Carbon Monoxide Intoxication Presented as Paroxysmal Atrial Fibrillation

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Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of fuels or other material containing carbon. CO poisoning is the leading cause of accidental deaths in many countries, with peak incidences during the cold seasons. The clinical manifestations of CO poisoning are diverse and easily confused with those of other illnesses. Delayed or missed diagnosis usually brings ominous outcomes, not only to the patient but also the cohabitants. Here we reported a 25-year-old woman who presented with paroxysmal atrial fibrillation. Although CO intoxication was not found during her first visit, it was found and treated in the returning visit. Please consider CO poisoning as differential diagnosis in the young, healthy patient who comes with atrial fibrillation, especially when no other significant provoking factor is found. A high index of suspicion and detailed history, particular about environments, cohabitants with similar symptoms, and use of fuel-consuming appliances, may be life-saving.

Key Words: Carbon monoxide intoxication • Paroxysmal atrial fibrillation

INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of fuels or other material containing carbon. It can come from various sources, such as furnaces, generators, gas heaters, and motor vehicles, that are common in homes or work places. CO poisoning is the leading cause of accidental deaths in the United States and may be responsible for more than half of all fatal poisonings worldwide, with peak incidences during the fall and winter seasons.^{1,2}

CO poisoning comes from relative anemia from carboxyhemoglobin (COHb) and direct toxicity at the cellular level. As exposure increases, patients become symptomatic, with oxygen-dependent organs showing the earliest signs of injury. Early manifestation may include dizziness, headache, nausea and vomiting. Increasing exposure may produce weakness, palpitation, breathlessness on exertion, and altered mental status. Seizure, hypotension, dysrhythmias and coma indicate severe intoxication and impending cardiopulmonary arrest. Although aggravation of ventricular arrhythmias has been extensively reported and studied,^{3,4} atrial fibrillation (AF) has seldom been mentioned without exploration.^{5,6} Here we reported a young, healthy lady with CO poisoning who presented as paroxysmal AF.

CASE REPORT

This 25 y/o lady was awakening from sleep because of palpitation, headache, nausea, and vomiting. She arrived at our Emergency Department (ED) in the middle of the night with clear consciousness. Her vital signs were respiration 22 breaths/min, pulse 166 beats/min, blood pressure 115/86 mmHg and temperature 34.6 °C. Physical exam revealed rapid, irregular heart beats but no other abnormality. Electrocardiography (ECG) showed atrial fibrillation with rapid ventricular response (Figure 1). Laboratory results, including complete blood count, biochemistry and cardiac markers, were all within normal limits. Computer tomography was arranged for the

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Figure 1. Surface Electrocardiogram of this patient on first ED presentation. Rapid heart rate with irregular irregularity showed the rhythm to be atrial fibrillation.

patient's headache and the result was unremarkable. After excluding the potential intracranial lesion, intravenous amiodarone was used to slow down her ventricular rate. Her heart rate decreased gradually, with return of sinus rhythm hours later. A cardiologist was consulted and nothing particular was found, including on the echocardiography. She was then discharged home.

Four hours later, the patient was brought back to our ED again because of unresponsiveness at home. Her consciousness was E4M6V5 on arrival, with vital signs of respiration 20 breaths/min, pulse 105 beats/min, blood pressure 113/78 mmHg and temperature 37.8 °C. Head-ache, nausea and vomiting were complained again but involuntary movement or seizure was denied. ECG showed sinus tachycardia. Her husband complained of general weakness and nausea, too. Although indoor burner use was denied initially, significant carboxyhemo-globinemia (COHb 43.2%) and metabolic acidosis with respiratory compensation (pH 7.403, PaO₂ 166.3 mmHg, PaCO₂ 35.3 mmHg, HCO₃⁻ 21.5 mEq/L, BE -2.5 mEq/L, SaO₂ 99.1% on O2 4 L/min, nasal cannula) were re-

vealed. High-flow oxygen was provided followed by hyperbaric oxygen therapy (HBOT) at 2.5 ATA for two hours. The patient received four more courses of HBOT because of persistent dizziness. She recovered without neurological or psychiatrical sequela. Her thyroid function test was later revealed to be euthyroid (T3: 1.37 ng/mL, T4: 9.16 μ g/dL). No recurrence of paroxysmal AF was found during six months of follow-up.

