWI) in bilateral globus pallidus and cerebellar hemispheres as well as hippocampal regions with the corresponding hypointensity on Apparent Diffusion Coefficient (ADC) map suggesting restricted diffusion (Fig. 1). All the changes were suggestive of CO poisoning or metabolic etiology. On taking a repeat history, it was revealed that the family used gas geysers in the house for heating water. A presumptive diagnosis of CO poisoning was made in view of the clinical picture, hypoxemia and MRI findings. Bedside EEG showed some background slowing but had no evidence of epileptiform activity.

The patient was continued on high flow oxygen, intravenous antibiotics and admitted under the care of neurology. Gradually the patient's sensorium improved, became more coherent but continued to have total amnesia of the episode. The patient was discharged within a week when the blood counts came back to normal and the patient was maintaining 100 % saturation on room air. On outpatient follow-up, the patient did not show any neurological sequelae and 2 weeks MRI brain showed significant resolution of her lesions (Fig. 2).

DISCUSSION

Gas geysers have emerged as a cost-effective; easy to use and an efficient method for heating water at homes especially in India. They run on liquid petroleum gas, the combustion of which leads to the generation of CO, hydrocarbons, and nitrogen oxides [8]. CO has 200-250 times greater affinity for hemoglobin than oxygen [9]. CO in high concentrations may lead to fatal outcomes due to the formation of carboxyhemoglobin (COHb) which impairs the oxygen-carrying capacity of the blood. The normal levels of COHb are less than 5% in smokers while it is less than

2% in non-smokers. Heavy smokers may have levels as high as 10-15% [10].

During the winter season, accidental CO poisoning has been reported in India mostly due to incomplete burning of coal [11]. CO poisoning can mimic other common ailments and hence can be missed leading to fatalities. It has been reported to be the most frequent cause of fatal poisoning, with an incidence rate of 31% [12]. There are very few cases reported about patients surviving CO intoxication due to gas geysers in India.

Patients may develop headache, nausea, dizziness, and confusion with levels of 10-30% and can become comatose at levels of 30-50%. At COHb levels higher than 50%, death may occur. Myocardial infarction, life-threatening dysrhythmias, and cardiac arrest are commonly described in victims of CO poisoning. Acute mortality in CO poisoning is usually due to ventricular dysrhythmias, probably caused by the accompanying hypoxia [13].

MRI appearances of the brain following acute CO poisoning are varied, and have previously been the subject of case reports or small studies; although the globus pallidus is the commonest site of abnormality in the brain [14]. The extent of damage correlates with clinical outcome and therefore aids in management and prognosis. The predilection for the globus pallidus may relate to the hypotensive effect of CO poisoning in the watershed territory of the arterial supply.

Treatment of patients exposed to CO and showing signs of toxicity include removal of the patient from the environment and administering high-flow normobaric oxygen. The use of hyperbaric oxygen in CO poisoning is still controversial and any consensus for its use is still needed [15]. We need to keep in mind to think of differential diagnoses like drug overdose, encephalitis, meningitis, cyanide intoxication, methemoglobinemia, trauma, severe depressions, etc while working up suspected CO poisoning patients.

CONCLUSION

In our case, the diagnosis of CO poisoning was made on the basis of history, vital signs, blood gas, and MRI scan. The patient recovered well after high flow normobaric oxygen therapy and follow-up MRI showed significant resolution of the initial CO poisoning findings. Public awareness of the sources of carbon monoxide and the harmful effects of the same is of utmost importance. Prevention precedes management. Having a high index of suspicion cannot be emphasized enough for all emergency physicians as early high flow oxygen therapy can be of vital importance even if the patient seems stable.

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