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Atrial Fibrillation Induced by Carbon Monoxide Poisoning and Successful Treatment with Hyperbaric Oxygen

By Maheedhar Gedela, MD; Nathan Y. Weltman, MD, PhD; Naga Sushma Chavvakula, MBBS; Paul L. Carpenter, MD, FACC; and Tamera Sturm, DO

Abstract

Carbon monoxide (CO) intoxication is one of the major public health hazards which may go unnoticed as this is a colorless, odorless and tasteless gas. The manifestations of the CO poisoning are far-reaching. Although CO affects almost every organ in the body, cerebral and myocardial involvement are predominant due to the hypoxia-induced cellular damage. The mainstay treatment is providing high-flow oxygen and in some instances hyperbaric oxygen therapy. In the literature, there have been few cases of CO poisoning-induced atrial fibrillation (AF) reported. We hereby report an AF caused by CO toxicity in a young male patient and successful conversion to sinus rhythm with the hyperbaric therapy.

Introduction

Carbon monoxide (CO) poisoning is a toxicological emergency, which is largely preventable by careful operation of household appliances and combustion engines. Although the majority of the cases of CO poisoning present with flu-like symptoms, neuro-psychiatric sequelae such as dementia, depression, personality changes, movement disorders and neuropathy have all been reported. Myocardial injury is another serious consequence of CO poisoning as the myocardium is susceptible to hypoxic damage. In this paper, we present a 22-year-old male who presented with acute onset atrial fibrillation (AF) following CO exposure and successful conversion to sinus rhythm with hyperbaric oxygen treatment.

Case Report:

A 22-year-old African-American male with no known medical history was brought into our facility by emergency medical services after he was found unresponsive in his car. The patient had backed his running vehicle against a snow embankment, inadvertently covering the cars exhaust pipe. The patient was sitting in the car with his friend and smoking marijuana when he noticed nausea and headache just prior to loss of consciousness. Upon admission to the emergency department, the patient's vital signs were as follows: temperature of 94.7 degrees Fahrenheit rectally, irregular pulse rate of 134 beats per minute, respiratory rate of 29 breaths per minute, blood pressure of 132/71 mmHg and oxygen saturation of 98 percent while on 2 liters' oxygen through the nasal cannula. The patient was drowsy and cranial nerves II through XII were intact without focal deficits on neurological exam. His cardiovascular examination revealed an irregularly irregular rhythm. Otherwise remaining physical exam was essentially unremarkable. His initial complete blood count, comprehensive metabolic panel, troponin and thyroid stimulating hormone values are shown in Table 1. The patient's arterial blood gas revealed pH of 7.36, pCO2 of 40 mmHg, pO2 of 94 mmHg, and bicarbonate of 22 mmol/L. His blood CO level was 42.3 percent (reference range 0.0-3.0 percent) and lactic acid level was 3.5 mmol/L (reference range 0.5-2.2). His urine toxicology screen was positive for cannabinoids. The patient's blood alcohol level was less than 10 (reference range <10 mg/dL). His initial electrocardiogram (ECG) revealed an AF with heart rate 130 (Figure 1). A transthoracic

Table 1. Laboratory Data		
Laboratory Parameter	**Result**	**Reference Range**
White blood count	9.4	4.5-11.0 K/μL
Red blood count	5.48	4.70-6.10 M/μL
Hemoglobin	17.1	14.0-18.0 g/dL
Hematocrit	48.5	40.0-54.0 %
Platelet count	232	140-440 K/μL
Sodium	143	133-145 mmol/L
Potassium	4.2	3.5-5.1 mmol/L
Chloride	102	98-107 mmol/L
Anion gap	20	7-17 mmol/L
Blood urea nitrogen	12	6-20 mg/dL
Creatinine	0.7	0.7-1.2mg/dL
Glucose	112	70-105 mg/dL
Calcium	9.4	8.4-10.4 mg/dL
Phosphorous	3.2	2.7-4.5 mg/dL
Magnesium	1.9	1.6-2.6 mg/dL
Aspartate aminotransferase	33	0-37 U/L
Alanine aminotransferase	20	0-41 U/L
Alkaline phosphatase	68	40-129 U/L
Total bilirubin	0.3	0.0-1.0 mg/dL
Troponin T	0.03	0.00-0.10 ng/mL
Thyroid stimulating hormone	0.428	0.270-4.200 μIU/mL

echocardiogram was performed, which showed ejection fraction 55-60 percent and no wall motion abnormalities.

