

Asymptomatic Myocardial Injury in a Low Level of Carbon Monoxide Poisoning

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Abstract

Carbon monoxide (CO) poisoning is an important cause of mortality and morbidity. Although measured in CO levels are not always correlated with clinical symptoms, neurological symptoms may present in lower CO levels, and cardiac signs and symptoms may occur in high CO levels. Low levels of CO exposure are very rare causes of myocardial injury. In this case presentation, we aimed to report on a patient who had a 20% level of CO and high troponin level without cardiac signs and symptoms. A 26-year-old male presented to the emergency department with headache, nausea, and vomiting. The initial electrocardiogram showed ST-segment depression of 1 mm in Lead II(II)-Lead III(III) - Lead augmented vector foot (aVF) and ST-segment elevation in Lead I(I)- Lead augmented vector left (aVL). The patient's laboratory values were as follows: troponin I: 1.5 ng/mL and FCOHb: 20.7%. The first echocardiogram of the patient demonstrated global hypokinesia of the left ventricle. The coronary angiogram of the patient was normal. All patients considered to have CO poisoning should be evaluated with electrocardiograms, cardiac necrosis marker measurements, and an echocardiogram for myocardial injury regardless of the level of CO or the absence of cardiac symptoms and signs. (*JAEM 2015; 14: 91-3*)

Keywords: Carbon monoxide poisoning, myocardial injury, carboxyhemoglobin level

Introduction

Carbon monoxide (CO) poisoning is an important cause of mortality and morbidity. CO toxicity appears to result from a combination of tissue hypoxia and direct damage at the cellular level (1).

Carbon monoxide poisoning can lead to a variety of symptoms such as flu, headache, dizziness, weakness, nausea, confusion, coma, or even death (2).

Although measured in CO levels are not always correlated with clinical, neurological symptoms may present in lower CO levels, and cardiac signs and symptoms may occur in high CO levels. Headache is often the first symptom to appear at blood levels of approximately 10% of CO. Dizziness, dyspnea, and nausea may develop at blood levels of 20-30% of CO. Seizures, coma, cardiopulmonary dysfunction, and deaths may also appear at blood levels of 50-70% of CO (Table 1). Exposure to low levels of CO is a very rare cause of myocardial injury (1, 3-5).

We aimed to offer a patient who had a 20% level of CO and high troponin level without cardiac signs and symptoms.

Case Presentation

A 26-year-old male, who lives in a stove heating-system house in the winter, presented to the emergency department with headache,

nausea, and vomiting at 09:00 AM. He did not have any cardiac signs and symptoms such as chest pain, palpitation, and dyspnea on exertional presyncope. The exposure period of our patient to CO was not clearly known. There was no disease and no smoking in his medical history. The patient's general condition was good. He was conscious, oriented, and cooperative. The patient's vital signs were as follows: body temperature of 36.7°C, blood pressure of 130/80 mmHg, heart rate of 82 beats/min, and rhythmic respiratory rate of 14 breaths/min, and his physical examination signs were all normal.

The initial electrocardiogram showed ST-segment depression of 1 mm in II-III-aVF and ST-segment elevation in I-aVL. ST-segment elevation in the anterior chest leads was due to early repolarization (Figure 1).

The patient's laboratory values were as follows: white blood cell count 15160/ μ L, hemoglobin 12.1 g/dL, pH 7.33, carbon dioxide partial pressure 44.8 mmHg, and carboxyhemoglobin (COHb) 20.7%. The cardiac necrosis marker levels were as follows: troponin I: 1.5 ng/mL (cutoff value=0.01), creatinine kinase-MB: 13.3 ng/mL, and myoglobin: 172.9 ng/mL.

High-flow (12-15 L/min) 100% oxygen was administered with a face mask to the patient who was considered to be exposed to CO poisoning. The first echocardiogram performed by cardiologists

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Table 1. Symptoms of acute CO poisoning based on COHb levels (5)

COHb%	Symptoms
10	Asymptomatic or may have headache
20	Dizziness, nausea, dyspnea
30	Visual disturbances
40	Confusion, syncope
50	Seizures and coma
≥60	Cardiopulmonary dysfunction and death
CO: carbon monoxide; COHb: carboxyhemoglobin	

demonstrated global hypokinesia of the left ventricle. The patient was transferred to the primary percutaneous coronary intervention center.

Although there were no wall motion abnormalities on the second echocardiogram, the patient was qualified for coronary angiography because of a marked elevation of cardiac necrosis markers and changes in electrocardiogram. The coronary angiogram was normal. The patient was monitored for 3 days in the hospital. He was discharged after treatment.

Discussion

Carbon monoxide is a toxic gas that consists of one carbon atom covalently bonded to one oxygen atom and is usually formed during the incomplete combustion of carbon-based compounds (4).

Sources of CO in patients with CO poisoning include motor vehicle exhaust smoke, fumes resulting from fires, stove, barbecue, gas water heater, and paint containing methylene chloride (6). CO poisoning is most commonly caused by stove smoke during the winter like in our case (7).

Carbon monoxide is known as a silent cause of death because of its lack of irritation, color, taste, and odor (8).

Carbon monoxide poisoning may be misdiagnosed because the clinical signs and symptoms of CO poisoning are nonspecific and may mimic a lot of diseases. Therefore, the true incidence of CO poisoning is not known exactly (4). In a study conducted in Turkey, the cause of deaths in 3.85% of autopsies was found to be CO poisoning (8).

