CASE REPORT

A Case of Carbon Monoxide Poisoning Presenting with Supraventricular Tachycardia

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Abstract

Carbon monoxide intoxication is one of the most common types of poisoning in the world. Cardiac manifestations after exposure to carbon monoxide including myocardial ischemia, heart failure and arrhythmias have been reported. A 17-year-old woman was admitted to the emergency service with the complaints of palpitation, headache and nausea. Electrocardiogram revealed supraventricular tachycardia. The arterial blood gas analysis was normal. Her carboxyhemoglobin level was 19% and oxygen treatment was started promptly. Echocardiographic examination demonstrated normal cardiac function. To the best of our knowledge, this case report is the first carbon monoxide intoxication case in the literature presenting with supraventricular tachycardia attack.

Key words: carbon monoxide poisoning, arrhythmia, supraventricular tachycardia

(Intern Med 50: 2607-2609, 2011)  
(DOI: 10.2169/internalmedicine.50.5929)

Introduction

Carbon monoxide (CO) intoxication is one of the most common types of poisoning, and it is the leading cause of death by poisoning in the world (1). CO binds rapidly to hemoglobin with greater affinity than oxygen (O₂) and forms carboxyhemoglobin (COHb), which leads to a decrease in the O₂ carrying capacity of the blood and subsequent tissue hypoxia. Brain and heart may be severely affected by CO exposure because these organs are very sensitive to hypoxic injury. The patients with acute CO poisoning have various clinical manifestations ranging from mild symptoms such as headache, nausea, dizziness, and palpitation to severe symptoms such as mental confusion, convulsion, paralysis, coma and death (1). We report the first apparent case of acute CO poisoning presenting with supraventricular tachycardia (SVT).

Case Report

A 17-year-old Caucasian woman was admitted to the emergency service with complaints of palpitation, headache and nausea. She had no known cardiovascular disease or smoking habit. She appeared exhausted and, her blood pressure was 116/72 mmHg, pulse 170/bpm, O₂ saturation was 97% by pulse oxymeter. Physical examination was normal except for a regular tachycardia on cardiac examination. Electrocardiogram (ECG) revealed VT with a heart rate of 170/bpm (Fig. 1). The tachycardia was characterized by negative and retrograde P waves in inferior leads and the RP interval was <80 ms (Fig. 1). All these features were suggestive of an atrioventricular nodal reentrant tachycardia. As she was hemodynamically stable, carotid sinus massage was performed initially but she did not respond. Then diltiazem 25 mg was given intravenously and sinus rhythm was achieved (Fig. 2). Echocardiographic examination performed subsequently, demonstrated totally normal cardiac function. She

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Received for publication May 31, 2011; Accepted for publication August 10, 2011
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had normal total blood count, biochemistry and thyroid function tests. The present patient’s mother was admitted to emergency service with the complaints of headache, nausea and dizziness. As two members of a family had similar symptoms at the same time we considered the possibility of poisoning. The arterial blood gas analysis revealed normal findings. Her COHb level was found to be 19% and O2 treatment was started promptly. There was no ECG changes during her follow-up and in serial measures there were no increase in serum cardiac troponin, creatinine kinase or creatinine kinase-MB levels. After O2 treatment, her COHb level returned to normal range and no other cardiac and neurologic symptoms appeared. Supraventricular tachycardia did not recur during one-year follow-up. We did not consider an electrophysiological study as she did not have any palpitation previously and remained asymptomatic during one-year follow-up.

**Discussion**

Cardiac manifestations of CO exposure have been reported as myocardial ischemia, heart failure and arrhythmias (2). Cardiac toxicity may occur because of myocardial hypoxia or a direct toxic effect of CO on myocardial mitochondria. CO binds to intracellular myoglobin in the myocardium and impairs the O2 supply to the mitochondria. This negatively affects the oxidative phosphorylation and consequently it affects the energy source of myocardium.

In our opinion, myocardial ischemia was not the leading mechanism of SVT in the present patient. We did not come across any study concerning SVT induced by coronary ischemia in a literature search. We predominantly think that autonimous dysfunction caused by CO intoxication induced systemic ischemia which thereby triggered a possible concealed accessory pathway or slow pathway causing SVT in our patient. She was totally asymptomatic previously and during one-year follow-up which supported our consideration as well.

Despite the treatment, cardiac sequelae often occur after CO poisoning and these patients are thought to have an increased risk of mortality due to myocardial injury (3-5). Therefore, patients admitted to the hospital with CO poisoning should have examinations for baseline ECG and serial cardiac enzymes. The severity and duration of myocardial injury depend on the duration and amount of CO exposure (6).

Myocardial injury in CO poisoning can be demonstrated by elevated cardiac injury biomarkers, e.g., troponin, brain-natriuretic peptide, creatinine kinase, and creatinine kinase-MB and ischemic ECG changes (2, 6). Decreased left ventricular ejection fraction and right ventricular dysfunction also can be observed in CO-poisoned patients (2, 7-9). Ar-
rhythmias, e.g., sinus tachycardia, atrial fibrillation, premature atrial complexes, conduction abnormalities, premature ventricular complexes and, ventricular fibrillation have been reported in patients with mild to severe CO poisoning (10, 11).

All suspected victims should be treated with normobaric O2 inhalation immediately after arterial blood gas analysis in the emergency department, because the goal is to raise the O2 levels. The role of hyperbaric O2 in the treatment of CO poisoning remains controversial. Hyperbaric O2 therapy is considered the treatment of choice for the cases present with syncope, coma, seizure and focal neurological deficit or COHb >25% (12, 13). Weaver et al (14) concluded that hyperbaric O2 therapy is indicated for acute CO poisoning cases ≥36 years of age or with exposure duration ≥24 h or those presenting with loss of consciousness, or with higher COHb levels.

To date, there seem to be only two CO-induced SVT cases which have been reported in the literature. In those cases SVT developed on days 4 and 11 of hospitalization (15). Furthermore, those patients had been hemodynamically unstable and had required dopamine, dobutamine and epinephrine infusions which had arrhythmogenic potential before SVT attack. In those patients SVT was not the initial manifestation at presentation and would have been due to cardiac inotropes rather than due to a direct toxicity effect of CO. In addition, in those case reports SVT was not well-documented by 12-lead ECG. Contrary to those CO poisoning cases our patient presented with SVT.

In conclusion, SVT may be the initial manifestation of CO poisoning at presentation in addition to the classical manifestations such as headache, nausea and dizziness. Emergency care physicians should take into consideration that CO poisoning may result in SVT which can be easily treated with calcium channel blockers.

The authors state that they have no Conflict of Interest (COI).

References