A CASE OF MYOCARDIAL INJURY IN AN INFANT WITH CARBON MONOXIDE POISONING

Wong KCS[1], Ismail AK[1], and Al Kuddoos NM[1].
[1]Department of Emergency Medicine, Faculty of Medicine, Universiti Kebangsaan Malaysia, Jalan Yaacob Latiff, Bandar Tun Razak, 56000 Cheras, Kuala Lumpur, Malaysia

Correspondence:
Ahmad Khaldun Ismail,
Department of Emergency Medicine,
Faculty of Medicine,
Universiti Kebangsaan Malaysia,
Jalan Yaacob Latiff, Bandar Tun Razak,
56000 Cheras, Kuala Lumpur, Malaysia
Email: khaldun_ismail@yahoo.com

Abstract

Carbon monoxide poisoning is described as "silent killer" because carbon monoxide is an odourless, colourless, tasteless gas and the clinical presentation is non-specific. A two-month-old boy presented with rapid breathing, irritable, and refused to be fed. The symptoms occurred approximately 1 h after waiting with his father in the car with the engine running. The father also presented with symptom of carbon monoxide poisoning. The child had respiratory distress and sinus tachycardia. The child's carboxyhaemoglobin level was normal, but the father's level was elevated. Serum lactate and troponin I were raised. He was given 100% normobaric oxygen and was admitted. Subsequently his condition improved and was discharged the next day. Infants are more prone to the effects of carbon monoxide poisoning. When an infant suddenly become unwell, high index of suspicion and detail collaborative history are required so that carbon monoxide poisoning will not be missed out.

Keywords: Carbon Monoxide, Clinical Toxicology, Emergency, Paediatric

Introduction

Carbon monoxide (CO) is an odourless, colourless, and tasteless gas. It is generated through the incomplete combustion of hydrocarbon compound. It is usually present in air at 10 parts per million (1). The common sources of CO emission are from fire, motor-vehicle engine exhausts, and faulty heating devices (1-3). CO poisoning is often described as "silent killer" because of the nature of CO and the non-specific clinical symptoms of CO poisoning despite causing tissue hypoxia and myocardial injury. CO causes tissue hypoxia by binding to the haem of haemoglobin with the affinity of 250-fold greater than that of oxygen, forming carboxyhaemoglobin (COHb) which reduces the oxygen carrying capacity of haemoglobin (3). Tissue hypoxia will lead to lactic acidosis especially in organs with high metabolic demand such as brain and heart. CO also triggers inflammatory cascade, which leads to neurological and myocardial injuries (3).

The most common presenting signs and symptoms in children with CO poisoning are headache, nausea, vomiting, and change of consciousness, mood disorders, personality changes, memory loss to focal neurological injuries, and severely disabling manifestations of hypoxic brain injury (4, 5). CO poisoning in infants may manifest as irritability, inconsolable cry and feeding difficulty (1, 6). Neurological examination will be variable and challenging in infant. The classical pathognomonic finding of cherry red oral mucosa and skin in CO poisoning is only seen post mortem, hence, it does not have diagnostic value (7). The vague and non-specific presentation in infants may lead to misdiagnosis of this condition as food poisoning or gastroenteritis in infants. Pulse oximetry is not helpful in detecting CO poisoning, because the conventional two-wavelength pulse oximetre cannot distinguish COHb and oxyhaemoglobin. COHb level need to be measured by co-oximetry of an arterial or venous blood gas sample. Normal COHb level in non-smoking adult is less than 3%, while in smoker can be up to 10–15% (2). COHb level in infant is also higher, up to 3–7% (7). Myocardial injury due to CO poisoning may leads to changes on ECG, such as sinus tachycardia, ST-segment changes, and arrhythmias (7, 8).

Case Report

A two-month-old infant boy was brought in by the mother with a sudden onset of change of behaviour for 2 h. The mother went for shopping, while the father was waiting inside the car with the child for approximately 1 h. The car was parked under the hot sun with the engine and

air-conditioner running with closed windows. When the mother came back, she noticed the child had rapid breathing, irritable, and refused to be fed, while the father was lethargic and complaining of headache. The mother brought both the child and father to the emergency department for medical attention. Further history from the parents noted that the car was not sent to service for one year, which suggested the possibility of the malfunction of the car's exhaust system and can lead to the leakage of CO into the vehicle's cabin.

