Hypoglycemia in carbon monoxide poisoning: A case report

Akant Arora[1], Krunal Desai[1], Sandeep Jain[2], Manish Mehta[2]

From [1]DNB Resident, [2]Principal Consultant, Department of Emergency Medicine, Max Superspeciality Hospital, Saket, New Delhi, India.

Correspondence to: Dr. Akant Arora, Department of Emergency Medicine, Max Superspeciality Hospital, Saket, New Delhi-110001, India. E-mail: akant469@gmail.com

Received - 30 May 2019 Initial Review - 14 June 2019 Accepted - 22 July 2019

ABSTRACT

Carbon monoxide poisoning is a common presentation in the Emergency Department. The clinical presentation runs a spectrum, ranging from headache and dizziness to coma and death, with a mortality rate ranging from 1 to 3 %. Administration of normobaric 100% oxygen is the therapy of choice for most cases, while hyperbaric oxygen therapy is reserved for severe poisoning. Hyperglycemia is commonly seen with this poisoning but the association of hypoglycemia is not well described in human studies. Here, we present the case of hypoglycemia in carbon monoxide poisoning with good clinical outcome in a 22-year-old male.

Keywords: *Carbon monoxide poisoning, Carboxyhemoglobin, High flow oxygen, Hypoglycemia.*

Carbon monoxide (CO) is a non-irritant, colorless, odorless gas produced by the incomplete burning of carbon-containing fossil fuels. It is one of the commonly encountered poisons in our environment and a leading cause of poisoning worldwide [1]. Effect of carbon monoxide toxicity occurs both as a result of hypoxia and direct effect of carbon monoxide at the cellular level.The central nervous system (CNS) and heart are the most susceptible organs due to their high oxygen needs. Neuro-cognitive deficit occurs in 15-40% of patients, whereas, approximately one-third of moderate to severely poisoned patients exhibit cardiac dysfunction including arrhythmia, left ventricular systolic dysfunction and myocardial infarction [2]. Hypoglycemia in carbon monoxide poisoning has been shown to be associated with poor prognosis and high mortalityin some animal studies [3].

To the best of our knowledge, there are no human data available to support this association. Hence, we are presenting the case of hypoglycemia in carbon monoxide poisoning so that in future more studies can be done to validate this association.

CASE REPORT

A 22-year-old male patient was brought to the emergency department (ED) by his father in an unconscious state with a history of exposure to smoke in a closed room a few hours before. There was no evidence of associated tonic-clonic movements, tongue bite, frothing from mouth, vomiting, bladder or bowel incontinence or trauma. There was no history of fever, headache, chest pain and substance abuse prior to this episode. He also did not have any past medical history.

On initial assessment, the patient's airway was maintainable. He was tachypnoeic with a respiratory rate of 34 per minute and oxygen saturation of 84% at room air. His blood pressure was 80/60 mm Hg and he had tachycardia with a heart rate of 134/minute. He was drowsy with Glasgow coma score (GCS) of E1M3V1. His blood glucose was 34 mg/dl.

The patient was managed with high flow oxygen with a non-rebreathing oxygen face mask. Approximately, 100 ml of 50 percent Dextrose and 1-litre bolus intravenous normal saline were transfused. On reassessment, his vitals improved - oxygen saturation of 100% with high flow oxygen, blood pressure of 110/70 mm Hg, tachycardia reduced to 108/min and blood glucose of 283 mg/dl.Systemic examination revealed audible as well as auscultatory wheezing with the rest of the examination being normal. Pupils were normal size and well reacting to light and plantar reflex was flexor bilaterally.

The co-oximetry analysis showed severe metabolic acidosis with carboxyhemoglobin level (CoHb) of 45 gm% (Table 1). Chest

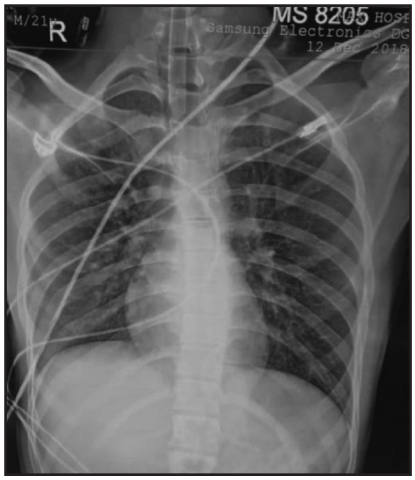

Figure 1: Normal chest X-ray in carbon monoxide poisoning case

Table 1: Comparison of blood gases at different times since arrival in the emergency department

Arterial blood gas	Time elapsed since arrival		
	10 min	150 min	330 min
pH	7.19	7.39	7.34
PCo2	42.1	25.6	39.8
pO2	59.5	460	493
COHb (%)	45	-	0.4
HCo3	13.6	15.3	21.1
Lactate	12.5	6.9	1.6

X-ray did not show any significant abnormality (Fig. 1). ECG revealed sinus tachycardia. Urine toxicology returned normal.

In view of severe metabolic acidosis and increased work of breathing, the patient was intubated, mechanically ventilated and managed with other supportive treatment. The patient was transferred to the Intensive Care Unit and supportive care continued. The patient showed progressive improvement with normalization of CoHb within 6 hours. Blood sugars remained stable during the stay. He was extubated twelve hours later and discharged with no neurological deficit after thirty-six hours of ED arrival.