Given the acute CO poisoning, the patient received 100 percent oxygen at 2.5 atmospheres absolute (ATA) for 90 minutes followed by another hyperbaric oxygen therapy at 2.0 ATA for 90 minutes for a total of two treatments. Subsequently, his CO level was brought down to 2.1 percent and lactic acid normalized to 1.0. The patient eventually converted back to normal sinus rhythm as shown below in the telemetry (Figure 2).

Discussion

Each year 40,000 patients are admitted to the hospital due to CO poisoning with associated mortality ranging from 0-31 percent.[1] Due to its lack of odor, color, tastelessness and irritancy, CO inoculation is often initially unrecognized.[2] CO exerts its toxic effects on almost every organ in the body through tissue hypoxia and direct CO-mediated cell damage.[3,4] The central nervous system and myocardium are particularly affected by CO poisoning as these are most sensitive organs to oxygen deprivation.[5] The clinical manifestations of the CO poisoning are wide-ranging and presents with headache, dizziness, lassitude, muscular weakness, nausea, vomiting, cherry-red colored lips and skin, diminished vision, seizure, unconsciousness and ultimately death.[3,4,6]

The affinity of CO for hemoglobin is 200-250 times that of oxygen and this forms a carboxyhemoglobin (CO-Hb) complex which leads to a leftward shift of the oxygen-hemoglobin dissociation curve. These biochemical alterations ultimately produce cellular and tissue hypoxia due to impaired oxygen release.[4] When CO attaches to the myoglobin within cardiac myocytes, it diminishes the transport of oxygen to mitochondria and leads to myocardial dysfunction by impairing myocyte cellular respiratory function.[7] Moreover, direct toxic damage to the coronary arteries and CO-Hb induced hypoxia contribute to the myocardial damage.[1] The various cardiovascular manifestations of CO poisoning include tachycardia, bradycardia, atrioventricular conduction disturbances, cardiomegaly, T-wave and ST-segment changes on ECG, angina pectoris, acute myocardial infarction, AF, premature ventricular contraction, ventricular fibrillation and cardiogenic shock.[2,3] The most prevalent features of the ECG are flattening or biphasic changes in T-wave morphology followed by a variable extent of T-wave inversion.[3,8] The persistence of ECG abnormalities in each patient typically varies from minutes up to several hours.[5,9] However, Shafer et al. reported progressive and persistence of ECG abnormalities and symptomatic sequela for approximately two years in a 35-year-old male patient who has myocardial disease from acute CO poisoning.[6] The actual incidence of AF-related CO intoxication is unknown. In a study examining 2,579 Korean patients with acute CO poisoning, AF was noted in a total of eight (0.3 percent).[3] Although several cases of AF secondary to CO poisoning have been reported in the literature, there is only one published case report of an 82-year-old female who was successfully treated with hyperbaric oxygen therapy.[4] Akademir et al. reported a case of 42-year-old female who had AF due to CO poisoning, in which the rhythm restored to sinus rhythm with normobaric oxygen therapy.[10]

The management of CO poisoning should focus on the correction of tissue hypoxia. High-flow oxygen is essential in the management of CO poisoning as it decreases the half-life ($t_{1/2}$) of CO-Hb to four to six hours and the hyperbaric oxygen therapy further reduces the $t_{1/2}$ to 15-30 minutes.[4]

Figure 1. ECG shows atrial fibrillation with rapid ventricular response.