On examination in the resuscitation room, the child was lethargic, tachycardiac and normotensive with initial heart rate of 170-180 bpm, blood pressure of 77/49 mmHg, oxygen saturation of 99%, and temperature of 36.9 ºC. His anterior fontanelle was sunken, skin was mottling, and the capillary refill time was about three seconds. He was in respiratory distress with the respiratory rate of 50 breathes per min and subcostal recession despite able to maintain his oxygen saturation under room air. Initial venous blood gas showed respiratory alkalosis, with pH 7.537, $PaCO_2$ 3.29 kPa, bicarbonate 23 mmol/L, base excess -1.9, and serum lactate 2.3 mmol/L. In view of the history of prolonged stay in motor-vehicle and the father has headache, CO poisoning was suspected. The COHb level of the baby at 4-h post-exposure was 2.5%. Supplemental oxygen was given at 15 L/min via non-rebreather mask (NRM) and intravenous (IV) drip of 1/5 normal saline dextrose 10% was initiated at the rate of 120 ml/kg/day. The baby was admitted to paediatric high dependency unit (PHDU).

In PHDU, the COHb level of the child at 7-h post-exposure dropped down to 0.2%, and serum lactate reduced to 1.1 mmol/L. A troponin I level was taken in view of persistent sinus tachycardia of 130 bpm, and the level was 1078.1 pg/ml (normal range <34.2 pg/ml). However, electrocardiography (ECG) of the child showed sinus tachycardia without ST-segment deviation (Figure 1). On the second day, the child's condition improved and tolerated breast feeding well. Oxygen support was weaned off and IV drip was stopped. Repeated troponin I level decreased to 349 pg/ml. The child was discharged and given follow-up at the nearest clinic in two weeks' time to review the child's general condition.