DISCUSSION

The clinical manifestations of CO poisoning are diverse and easily confused with those of other illnesses, such as nonspecific viral illness, influenza, gastroenteritis, benign headache, and various neurologic and cardiovascular syndromes. An estimated one third of CO poisonings may go undetected.^{7,8} A high index of suspicion and detailed history about particular environments, cohabitants with similar symptoms, and use of fuel-consuming appliances are essential to make this diagnosis.

Atrial fibrillation is a rhythm disturbance of the atria that results in rapid, irregular fibrillation waves that vary in size, shape, and timing. Reentrant circuits arising in the atria have been found responsible for this arrhythmia. More recently reported, rapidly firing foci, usually located in or near the pulmonary veins, can produce a similar appearance of AF on the surface ECG., or may degenerate into or trigger reentrant AF.^{9,10} Clinically, AF can occur as a primary arrhythmia in the absence of identifiable structural heart disease; it can also occur secondary to ischemic, inflammatory, structural or infiltrative heart diseases, sick sinus syndrome, cardiomyopathy, pulmonary embolism, cardiothoracic surgery, hyperthyroidism, ethanol or some sympathomimetic drug use.

Paroxysmal AF has been reported to be associated with a higher restrictive filling pattern of heart that comes from respiratory distress or myoischemia.^{11,12} Vasospasm-related paroxysmal AF has been reported in young peoples with normal coronary arteries.¹³ CO exposure can affect the rapid repolarization of myocytes that may establish a period of vulnerability for arrhythmias.¹⁴ However, to our knowledge, there has been neither study about the pathogenesis of paroxysmal AF in CO poisoning nor recommendation of such poisoning in differential diagnosis for paroxysmal AF. Although hypoxemia resulted from CO poisoning is thought to provoke the paroxysmal AF, further studies are necessary to prove the pathogenesis of CO poisoning to paroxysmal AF in healthy patients.

Treatment of the CO-poisoned patient begins with immediate high-flow oxygen therapy and aggressive supportive care. Although whether oxygen therapy should be given under ambient pressures or increased pressure remains controversial, in-time diagnosis and immediate oxygen therapy are the gold standard of life-saving and morbidity reduction. HBOT is often recommended for CO poisoning patients who have any history of loss of consciousness, neurologic signs, cardiovascular dysfunction, severe metabolic acidosis, COHb levels > 25%, or are refractory to ambient-pressure oxygen therapy.¹⁵

The clinical presentations of CO poisoning are often diverse and easily confused. Delayed or missed diagnosis usually brings ominous outcomes not only to the patient but also the cohabitants. Please consider CO poisoning in the differential diagnosis of the young, healthy patient coming with atrial fibrillation, especially if there is no other significant provoking factor found. A high index of suspicion and detailed history about particular environments, cohabitants with similar symptoms, and use of fuel-consuming appliances may save lives.

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以突發性心房顫動做為表現的一氧化碳中毒

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一氧化碳是一種無色、無味的氣體,只要各種燃料或是含碳物不完全燃燒就會產生。許多 國家裡,一氧化碳高居意外死亡原因之首,特別是在寒冷的季節。一氧化碳中毒的臨床表 現非常多變,很容易與其他疾病混淆。錯誤或者僅是延遲診斷,不但危及患者的生命,甚 至其共同居住者也有危險。在此我們報導了一位二十五歲的女性,她就診時表現為突發性 的心房顫動。雖然首次並未能診斷出其一氧化碳中毒,幸好有機會於隨後的返診作出診斷。 因此在遇到突發性的心房顫動的患者時,特別是找不出顯著的致病因子,千萬不要忘記一 氧化碳中毒的可能性。高度的懷疑加上詳細的病史詢問,特別是有關居住環境、同居者是 否有類似症狀、有無使用燃具,就有可能成為救命的關鍵。

關鍵詞:一氧化碳中毒、突發性心房顫動。