ASIA PACIFIC JOURNAL of MEDICAL TOXICOLOGY
APJMT 13:4 http://apjmt.mums.ac.ir December 2024

CASE REPORT

Carbon Monoxide Poisoning with Neurological, Ocular, and Myocardial Damage: A Case Report

DANG DUC NGUYEN[1,*], PHUONG SINH NGUYEN[2], NGUYEN HONG ANH LAM[2]

[1]Poison Control Center of Bach Mai Hospital, Ha Noi, Vietnam
[2]Thai Nguyen University of Medicine and Pharmacy, Thai Nguyen, Vietnam

Abstract

Introduction: Carbon monoxide (CO) gas is produced by incomplete combustion of carbon-containing substances. CO has a high affinity with hemoglobin because it reduces oxygen transport causing tissue hypoxia, lactic acidosis, and cell death.
Case report: A 32-year-old female patient entered Poison Control Center of Bach Mai Hospital, Ha Noi, Vietnam, because of decreased consciousness, loss of vision in the right eye. Three people sat in a car, the patient sat in the back position, the patient's husband drove, patient's sister sat in the front. They went from Hanoi to Phu Tho province about 50 kilometers with 1.5 hours. 20 minutes after leaving Hanoi, the patient felt headache, dizziness, nausea. Everybody rested in Phu Tho for 45 minutes after returning to Hanoi together, the car ran about 10 minutes, patient felt headache, dizziness, a lot of nausea. The patient's husband felt a terrible headache. Hence, he parked the car on the side of the road and turned off engine but still closed window, all three people lost consciousness one hour later, and they were brought to hospital, at the hospital the right eye of the patient only can see fingers count 1.5 meters, left eye is normal.
Discussion: Our hospital didn't use hyperbaric oxygen therapy so patients could only breathe 100% oxygen through nose. The patient was infused with Solumedrol solution in a high dose. Patient's HbCO test was 3.8 % after treatment decreasing 1.4%, cardiac enzymes gradually decreased from 78.6 to 15.9 ng/L.
Conclusion: After 3 days of monitoring, treatment, the patient recovered her vision and was discharged.

Keywords: Increased HbCO, Metabolic acidosis, High-dose Corticoid Therapy

How to cite this article: Nguyen DC, Nguyen PS, Lam AHN. Carbon Monoxide Poisoning with Neurological, Ocular, and Myocardial Damage: A Case Report. *Asia Pac J Med Toxicol* 2024; 13(4): 167- 170.

INTRODUCTION

The CO gas is a highly toxic, colorless, odorless, tasteless, non-irritating gas. It is often produced in fires, incomplete combustion of coal, and internal combustion engines using gasoline or oil operating in closed rooms or poorly ventilated places. CO has a high affinity with hemoglobin because Hemoglobin reduces oxygen transport causing tissue hypoxia, lactic acidosis, and cell death [1,2]. CO binds with myoglobin, especially in the heart muscle, causing myocardial embolism and decreased cardiac output. CO inhibits cytochrome oxidase and leads to the formation of inflammatory substances and free radicals that will cause cell death and late nerve damage, especially in the brain. Pediatrics are particularly sensitive to CO toxicity [3]. The half-life of CO is 80 minutes when breathing room air and the half-life is 23 minutes under high-pressure oxygen [4,5]. In this report, we presented a case of a 37-year-old female patient who was admitted to our poison control center due to CO poisoning, the patient was brought to our hospital in a condition, she decreased consciousness, chest tightness, shortness of breath and suddenly decreased in vision in the right eye, arterial blood gas of patient had metabolic acidosis and HbCO was 3.8%.

CASE PRESENTATION

A 37- year old female patient with a healthy history was admitted to Poison Control Center of Bach Mai Hospital, Ha Noi, Vietnam with clinical symptoms such as headache, chest tightness, shortness of breath, and blurred vision in the right eye. Earlier that day, around 11:00 a.m. the patient, husband, and older sister returned to Ha Noi from Viet Tri City, Phu Tho province in the old car. She had the above symptoms about 10 minutes after leaving Viet Tri City.