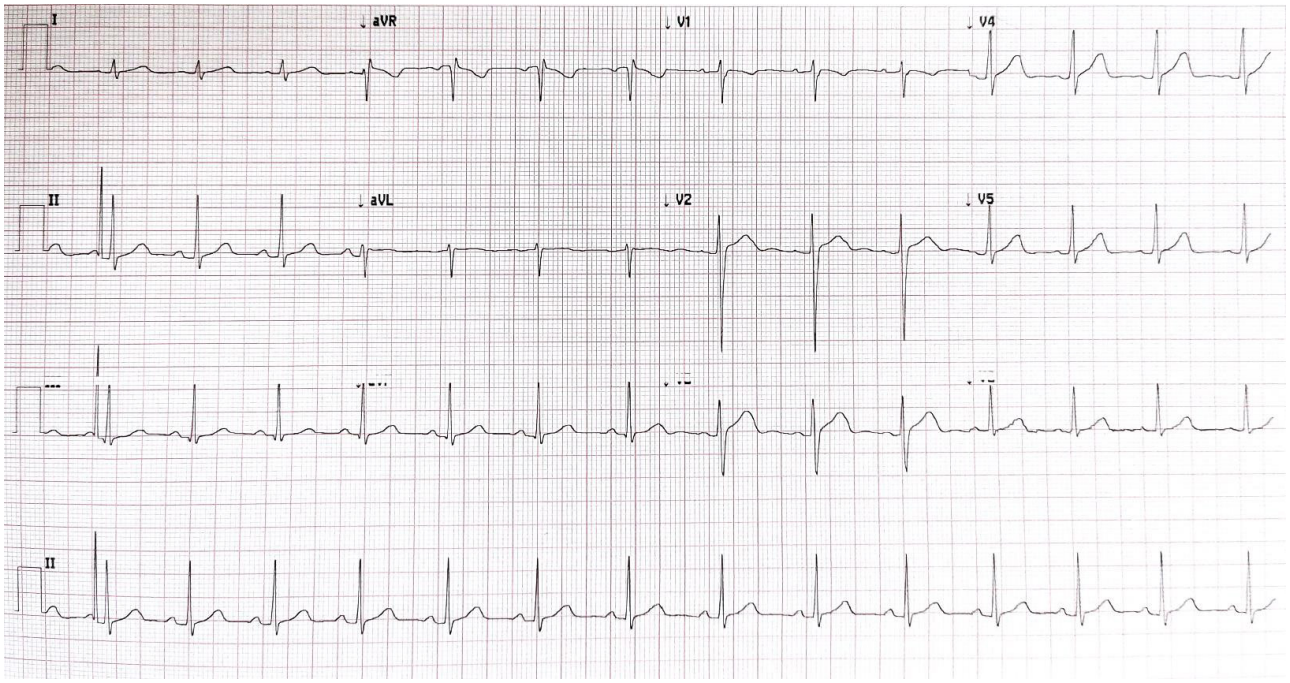

Figure 1: Electrocardiography of the patient showed sinus tachycardia

Discussion

The diagnosis of CO poisoning is usually established by this clinical triad: (a) symptoms consistent with CO poisoning; (b) history of CO exposure; and (c) raised COHb levels (3). However, in infants, the diagnosis of CO poisoning is more challenging as they cannot verbalise their complaints. Although the COHb level of this patient upon arrival is 2.5% which is in the normal range for his age, the history of prolonged stay in a motor-vehicle and the collaborative history of his father are pointing toward the diagnosis of CO poisoning.

The father of the patient, who had the same exposure with the patient, was complaining of headache upon arrival to the emergency department. The physical examination of the father was unremarkable. However, the father's COHb level was elevated at 20%, which confirmed the diagnosis of CO poisoning. The father was given high flow oxygen and was admitted to the observation unit of the emergency department.

Infant and children are more susceptible to the effects of CO poisoning because affinity of CO is higher for fetal

haemoglobin than for adult haemoglobin. The fetal COHb half-life is also longer than adult COHb. Paediatric population has high metabolic rate, heart rate, minute ventilation, and immature central nervous systems, hence, they might develop clinical symptoms of CO poisoning earlier than the adults who experienced the same exposure (2, 9). If the history that suggests of CO exposure is not obtained, the clinician might attribute the symptoms to other diseases such as infection and miss the diagnosis of CO poisoning. In this case, the history of prolonged stay in a motor-vehicle and the symptom of headache in the patient's father had raised the suspicion of CO poisoning.

The COHb level in this patient was significantly lower than his father's COHb level despite exposed to the CO in same environment and same duration. However, the serum lactate level was elevated which indicates the presence of tissue hypoxia. The child had severe CO poisoning in view of elevated troponin I level, which indicates myocardial injury. Based on the COHb level, the clinical signs and symptoms, the severity of CO poisoning is divided into mild (COHb level <10% without clinical signs or symptoms), moderate (COHb level 10–25% with minor clinical signs and symptoms), and severe (COHb level >25% with loss of consciousness and confusion or signs of cardiac ischemia) (10). The COHb level alone does not truly reflect the severity of the symptoms of CO poisoning in children (5). There is a positive correlation between increasing age and higher COHb levels, whereby younger children have higher minute ventilation leading to faster clearance of CO from the body (5). However, this does not imply that the damage from CO is lesser in younger children. Younger patients can develop a severe insult from CO poisoning with misleadingly low COHb level (5). In the case of suspected CO poisoning in children with COHb level within normal limits, serum lactate level should be measured because it serves as better indicator of tissue hypoxia, but a lower level does not exclude the diagnosis of CO poisoning (7).

In this patient, persistent tachycardia raised the suspicion of myocardial injury. Patient's ECG showed sinus tachycardia only, but the troponin I level is elevated. In the case series of CO poisoning in young adults reported by Aslan *et al*, ECG was normal in 48.2% of the cases, with 92.8% has normal echocardiogram finding (8). Teksam *et al.* also reported that 16 (15%) out of 107 children with CO poisoning had myocardial injury confirmed with cardiac biomarkers (CKMB and troponin T), but none of them had ischemic changes on ECG (11). CO poisoning may cause myocardial injury in children despite the absence of abnormal ECG or severe clinical signs (8, 11). Hence, it is important to measure the cardiac biomarker such as troponin level in CO poisoning patient even if the ECG finding is normal.

