

Carbon Monoxide Poisoning in a Child: A Case Report

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Summary

Asani MO, Belonwu R, Rajasekaran S, Ibrahim M. Carbon Monoxide Poisoning in a Child: A Case Report. *Nigerian Journal of Paediatrics* 2004; 31:56. The exact incidence of carbon monoxide (CO) poisoning in Nigeria is unknown. Globally, CO poisoning is frequently unrecognized and under-reported since the clinical presentation is relatively non-specific. The circumstances usually involve an unsuspected increase of CO in an enclosed environment. We present the case of a five-year old girl who was brought to the Emergency Paediatrics Unit with altered consciousness and profound muscle weakness following exposure to exhaust fumes from a petrol powered electric generator. The generating set was usually operated within the kitchen during the preceding year, but on that particular day, a large refrigerator was placed beside the kitchen door thereby preventing its complete closure. She made an uneventful recovery following treatment with 100 percent oxygen. The case highlights the susceptibility of children to CO poisoning caused by operating petrol powered electric generators within the house. Health education of the community is necessary to avert such hazards.

Introduction

CARBON monoxide (CO) is a major indoor pollutant in the developing world due to practices such as the use of firewood indoors for cooking and heating.¹ The magnitude of this problem was highlighted by the World Health Organisation (WHO) which estimated that 2500 million people, the majority from developing nations, are exposed to excessive levels of indoor air pollution annually.¹ It is also an important cause of mortality worldwide.²⁻⁴ CO poisoning has been called "the disease of a thousand faces" and its symptoms range from vague malaise or flu-like symptoms to profound central nervous system dysfunction with overt psychiatric manifestations.⁵ It is therefore imperative that medical practitioners familiarize themselves with the diverse presentations of CO poisoning and develop a high level of suspicion whenever faced with such symptoms. This will involve the development of diagnostic criteria that are based more on clinical features rather than laboratory parameters, considering the paucity of laboratory equipment such as blood gas analyzers in the developing

world. Like most environmental hazards, CO poisoning is often the end-result of a series of interactions between man and his environment. This index case of a five-year old girl illustrates this.

Case Report

IG, a five-year old girl, was brought to the Emergency Paediatrics Unit (EPU) of the Aminu Kano Teaching Hospital (AKTH) on November 6, 2002, with a history of not waking up from the previous night's sleep. According to her parents, the family of six had gone to bed two nights prior to presentation and left their portable petrol generator on, in the kitchen. Further questioning revealed that the generator was kept in the kitchen for security reasons. This had been the practice for the previous three years. On this particular day, the family placed a large refrigerator against the door preventing its complete closure, thereby diverting the exhaust fumes of the generator into the adjacent common bedroom. The family woke 28 hours later to find that the generator had stopped working, having run out of fuel. However, it was difficult to rouse IG who seemed to be most severely affected. She remained drowsy and kept drifting back to sleep whenever left alone. Her parents were also concerned that the other children demonstrated some degree of physical weakness. They decided to bring the entire family to the hospital for evaluation and treatment.

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On examination, IG was comatose with a Glasgow coma scale of 8. Her mucous membranes were pink. She was acyanotic, anicteric, well hydrated and febrile with an axillary temperature of 38.6 °C. Evaluation of the central nervous system revealed an unconscious child with normal sized pupils that were symmetrical and reacted to light; other cranial nerves were intact with no detectable meningeal signs. Muscle bulk was normal, power was 3/5 in all the limbs with an increased tone globally and exaggerated knee jerk reflexes. Other systems were essentially normal.

Based on the history of exposure to generator exhaust fumes and abnormal CNS findings, a diagnosis of carbon monoxide poisoning was made. She was commenced on treatment with 100 percent oxygen. Due to the absence of appropriate facilities, measurement of blood carboxyhaemoglobin (COHb) could not be carried out. However, arterial blood gas analysis revealed a pH of 7.554 (7.350 - 7.450), $p\text{CO}_2$ of 28.9 mmHg (35 - 45), and $p\text{O}_2$ of 475 mmHg (80 - 100). Packed cell volume was 36 percent, while the random blood sugar, electrolytes and urea were essentially normal. She also received dexamethasone as treatment for presumed cerebral oedema.