When she came to our department, her clinical and laboratory tests had the following signs:

- **Vital signs:** Pulse 120 bpm, blood pressure 122/70 mmHg, respiratory rate 23/min, temperature 37.3°C.
- **Neurological status:** Slow to awaken, Glasgow Coma Scale (GCS) 14 points, pupils equal (3 mm), reactive to light, no neck stiffness or meningeal signs.
- **Ophthalmic findings (Figure 1):**
- Right eye vision: finger count at 1.5 meters.
- Left eye vision: normal
- Intraocular pressure: 19 mmHg bilaterally
- Fundoscopy: corneal edema and mild optic disc edema bilaterally

*Correspondence to: Dr. Dang Duc Nguyen, Poison Control Center of Bach Mai Hospital, Ha Noi, Viet Nam.
Email: dangducnguyen79@gmail.com, Tel: +84973221357.

- Ultrasound of the eye: Normal bilaterally
- **Laboratory results (table 1):**
- Arterial blood gas: pH 7.41, PCO2 29 mmHg, PO2 218 mmHg, HCO_3^- 18.4 mmol/L, base excess -6.2 mmol/L, lactate 0.9 mmol/L
- HbCO 3.8%, MetHb 0.7%
- Troponin T hs: 78.6 ng/L
- Hepatic and renal function: normal
- Cranial MRI showed no abnormalities
- Echocardiogram revealed normal left ventricular size and function

The patient was infused with Solumedrol 500mg/day to reduce optic disc edema and breathed oxygen 100/% via the nose. After that her symptoms gradually improved as below (Figure 2):

- Reduced blurred vision
- Right eye vision was 20/25, Left eye vision was 20/20
- The fundus of the eye was not damaged
- HbCO reduced to 1.4%
- MetHb reduced to 0.6%

Troponin T hs decreased to 51.44ng/L in the second sample and 15.9ng/L in the third sample.

The patient was discharged after 3 days of monitoring and treatment in stable condition.

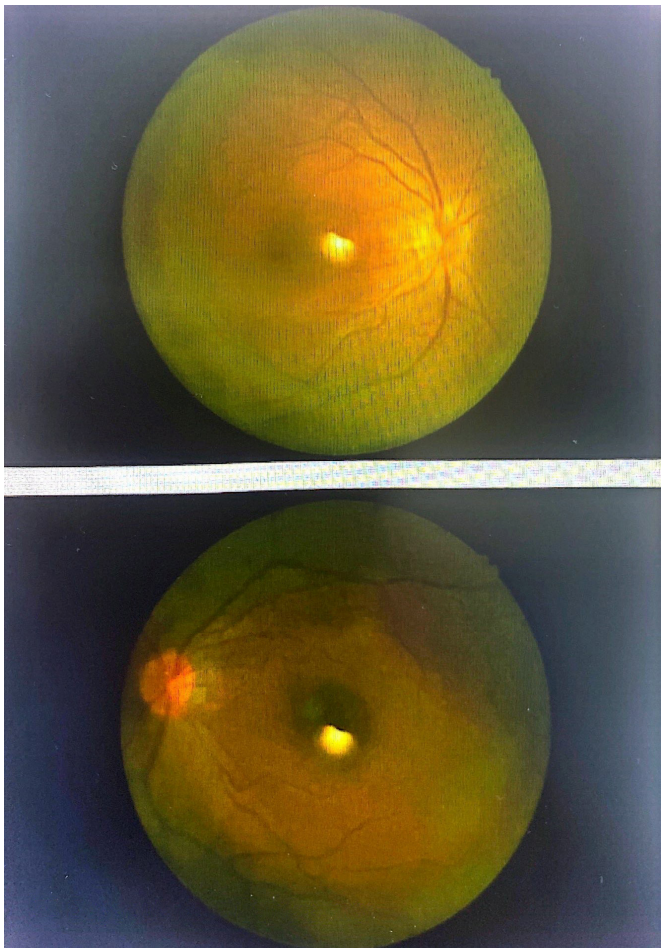

Figure 1. Non-fluorescent fundus scan of the patient

Table 1. Laboratory tests results of the patient at the beginning of entering the hospital

