An Unusual Cause of Supraventricular Tachycardia: Acute Carbon Monoxide Poisoning

Supraventriküler Taşikardinin Nadir Bir Nedeni: Akut Karbon Monoksit Zehirlenmesi

Karbon monoksit ve Supraventriküler Taşikardi / Carbon monoxide and Supraventricular Tachycardia

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Özet

Karbon monoksit (CO); karbon ihtiva eden bileşiklerin tam yanmamasıyla meydana gelen toksik bir gazdır. Yüksek konsantrasyonlarda CO'e maruz kalmak öldürücü olabilir ve dünya genelinde zehirlenmelere bağlı ölümlerin en sık nedenidir. CO'e maruziyet sonrası, miyokard iskemisi, kalp yetmezliği ve aritmi gibi kardiyak belirtiler bildirilmiştir. 28 yaşında bir hasta, soba kaynaklı akut CO'e maruz kalmanın bir sonucu olarak bilinç kaybı ile acil servise kabul edildi. Karboksihemoglobin seviyesi % 39 idi. Hemen oksijen tedavisine başlandı ve terapötik eritrosit değişimi yapıldı. Elektrokardiyogramda supraventriküler taşikardi (SVT) saptandı ve ekokardiyografik incelemede normal kardiyak fonksiyonlar görüldü. Bizim bilgimize göre bu çalışma, akut CO zehirlenmesine bağlı ikinci SVT olgusu raporudur. Bu yazıda CO ile zehirlenmiş hastalardaki bu komplikasyona yaklaşım tartışılmaktadır.

Anahtar Kelimeler

Karbon Monoksit Zehirlenmesi; Supraventriküler Taşikardi; Acil Servis

Abstract

Carbon monoxide (CO) is a toxic gas produced by the incomplete combustion of carbon-containing compounds. Exposure to high concentrations of CO can be lethal and is the most common cause of death from poisoning worldwide. Cardiac manifestations after exposure to CO, including myocardial ischemia, heart failure, and arrhythmias, have been reported. A 28-year-old a patient was admitted to our emergency department with altered consciousness as a consequence of acute domestic exposure to CO from a stove. His carboxyhemoglobin level was 39%. The oxygen treatment was started promptly, and therapeutic red cell exchange was performed. An electrocardiogram revealed supraventricular tachycardia (SVT), and an echocardiographic examination demonstrated normal cardiac functions. To the best of our knowledge, this study is the second to report a case of SVT attack due to acute CO intoxication. This paper discusses the management of this complication in patients poisoned with CO.

Keywords

Carbon Monoxide Poisoning; Supraventricular Tachycardia; Emergency Department

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Introduction

Carbon monoxide is a colorless, odorless, tasteless, and non-irritant gas produced primarily as a result of the incomplete combustion of any carbonaceous fossil fuel. CO poisoning is a serious health problem that results in approximately 50,000 visits to the emergency department and is responsible for 2,700 deaths annually in the United States [1,2]. CO binds to hemoglobin with an affinity more than 200 times that of oxygen and causes a leftward shift in the oxygen-hemoglobin dissociation curve. This shift leads to a decrease in the O2-carrying capacity of the blood and to subsequent tissue hypoxia [3]. Nonspecific symptoms after CO exposure include headache, nausea, vomiting, palpitations, dizziness, and confusion. As exposure increases, patients develop more pronounced and severe symptoms, with oxygen-dependent organs, such as the brain and the heart, showing the earliest signs of damage. Below is a case of acute CO poisoning, which, in the literature, is the second acute CO intoxication case that presents with SVT attack.

Case Report

A 28-year-old man was admitted to our emergency department with altered consciousness as a consequence of acute domestic exposure to CO from a stove. On preliminary evaluation, he was unconscious, the Glasgow Coma Scale was 3, the body temperature was 36.5°C, the pulse rate was 172 beats per minute (bpm), the respiratory rate was 21 breaths/min, the O2 saturation was 95%, and the blood pressure was 160/90 mm Hg. A pulse oximetric measurement performed using a signal extraction pulse CO-oximetry device (Masimo Co., USA) revealed a serum carboxyhemoglobin (COHb) level of 39%. The electrocardiogram (ECG) results revealed SVT with a heart rate of 169 bpm (Fig. 1). According to the information acquired from his relatives, he

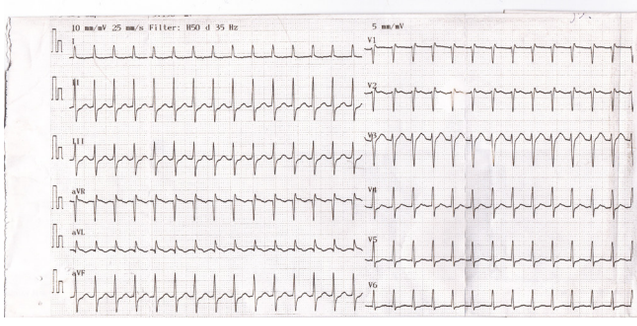


Figure 1. ECG at the time of admission to the emergency service showing supraventricular tachycardia.

had no other disease and did not use any drugs. All these information were suggestive of an atrioventricular nodal reentrant tachycardia due to acute CO poisoning. Oxygen treatment was started immediately. Carotid sinus massage was performed initially for SVT, but the patient did not respond. Then, diltiazem (25 mg) was given intravenously, and sinus rhythm was returned (Fig. 2). Hyperbaric oxygen (HBO) therapy was planned for the patient, but HBO was not available either at our hospital or at another local hospital. Therefore, we used therapeutic red cell exchange (TREX) by automated apheresis to treat our patient. The TREX was performed within first hour of admission to the emergency room. The TREX lasted 55 minutes and used 10 packed red cells (each packed red cell is approximately 400 mL in volume). The COHb level was reduced to 10% after TREX,