The half-life of CO while patient is breathing room air at normal atmospheric pressure is around 250 to 320 min, while breathing 100% normobaric oxygen (NBO_2) via NRM is around 74 to 90 min (2, 3). The half-life of CO will further reduce to approximately 20 to 30 min with hyperbaric oxygen (HBO_2) at 2.5–3 atmospheres (2, 3). Hence, the mainstay of treatment for CO poisoning is either NBO_2 or HBO_2. HBO_2 is indicated for severe CO poisoning (1). Although this patient had evidence of myocardial injury, he did not require HBO_2 due to rapid improvement in condition with NBO_2. The COHb level rapidly declined after receiving 100% NBO_2 via NRM and the child was able to feed well. The troponin I level reduced significantly within 24 hrs.

Faulty motor-vehicle engine exhausts is one of the common sources of CO emission (1-3). In this case it was not confirmed whether there was leakage of gases from exhaust into the car's cabin. It is advisable that all motor-vehicles to be sent to workshop for regular service to detect malfunction of car exhaust system and check for leak of gases such as CO, hydrocarbons, nitrous oxide and carbon dioxide. The public should be educated not to rest or sleep in the car with the engine and air-conditioner running.

Conclusion

The clinical presentation of CO poisoning is non-specific and the diagnosis will be more challenging in infants who cannot verbalize their complaints. When an infant suddenly become unwell, high index of suspicion and detail collaborative history are required so that CO poisoning will not be missed out. A normal COHb level especially in paediatric population may not be sufficient to determine the severity of CO poisoning. Hence, serum lactate and cardiac biomarkers level should be measured to look for the extend of tissue hypoxia and evidence of myocardial injury.

Competing interests

The authors declare that they have no competing interests.

Ethical Clearance

Informed consent to use this case report for publication had been obtained from the patient's guardian.

References

1. Maloney GE. Carbon Monoxide. In: Tintinalli JE, ed. Tintinalli's Emergency Medicine: A comprehensive study guide. 9th Ed. New Yoke, USA: The Mc Graw-Hill. 2020:1414-7.
2. Clardy PF, Manaker S, Perry H. Carbon monoxide poisoning. On UpToDate. 2019. Available at: https://www.uptodate.com/contents/carbon-monoxide-poisoning. Assessed 17 August 2020.
3. Rose JJ, Wang L, Xu Q, McTiernan 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(5):596-606.
4. Chang YC, Lee HY, Huang JL, Chiu CH, Chen CL, Wu CT. Risk factors and outcome analysis in children with carbon monoxide poisoning. Pediatr Neonatol. 2017;58(2):171-7.
5. Sethuraman KN, Douglas TM, Bostick BB, Comer AC, Myers B, Rosenthal RE. Clinical characteristics of

pediatric patients with carbon monoxide poisoning. Pediatr Emerg Care. 2020;36(4):178-81.
6. Rudge FW. Carbon monoxide poisoning in infants: treatment with hyperbaric oxygen. South Med J. 1993;86(3):334-7.
7. Gozubuyuk AA, Dag H, Kacar A, Karakurt Y, Arica V. Epidemiology, pathophysiology, clinical evaluation, and treatment of carbon monoxide poisoning in child, infant, and fetus. North Clin Istanb. 2017;4(1):100-7.
8. Aslan S, Uzkeser M, Seven B, Gundogdu F, Acemoglu H, Aksakal E, *et al*. The evaluation of myocardial damage in 83 young adults with carbon monoxide poisoning in the East Anatolia region in Turkey. Hum Exp Toxicol. 2006;25(8):439-46.
9. Akcan Yildiz L, Gultekingil A, Kesici S, Bayrakci B, Teksam O. Predictors of severe clinical course in children with carbon monoxide poisoning. Pediatr Emerg Care. 2021;37(6):308-11.
10. Fruchtman Y, Perry ZH, Leibson T, Vered Cohen L, Lifshitz M, Leibovitz E. Pediatric carbon monoxide poisoning in Southern Israel: A cross-sectional study. Pediatr Emerg Care. 2020;36(11):532-6.
11. Teksam O, Gumus P, Bayrakci B, Erdogan I, Kale G. Acute cardiac effects of carbon monoxide poisoning in children. Eur J Emerg Med. 2010;17(4):192-6.