Time Index	Day of admission to hospital	After 3 days of treatment
BE	- 6.2 mml/L	-1.1 mmol/L
HCO_3^-	18.4 mml/L	22.4 mml/L
HbCO	3.8 %	1.4 %
MetHb	0.7 %	0.6 %
Troponin T hs	78.6 ng/L	15.9 ng/L

DISCUSSION

Carbon monoxide (CO) is produced by incomplete combustion of carbon-containing substances. CO has a high affinity with hemoglobin, reducing oxygen transport and causing tissue hypoxia, lactic acidosis, and cell death. CO binds with myoglobin, particularly in heart muscle, leading to myocardial ischemia and decreased cardiac output. Additionally, CO inhibits cytochrome oxidase forming free radicals and anti-inflammatory agents, causing cell death and delayed neurological damage, especially in the brain. The fetus is susceptible to CO toxicity [6]. CO binds to hemoglobin more strongly than oxygen, forming carboxyhemoglobin (COHb), impairing oxygen transport and use. CO can trigger inflammatory cascades, leading to lipid peroxidation in the central nervous system (CNS) and long-term neurological complications [6].

The clinical symptoms of CO poisoning are typically non-specific, affecting organs sensitive to hypoxia such as the brain, heart, and eyes. Common symptoms include headache (58%), nausea (33%), and dizziness (29%) [7]. Examination findings often reveal altered mental status, ranging from mild confusion to coma, along with tachycardia and rapid breathing. Severe cases may present with myocardial ischemia, ventricular arrhythmias, and pulmonary edema. Eye symptoms, such as blurred vision, light sensitivity, and double vision, are uncommon but can have serious consequences, especially if eye lesions are treated late [8]. There are 5 mechanisms leading to damage to the optic nerve (reduces oxygen delivery to tissues including the optic nerve, contributes to cellular apoptosis (programmed cell death) in optic nerve tissues, triggers an inflammatory cascade releasing cytokines that exacerbate damage to optic nerve tissues, manifest as optic neuritis (inflammation of the optic nerve) or ischemic optic neuropathy, lead to delayed neurological effects including optic nerve damage) [8]. The diagnosis of CO poisoning relies on medical history, exposure risk factors, and elevated HbCO levels (>3% in non-smokers, >10% in smokers). If higher HbCO levels (>15-20%) are associated with more severe symptoms, and levels >50% can be life-threatening) [8]. Elevated HbCO levels confirm CO exposure [9,10]. In patients with CO poisoning, tests should be conducted to evaluate the effects on various organs, including ECG, cardiac enzymes, echocardiography, blood gas analysis, chest X-ray, blood counts, liver and kidney function, and a

Người lấy mẫu: Tình trạng mẫu: Đạt

TG tiếp nhận chỉ định: 11:25 12/06/2024 TG nhận mẫu tại labo:

STT	Yêu cầu xét nghiệm	Kết quả	Đơn vị	Khoảng tham chiếu	Máy XN/PPXN
	Khí máu				
1	Xét nghiệm Khí máu (*)				
	pH	7.449		7.35 - 7.45	Cobas B221 (3)
	pCO2	33.0	mmHg	35 - 45	Cobas B221 (3)
	pO2	118.0	mmHg	70 - 99	Cobas B221 (3)
	HCO3	22.4	mmol/L	21.0 - 29.5	Cobas B221 (3)
	HCO3 chuẩn	23.4	mmol/L	25	Cobas B221 (3)
	BB	44.9	mmol/L	45 - 52	Cobas B221 (3)
	BE	-1.1	mmol/L	± 2.0	Cobas B221 (3)
	SatO2	98.8	%	70 - 90	Cobas B221 (3)
	COHb	1.4	%	0.5 - 2.5	Cobas B221 (3)
	MetHb	0.6	%	0.4 - 1.5	Cobas B221 (3)
	tHb	10.26	g/dL	11.5 - 17.4	Cobas B221 (3)

