

Carbon Monoxide Intoxication Induced Atrial Fibrillation

Karbonmonoksit Zehirlenmesine Bağlı Atrial Fibrilasyon

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Abstract

Carbon monoxide (CO) is a colorless, odorless gas which is a leading cause of accidental deaths during winter months. Its toxic effect occurs by binding to hemoglobin or directly at cellular level. Electrocardiographic findings due to CO intoxication are ST-segment changes, premature contractions, sinus tachycardia, bundle blocks and atrial fibrillation (AF). We report a 33-year-old man who presented with AF due to CO poisoning. The patient was treated with anti-ischemic drugs and oxygen therapy. (*The Medical Bulletin of Haseki 2012; 50: 25-6*)

Key Words: Electrocardiography, carbon monoxide, emergency department, atrial fibrillation

Özet

Karbonmonoksit gazı renksiz, kokusuz bir gaz olup kış aylarında olan kazara ölümlerde önemli bir yer almaktadır. Toksik etkisi doğrudan hücre düzeyinde veya hemoglobine bağlanarak meydana gelir. Premature kontraksiyonlar, sinüs taşikardisi, dal blokları, ST-segment değişiklikleri ve atrial fibrilasyon görülen elektrokardiografik değişikliklerden bazılarıdır. Bu çalışmada, 33 yaşında ve karbonmonoksit zehirlenmesine bağlı atrial fibrilasyon ile başvuran, kardiyak belirteçlerinde artış olup anti-iskemik ve oksijen tedavisi ile düzelen bir olgu sunulmaktadır. (*Haseki Tıp Bülteni 2012; 50: 25-6*)

Anahtar Kelimeler: Elektrokardiografi, karbonmonoksit zehirlenmesi, acil servis, atrial fibrilasyon

Introduction

Carbon monoxide (CO) poisoning is associated with a high incidence of morbidity and mortality. The central nervous system is the most sensitive organ system and the clinical findings of CO poisoning may vary from a simple headache to coma. While CO poisoning generally occurs in winter and the complaints resemble, it may be confused with influenza. Myocardial ischemia and fatal ventricular arrhythmias can be seen in CO poisoning due to hypoxia. (1) In this study, we present a case of AF due to CO intoxication.

Case: A 33-year old male patient with complaints of headache, nausea-vomiting and palpitation was admitted to our emergency department due to CO intoxication. His vital signs were as follows: respiration: 24 breaths/min, pulse: 166 beats/min, blood pressure: 110/70 mmHg and, temperature: 36.7°C. His complaints were headache, nausea-vomiting and palpitation. Physical examination revealed rapid, irregular heartbeats and mental confusion without other abnormalities. The complete blood count was

within normal limits. The laboratory examination including cardiac markers and biochemical tests demonstrated hypokalemia (3.4 mmol/L) and hypocalcemia (7.9 mg/dL) and blood glucose level of 196 mg/dL. He had AF with increased ventricular response in electrocardiogram.

His first troponin level was 0.03 ng/mL and increased to 0.18 ng/mL within 4 hours in spite of high-flow oxygen therapy (40%). The patient was referred to the cardiology department and treated with anti-ischemic and oxygen therapy. In the next day he returned to normal sinus rhythm and was discharged with 100 mg acetyl salicylic acid. Coronary angiography was not performed since the patient did not describe angina and he was not among the risky group. No recurrence of paroxysmal AF was found at the three-month follow-up.

Discussion

CO induced hypoxia following acute exposure is one of the causes of cardiac toxicity. Besides, cytotoxic effects are seen due to the inhibition of intracellular cytochrome

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Received/Geliş Tarihi: 24 June 2011 **Accepted/Kabul Tarihi:** 04 August 2011

Presented to Congress: VII.Acil Tıp Kongresi 26-29 Mayıs Maxx ,Royal Otel Antalya

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Haseki Tıp Bülteni,
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oxidase, which blocks the cellular respiration and generation of adenosine triphosphate. Depending on the carboxyhaemoglobin level, supraventricular and fatal ventricular arrhythmias can occur (1,2).

Atrial fibrillation affects nearly 1% of the population. It is a supraventricular chaotic tachyarrhythmia, characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. Hyperthyroidism, cardiothoracic surgery, pulmonary embolism, sick sinus syndrome, ethanol or sympathomimetic drugs are the other causes of AF in patients without structural heart diseases (3). Besides, AF is triggered by respiratory distress or myoischemia in increased restrictive filling pattern of the heart (4,5). Huang et al. (6) presented a case of AF due to CO poisoning. In a study by Keles et al. (6) the complaints associated with CO poisoning were; headache (55%), nausea (49%), dizziness (44%), syncope (28%), palpitations (13%), dyspnea (6%), seizures (4%), and chest pain (3%). Aslan et al. (5) reported in their study that, palpitation was observed in 39.8% of carbon monoxidepoisoned patients, while 26.5% had sinus tachycardia, 14.4% ischemic changes, 7.2% sinus tachycardia-ventricular extrasystoles, and 3.6% of patients had sinus arrhythmia.

Myocardial damage by carbon monoxide exposure is explained by two factors; decreased oxygen transport capacity of the blood which leads to a decreased amount of oxygen available to the tissues, and impaired mitochondrial function due to a reversible inhibition of the intracellular respiration by the formation of cytochrome aa3-CO ligand.

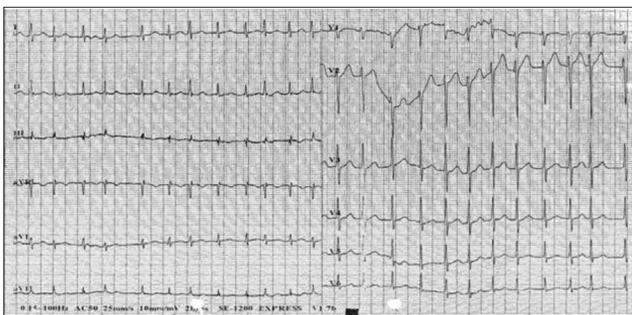


Figure 1. Atrial fibrillation electrocardiography

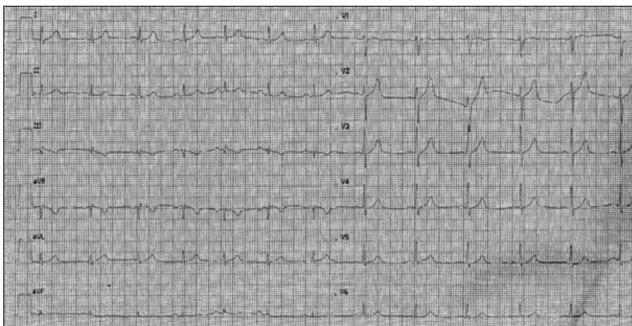


Figure 2. Normal sinus rhythm after treatment

Besides, changes in the hematocrit values, viscosity and platelet functions take an important place for inducing acute coronary syndromes among patients with carbon monoxide intoxication without any coronary artery disease (7). Gul et al. reported a patient who had increased cardiac biomarkers without myocardial infarction (8). In our case, coronary angiography was not planned since cardiac biomarkers of the patient returned to the normal ranges in one day and the patient did not describe any angina.

In conclusion, CO poisoning-related AF and myocardial damage occurrence must be kept in mind in the differential diagnosis and it can be treated by anti-ischemic drugs and high-flow or hyperbaric oxygen therapy.

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