The severity of poisoning is determined by environmental and personal factors such as exposure time and ambient concentration of CO, pulmonary ventilation frequency and capacity, and the presence of additional diseases (4). In a study conducted by Kalay et al. (9), in all patients who had high levels of cardiac markers, prolonged exposure to high levels ($37\pm 7\%$) of CO were found. The exposure period of our patient to CO was not known clearly, but the CO level of our patient was lower than that of Kalay et al. (9).

Although the measurement of COHb level is useful for diagnosis when detected, the COHb level does not always correlate with clinical manifestation (10).

Generally, neurological symptoms such as headache and dizziness occur in lower CO levels between 10 and 20%, but cardiac signs and symptoms occur in high levels of CO (1-3). In our case, myocardial injury occurred in a low level of measured CO (20%) in the blood test and without any cardiac symptoms.

Cardiovascular manifestations of CO poisoning include heart rate changes, arrhythmias, contractile dysfunction, myocardial injury

**Figure 1.** This figure shows the patient's electrocardiogram

ry ranging from mild and transitory injury to necrosis, cardiogenic shock, and sudden death (2, 4).

Although patients with an underlying cardiac disease are at a greater risk of myocardial injury and infarction, CO poisoning can trigger myocardial injury and infarction even in patients with normal coronary arteries (11).

Many mechanisms contribute to myocardial injury. These mechanisms are cellular hypoxia due to the high affinity of hemoglobin for CO, direct negative inotropic effect of CO independent of hemoglobin-binding ability, inhibition of myoglobin function due to hypoxia, oxidative stress, which could contribute to free radical overload in the heart, similar to ischemia reperfusion injury, and prothrombotic potential of CO, which can trigger arterial and venous thrombosis (10, 12).

Treatment of CO poisoning includes removal from the site of exposure, administration of supplemental oxygen, and general supportive care that consists airway maintenance, blood pressure support, and cardiovascular stabilization until the COHb level is normal ($\leq 3\%$) and the patient's presenting symptoms of CO poisoning have resolved, usually for approximately 6 h. The administration of 100% oxygen at atmospheric pressure (normobaric oxygen) helps in reducing damage from CO-induced hypoxia and in accelerating the elimination of CO from body (10). In our patient, after treatment with 100% O₂, the global hypokinesia seen in the first echocardiogram improved, and the level of cardiac enzymes decreased.

In a study conducted by Kalay et al. (9), in 6 of 20 patients exposed to CO, the level of cardiac enzymes was higher, but the angiograms were normal. Coronary angiography was performed in our patient, and the result was normal. Percutaneous coronary intervention should not be ignored in myocardial infarction caused by CO poisoning because CO can trigger arterial and venous thrombosis.

Conclusion

Myocardial injury in patients with CO poisoning are difficult to identify, especially in asymptomatic patients. All patients considered to have CO poisoning should be evaluated with electrocardiograms, cardiac necrosis marker measurements, and an echocardiogram for myocardial injury regardless of the level of CO or the absence of cardiac symptoms and signs.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

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References

1. Zahger D, Slutzky O. Severe reversible left ventricular dysfunction induced by carbon monoxide intoxication. *Am J Emerg Med* 2002; 20: 572-3. [\[CrossRef\]](#)
2. Sohn YH. Carbon monoxide poisoning. *Encyclopedia of Movement Disorders* 2010: 187-9. [\[CrossRef\]](#)
3. Lee SJ, Kang JH, Kim NY, Baek IW, Park MY, Shim BJ, et al. A case report of carbon monoxide poisoning induced cardiomyopathy complicated with left ventricular thrombus. *J Cardiovasc Ultrasound* 2011; 19: 83-6. [\[CrossRef\]](#)
4. Lippi G, Rastelli G, Meschi T, Borghi L, Cervellini G. Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbon monoxide poisoning. *Clin Biochem* 2012; 45: 1278-85. [\[CrossRef\]](#)
5. Varon J, Marik PE, Fromm Jr RE, Gueler A. Carbon monoxide poisoning: a review for clinicians. *JAEM* 1999; 17: 87-93. [\[CrossRef\]](#)
6. Omaye ST. Metabolic modulation of carbon monoxide toxicity. *Toxicology* 2002; 180: 139-50. [\[CrossRef\]](#)
7. Kandış H, Katırcı Y, Çakır Z, Aslan Ş, Uzkeser M, Bilir Ö. A Retrospective analyze of the patients admitted to emergency service for carbon monoxide intoxication. *JAEM* 2007; 6: 21-5.
8. Cantürk N, Başbulut AZ, Cantürk G, Dağalp R. Evaluation of the autopsy cases carbon monoxide poisonings in Ankara between 2002-2006. *J For Med* 2008; 22: 25-30.
9. Kalay N, Ozdogru I, Cetinkaya Y, Eryol NK, Dogan A, Gul I, et al. Cardiovascular effects of carbon monoxide poisoning. *JACC* 2007; 99: 322-4. [\[CrossRef\]](#)
10. Buckley NA, Juurlink DN, Isbister G, Bennett MH, Lavonas EJ. Hyperbaric oxygen for carbon monoxide poisoning. *Cochrane Database Syst Rev* 2011: CD002041. [\[CrossRef\]](#)
11. Dziewierz A, Ciszowski K, Gawlikowski T, Rakowski T, Kleczyński P, Surdacki A, et al. Primary angioplasty in patient with ST-segment elevation myocardial infarction in the setting of intentional carbon monoxide poisoning. *J Emerg Med* 2013; 45: 831-4. [\[CrossRef\]](#)
12. Dileo PA, Tucciarone M, Castro ER, Guerrero M. Late stent thrombosis secondary to carbon monoxide poisoning. *Cardiovasc Revasc Med* 2011; 12: 56-8. [\[CrossRef\]](#)