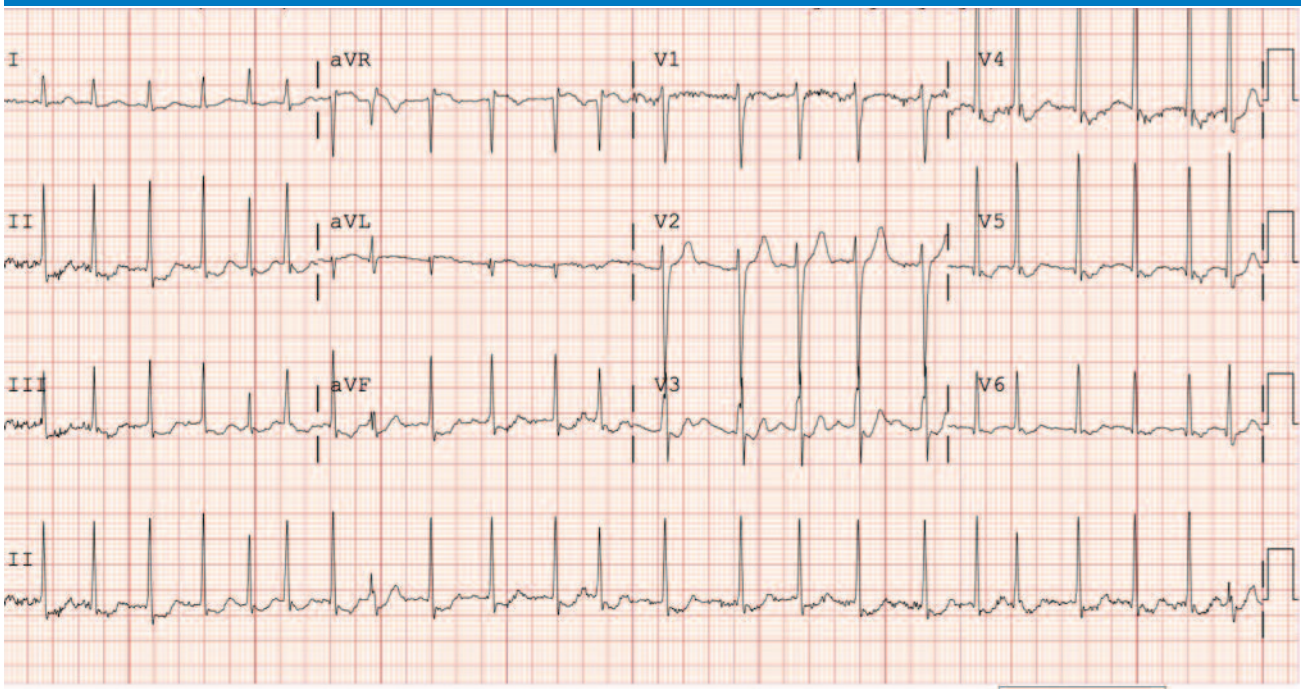
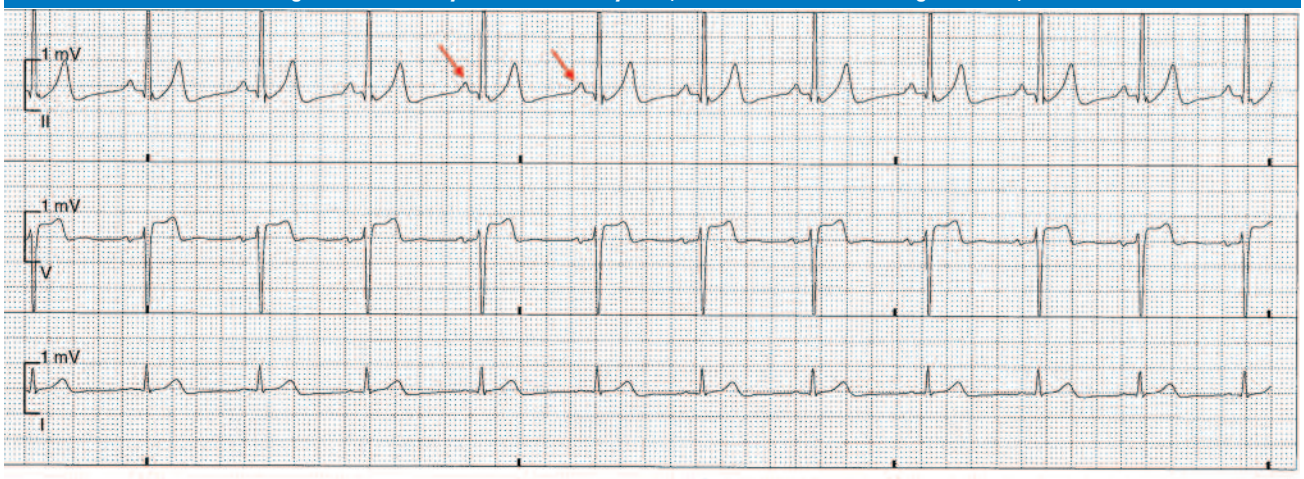


Figure 2. Telemetry shows sinus rhythm (red arrows demonstrating P waves).



To our knowledge, in the English literature, this report represents only the second known case of successful conversion of AF to sinus rhythm using hyperbaric oxygen therapy in acute CO poisoning. In patients with suspected acute CO poisoning, a thorough exposure history, blood CO-Hb levels and ECG should be obtained to identify the patients who require hyperbaric oxygen therapy to prevent myocardial damage.

REFERENCES

Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

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