Figure 2. Arterial blood gas results after 3 days of monitoring and treatment

BS chỉ định: PHAN THỊ LAN HƯƠNG

Người lấy mẫu: Tình trạng mẫu: Đạt

TG tiếp nhận chỉ định: 15:47 12/06/2024 TG nhận mẫu tại labo: 15:57 12/06/2024

STT	Yêu cầu xét nghiệm	Kết quả	Đơn vị	Khoảng tham chiếu	Máy XN/PPXN
	Miễn dịch				
1	Định lượng Troponin T hs (*)	15.9	ng/L	=< 14	Cobas8K2

Figure 3. Troponin T hs **results after 3 days of monitoring and treatment**

systematic eye examination.

Our patient's cardiac enzymes showed a slight increase, which later normalized. Management focuses on removing CO sources and ensuring airway, breathing, and circulation (ABCs) in severe cases. Treatment includes high-flow oxygen (100%) for suspected or confirmed CO poisoning, and hyperbaric oxygen therapy should be considered within the first 24 hours for patients with severe symptoms [11].

CONCLUSION

In our patients, the symptoms are non-specific. The diagnosis of a patient with CO poisoning is determined through questioning, determining risk factors for exposure to CO, and testing for HbCO concentration, and MetHb concentration. CO poisoning can cause optic nerve, neurological, and myocardial damage. These lesions can be permanent or temporary. In more severe cases, it can lead to death.

Ethical approval: All procedures were conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants prior to their inclusion in the study.

Funding and Support: None
Conflict of Interest: None

REFERENCES

1. Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med, 1998; 339:1603.
2. Hardy KR, Thom SR. Pathophysiology and treatment of carbon monoxide poisoning. J Toxicol Clin Toxicol, 1994; 32:613.

3. Peabody T, Furr A, Ditmetaroj N. Carbon Monoxide and the Eye: A Teaching Case Report. Journal opted, 2013; 38(3).
4. Satran D, Henry CR, Adkinson C, Nicholson CI, Yiscah B, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. J Am Coll Cardiol, 2005; 45:1513-6.
5. Touger M, Gallagher EJ, Tyrell J. Relationship between venous and arterial carboxyhemoglobin levels in patients with suspected carbon monoxide poisoning. Ann Emerg Med 1995; 25:481.
6. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Carbon Monoxide Poisoning; Wolf SJ, Maloney GE, Shih RD, Shy BD, Brown MD. Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Acute Carbon Monoxide Poisoning. Ann Emerg Med 2017; 69(1):98-107.e6.
7. Leach RM, Rees PJ, Wilmshurst P. Hyperbaric oxygen therapy. BMJ 1998; 317:1140.
8. Claudio Bucolo, Filippo Drago. Carbon monoxide and the eye: Implications for glaucoma therapy. Pharmacology & Therapeutics, 2011; 130(2): 191-201.
9. Quinn DK, McGahee SHM, Politte LC, Duncan GN, Cusin C, Hopwood CJ, et al. Complications of Carbon Monoxide Poisoning: A Case Discussion and Review of the Literature. Prim Care Companion J Clin Psychiatry, 2009;11(2):74–79.
10. Gandini C, Castoldi AF, Candura SM, Priori S, Locatelli C, Butera R, et al. Cardiac Damage in Pediatric Carbon Monoxide Poisoning. J Toxicol Clin Toxicol, 2001; 39(1):45-51.
11. Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002; 347(14):1057-67.