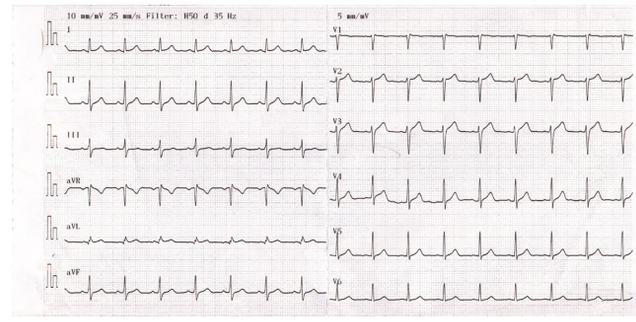


Figure 2. ECG following intravenous administration of diltiazem showing sinus rhythm

and the Glasgow Coma Scale increased to 9. His Glasgow Coma Scale was 15 by the eighth hour after TREX.

An echocardiographic examination performed subsequently demonstrated totally normal cardiac functions. A preliminary blood analysis revealed creatine kinase (CK) of 252 U/L, creatine kinase MB (CK-MB) of 52 U/L, and troponin T of 0.103 ng/mL. The other complete blood cell count parameters, coagulation parameters, biochemical values, and thyroid function tests were normal. The patient's follow-up showed no ECG change, and the serial measures indicated no increase in CK or CK-MB levels. However, serum cardiac troponin increased to 0.11 ng/mL after 4 h. The patient was managed with supportive care. He improved rapidly and made a complete recovery, with all laboratory parameters normalized after 36 h. He was discharged after 12 days of hospitalization. Supraventricular tachycardia did not recur during a 3 month follow-up.

Discussion

CO binds to many heme-containing proteins other than hemoglobin, including cytochromes, myoglobin, and guanylyl cyclase. CO reversibly binds to hemoglobin and causes a leftward shift in the oxygen-hemoglobin dissociation curve. This shift, combined with the CO inhibition of cytochrome P-450-mediated cellular respiration, produces tissue hypoxia, anaerobic metabolism, and lactic acidosis [3].

Myoglobin is another heme-containing protein that binds carbon monoxide, reducing muscular oxygen stores. Myoglobin's affinity for carbon monoxide is 30–50 times greater than its affinity for oxygen. CO binds to intracellular myoglobin in the myocardium and impairs the O2 supply to the mitochondria, which may lead to arrhythmias and cardiac dysfunction [3,4]. CO poisoning can cause oxidative stress, which increases oxidants such as nitric oxide and pro-inflammatory mediators in the bloodstream [3]. This may lead to arrhythmias.

To date, there seem to be only three CO-induced SVT cases, which have been reported in the literature. In one of those cases, SVT developed during the acute phase [5]. In other cases, SVT developed on days 4 and 11 of hospitalization [4]. The two cases were hemodynamically unstable and required dopamine, dobutamine, and epinephrine infusions [4]. SVT might occur due to cardiac inotropes rather than to direct toxic effects of CO in those two cases.

Although we did not come across any study concerning SVT induced by coronary ischemia in a literature search, we predominantly thought that the cause of SVT in the present patient was myocardial ischemia. Still, autonomic dysfunction caused

by CO intoxication induced systemic ischemia, which triggered a possible concealed accessory pathway or a slow pathway or which deranged the cardiac neural conduction caused by oxidants. Also, pro-inflammatory mediators might lead to SVT in our patient.

The initial symptoms after CO exposure include headache, nausea, and dizziness. The clinical effects of CO poisoning are diverse and are easily confused with other illnesses, such as nonspecific viral illness, benign headache, and various cardiovascular and neurologic syndromes. The COHb levels do not correlate with the severity of signs and symptoms. The duration of exposure to CO is a more important factor that affects the clinical status [6]. As exposure increases, patients develop more pronounced and severe symptoms, with oxygen-dependent organs, such as the brain and the heart, showing the earliest signs of injury [3].

Early cardiovascular effects of CO poisoning are manifested as a response to hypoxia. Prolonged exposure results in hypotension, dysrhythmia, ischemia, infarction, and, in extreme cases, cardiac arrest. Early deaths after CO exposure may be due to cardiac dysrhythmias. CO poisoning also exacerbates underlying cardiovascular diseases [3]. CO poisoning may cause arrhythmias, such as sinus tachycardia, atrial fibrillation, premature atrial complexes, premature ventricular complexes, and ventricular fibrillation [6]. Myocardial injury from CO poisoning can be demonstrated by elevated cardiac injury biomarkers, such as troponin, CK, and CK-MB, and by ischemic ECG changes. A high index of suspicion is essential for making a diagnosis of occult CO poisoning. Serum COHb levels should be obtained from patients suspected of CO poisoning [3]. The standard treatment is the administration of 100% oxygen, which should be done until the COHb level is restored [3]. Typically, HBO is used to treat patients with severe CO poisoning; however, it may not be available in most places. In these cases, TREX is an alternative treatment method [7]. In our study, a 100% oxygen treatment was started immediately, but HBO was not available at our hospital or another local hospital, so we used TREX to treat our patient.

Despite an appropriate treatment, cardiac sequelae may occur after moderate to severe CO poisoning, and patients are thought to have an increased risk of mortality due to myocardial injury. Therefore, patients admitted to the hospital with CO poisoning should be tested for baseline ECG and serial cardiac enzymes [8].

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

Competing interests

The authors declare that they have no competing interests.

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