She made remarkable progress and 24 hours later, she was completely well oriented. Muscle power had improved to 4/5, muscle tone and reflexes were normal. She was discharged home on the fifth day of admission with no residual motor deficit.

Discussion

A diagnosis of carbon monoxide poisoning can be made even in the absence of specific laboratory parameters if there is a positive history of exposure to fumes supported by compatible neurological and/or neuropsychiatric findings.⁸ When available, blood carboxyhaemoglobin levels should be determined to confirm the diagnosis.⁸ The importance of a clinical diagnosis is further strengthened by studies showing better correlation between severity of toxicity with neurological manifestations than with blood CO levels.^{9,10} In our patient, the confluence of symptoms and signs along with an identifiable source of CO excluded other aetiology and satisfied the criteria for diagnosis. All family members were affected and suffered CO poisoning in varying degrees. Our patient had the most profound manifestation for reasons that were not very clear.

Arterial blood gas analysis showed profound alkalosis, that was most probably due to respiratory compensation with $p\text{CO}_2$ of 28.9 mmHg occurring in response to lactic acid production which is known to be increased in CO poisoning.¹⁰ Even though the commonest metabolic

disorder in CO poisoning is one of acidosis, some workers have also reported the presence of overcompensated alkalosis in CO toxicity.^{10,11} This phenomenon is thought to be due to a delay in the blood-brain barrier recognition of peripheral pH correction, thus resulting in overcompensation.¹² The $p\text{O}_2$ of 475 mmHg is attributable to the O_2 therapy.

The clinical manifestation of toxicity is a result of tissue hypoxia resulting from the increased affinity of CO for haemoglobin, being 200-250 times that of oxygen.² The higher oxygen demand of the brain and myocardium make these organs more susceptible to hypoxia. Eventually an "oxygen debt" is incurred, leading to lactic acid production and up-regulation of cyclic guanosine monophosphate (cGMP),¹³ a smooth muscle relaxant. These mediators account for the motor dysfunction seen in CO poisoning.^{11,13} Infants and children are more prone to the effects of carbon monoxide because of their higher metabolic rates and developing nervous systems.⁵ This may explain the severity of the poisoning in our subject.

The management of CO poisoning involves the inhalation of 100 percent oxygen which competitively displaces CO from its transport site on the haemoglobin molecule, thereby decreasing its half life from 200-300 minutes to 60-90 minutes.⁶ The use of hyperbaric chamber where available, can further shorten the half life to 30 minutes.⁶ Hyperbaric oxygen therapy is the administration of 100 percent oxygen at two to three times atmospheric pressure, which significantly increases dissolved oxygen in blood. The clinical data on the use of the hyperbaric chamber for CO toxicity remains contradictory. Hawkins *et al*, found significantly higher neurological morbidity in those treated with normobaric oxygen (NBO) compared to those who received hyperbaric oxygen (HBO).³ In contrast, Scheinkestel *et al*, in a randomized controlled clinical trial, compared the two modalities and demonstrated poorer neuropsychological outcomes in those treated with HBO.⁷ Their trial concluded that HBO therapy did not show any benefit and might even worsen the outcome. Administration of 100 percent oxygen at atmospheric pressure remains convenient, safe and inexpensive. Other aspects of care are mainly supportive such as protecting the airway, monitoring intracranial pressure and restricting fluid input so as not to worsen any associated cerebral oedema.

This near fatal case of CO poisoning highlights the danger of inhalation of exhaust fumes. It is important to enlighten the public on the need to operate exhaust fumes-emitting machines in open spaces that are removed from residential buildings in order to prevent this health hazard.

References

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