DISCUSSION

Carbon Monoxide is one of the leading causes of accidental poisoning. Sources of CO poisoning could be house fires, faulty furnaces, gas water heaters, wood burning stoves, motor vehicle exhaust and various propane fuel equipment [1]. The binding affinity of CO for hemoglobin is over 200 times greater than that of oxygen for hemoglobin. Formation of carboxyhemoglobin (COHb) decreases the oxygen-carrying capacity of blood and thus impairs the release of oxygen from hemoglobin for its utilization by tissues. Carboxyhemoglobin decreases the unloading of oxygen at the tissue level, as a result, hypoxic state develops which continuously utilizes glucose and produces elevated lactate levels.

Initially, during carbon monoxide exposure, increased catecholamine release occurs due to stress response which causes hepatic glycogenolysis and gluconeogenesis, resulting in hyperglycemia. Hyperglycemia ultimately results in increased plasma insulin secretion. Hyperinsulinemia and higher metabolic rate then tend to develop hypoglycemia. Hypoglycemia-induced neuro-cognitive deficit is probably due to the result of neuronal cell starvation secondary to hyperlactatemia and worsening acidosis [3]. To the best of our knowledge, hypoglycemia in a healthy, non-diabetic adult patient with carbon monoxide poisoning has not been reported in the literature before.

The symptoms of CO poisoning are nonspecific, frequently leading to misdiagnosis. Carbon monoxide can affect multiple organ systems, but the central nervous system and cardiovascular systems are the most severely affected due to their high oxygen requirements [4]. Longer exposures and higher concentrations of CO typically result in more severe symptoms, although there are no guidelines predicting outcomes based on duration and exposure. Symptoms generally do not occur until COHb concentration exceeds 10%. As the blood concentration increases above 20%; headache, nausea, dizziness and confusion may develop. COHb concentrations above 60% are often fatal. However, one should not rely on COHb concentrations to predict injury or outcome.

Metabolic acidosis is a more reliable prognostic indicator of serious sequelae than COHb concentration [5]. Death from acute CO poisoning is usually due to ventricular dysrhythmias secondary to hypoxia. Neurologic injury following acute carbon monoxide poisoning may be delayed for several weeks to months after exposure. Upto 10% of carbon monoxide poisoning survivors have a persistent gross neuro-psychiatric impairment and a much larger percentage suffers more subtle effects such as personality changes and memory impairment [6].

Carbon monoxide poisoning is an often overlooked diagnosis because not only the symptoms and signs are non-specific but also the initial investigations can be misleading. Despite decreased oxygen carrying capacity of CO-poisoned patients, pulse oximetry and arterial blood gases will fail to reveal CO-induced hypoxia because most pulse oximeters cannot differentiate COHb from oxyhemoglobin. The definitive diagnosis of carbon monoxide poisoning can be made by measuring elevated levels of carboxyhemoglobin [4].

Supplemental oxygen is the most important treatment for CO poisoning. High flow oxygen with a non-rebreathing oxygen face mask or endotracheal tube should be immediately provided to patients with a strong history or symptoms suggestive of carbon monoxide exposure [7]. Cardiac monitor and intravenous access should be immediately instituted in patients with severe poisoning. Hypotension can be treated with intravenous fluids and myocardial depression is treated with inotropic agents. Advanced Cardiac Life Support protocols should be followed for life-threatening dysrhythmias. Fingerstick glucose test is essential to rule out hypoglycemia in patients of altered mental status. Occasional seizure episode may require benzodiazepine administration. The use of hyperbaric oxygen is controversial but it is reserved for management of severe carbon monoxide poisoning [8].

CONCLUSION

Hypoglycemia is associated with worsening neurological deficit and increased mortality in carbon monoxide poisoning. Its early detection and immediate management in the emergency department can make the difference in survival. A proper history is key for early diagnosis. High flow oxygen is the mainstay of treatment for carbon monoxide poisoning and should be immediately started. Persistent hypoglycemia in such patients can help in predicting the outcome. Larger studies are required to validate this association.

REFERENCES

1. Mehta SR, Das S, Singh SK; Carbon Monoxide Poisoning; Med J Armed Forces India. 2007;63:362-5.
2. Rose JJ, Wang L, Xu Q, McKiernan CF, Shiva S, Tejero J, *et al.* Carbon Monoxide Poisoning: Pathogenesis, Management and Future directions of therapy. Am J Respir Crit Care Med. 2017;195:596-606
3. Penny DG, Helfman CC, Dunbar JC Jr, McCoy LE. Acute severe carbon monoxide exposure in the rat: effects of hyperglycemia and hypoglycemia on mortality, recovery, and neurologic deficit. Can J Physiol Pharmacol. 1991;69:1168-77.
4. Rucker J, Fisher J; Carbon Monoxide Poisoning. Chapter. 63:679-83
5. Tomaszewsi C. Carbon monoxide. In: Goldfrank's Toxocologic Emergencies. 7th ed. Goldfrank LR, Flomenbaum NE, Lewin NA, *et al*., eds. New York: McGraw-Hill. 1998:1478-91.
6. Varon J, Marik PE, Fromm RE Jr, Gueler A. Carbon monoxide poisoning: A review for clinicians. J Emerg Med. 1999;17:87-93.
7. Weaver LK. Hyperbaric oxygen in carbon monoxide poisoning: Conflicting evidence that it works. BMJ. 1999;319:1083-4.
8. Chou KJ, Fisher JL, Silver EJ. Characteristics and outcome of children with carbon monoxide poisoning with and without smoke exposure referred for hyperbaric oxygen therapy. Pediatric Emerg Care. 2000;16:151-5

Funding: None; Conflict of Interest: None Stated.

How to cite this article: Arora A, Desai K, Jain S, Mehta M. Hypoglycemia in carbon monoxide poisoning: a case report. Indian J Case Reports. 2019;5(4):357-359.

Doi: 10.32677/IJCR.2019.v05.i04.021