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(CASE REPORT)

Atrial fibrillation induced by carbon monoxide poisoning: Case report

Shanavaskhan Shamsudeenkutty [1], Ashraf ALakkad [2,*] and Khalid Parvaiz [2]

[1] *Department of Cardiology, Madinat Zayed Hospital, AL Dhafra region, UAE.*
[2] *Department of Internal Medicine, Madinat Zayed Hospital, AL Dhafra region, UAE.*

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Abstract

Background: Carbon monoxide (CO) poisoning is known to have direct toxic effects on the cardiovascular system, including the potential to cause arrhythmias by prolonging the QTc interval. The arrhythmogenic properties of CO are believed to be attributed to its impact on cardiac membranes.

Case Presentation: This case report presents the clinical course and management of a 21-year-old male who presented to the Emergency Department (ED) with an altered level of consciousness following exposure to carbon monoxide while cooking on a gas cylinder in a closed room. The patient had no history of chronic medical illness and was a non-smoker. Physical examination revealed stable vital signs, clear breath sounds, and a lax, soft, and non-tender abdomen. Laboratory investigations showed elevated troponin levels indicative of cardiac muscle injury but normal potassium levels. ECG revealed atrial fibrillation with a fast ventricular rate and transient ST elevation. The patient received high flow oxygen. The carboxyhemoglobin level decreased significantly after high-flow oxygen administration, confirming the successful treatment of carbon monoxide poisoning. Echocardiography and coronary angiography demonstrated normal cardiac function and ruled out coronary artery disease, respectively. The patient received high-flow oxygen therapy, continuous cardiac monitoring, and supportive care during hospitalization. Following stabilization and restoration of sinus rhythm, the patient was discharged in stable condition.

Conclusion: This case highlights the importance of prompt recognition and management of carbon monoxide poisoning, as well as the need for careful evaluation of cardiac function in such cases. The patient responded well to high-flow oxygen therapy, leading to a decrease in the carboxy hemoglobin level and resolution of symptoms. Timely recognition and appropriate management of carbon monoxide poisoning are crucial in preventing adverse cardiac events and ensuring a favorable outcome for the patient.

Keywords: CO poisoning; Atrial Fibrillation; ST Elevation

1. Introduction

In recent decades, carbon monoxide (CO) poisoning has become a significant global health concern, earning it the ominous nickname of the "silent killer." (1)This is attributed to the extensive clinical impact it exerts and the elevated rates of morbidity and mortality associated with its toxicological effects(2). The United States alone witnesses approximately 50,000 emergency department visits annually as a result of CO poisoning, with a staggering 2,700 individuals losing their lives to this silent threat(3).

Carbon monoxide is a highly toxic gas that possesses distinct characteristics, making it particularly insidious. It is odorless, tasteless, and lacks irritant properties, making it imperceptible to the human senses(4). Consequently, individuals can be exposed to CO without being aware of its presence, increasing the risk of poisoning(5). Various

* Corresponding author: Ashraf ALakkad

sources contribute to CO poisoning, with combustion-related emissions being a primary culprit. Common sources include vehicle exhaust, malfunctioning heating systems, and the burning of charcoal(6). Notably, in United states, charcoal and motor gasoline are the predominant sources of CO poisoning, emphasizing the need for awareness and preventive measures in different regions(7).

The toxic effects of carbon monoxide (CO) are mediated through a complex interplay of mechanisms that contribute to its detrimental impact on the body(8). One of the key mechanisms involves CO's remarkably high affinity for hemoglobin, which surpasses that of oxygen(9). This affinity allows CO to competitively bind with hemoglobin, reducing the availability of binding sites for oxygen molecules and impeding their transportation to tissues(10). Consequently, a state of profound tissue hypoxia ensues, as vital organs and tissues are deprived of an adequate oxygen supply(11).

The clinical manifestations of carbon monoxide (CO) poisoning are non-specific and can resemble various common disorders(12). The severity of symptoms can range from mild flu-like manifestations to severe cases of coma and even death(13). Due to their elevated metabolic activity, heart is particularly vulnerable to the toxic effects of CO. However, there is limited information available regarding the cardiovascular manifestations of CO poisoning in humans, with most available data consisting of case reports.

Research also suggests that carbon monoxide (CO) has a detrimental impact on various organs and tissues, although the toxidromes associated with CO poisoning lack clinical specificity and are frequently disregarded or misdiagnosed(1). Notably, due to the high oxygen requirements of the cardiovascular and central nervous systems, these organs are particularly prone to manifesting the acute and delayed clinical features of CO poisoning(14). It is important to recognize that cardiovascular disease stands as the leading cause of mortality globally, including in the United States, contributing to approximately 30% of all deaths(15).

This case report discusses a case of a 21-year-old patient who developed Atrial Fibrillation due to carbon monoxide poisoning as a result of exposure to smoke coming from the cooking.

2. Case Presentation

A 21-year-old male, a non-smoker, with no history of chronic medical illness, presented to the Emergency Department (ED) with an altered level of consciousness following exposure to carbon monoxide (CO) while cooking on a gas cylinder in a closed room.

There was no reported chest pain, nausea, or vomiting. On examination, the patient's vital signs were stable, with a blood pressure of 125/70 mmHg. Oxygen saturation was maintained at 100% on a non-rebreathing mask with a flow rate of 15 L/min. However, the patient was in atrial fibrillation with a fast ventricular rate, with a heart rate of 135 beats per minute, as observed on the electrocardiogram (ECG). The second ECG also showed transient ST elevation in the anterolateral leads.

A physical examination of the chest revealed clear breath sounds with no signs of tachypnea or dyspnea. The abdomen was lax, soft, and non-tender upon palpation. Laboratory tests revealed a troponin level of 14 ng/L, indicating cardiac muscle injury, and a potassium level of 3.4 mmol/L, within the normal range. Additionally, the carboxyhemoglobin (COHB) level decreased from 41% to 5% after the administration of high-flow oxygen, indicating successful treatment of carbon monoxide poisoning. An echocardiogram was performed, which revealed a normal study with no structural abnormalities or signs of cardiac dysfunction. Furthermore, a coronary angiogram was conducted, and the results were unremarkable, showing no evidence of coronary artery disease.

The patient was managed in the ED with high-flow oxygen, resulting in a significant reduction in the COHB level and improvement of the altered level of consciousness. Continuous cardiac monitoring and supportive care were provided throughout the hospital stay. Following stabilization, the patient was discharged in a stable condition with a sinus rhythm.

The patient was advised to follow up with a primary care physician for further evaluation and monitoring of cardiac function. It was recommended to avoid further exposure to carbon monoxide and ensure adequate ventilation during cooking activities. Additionally, the patient was educated about the potential risks and symptoms associated with carbon monoxide poisoning.

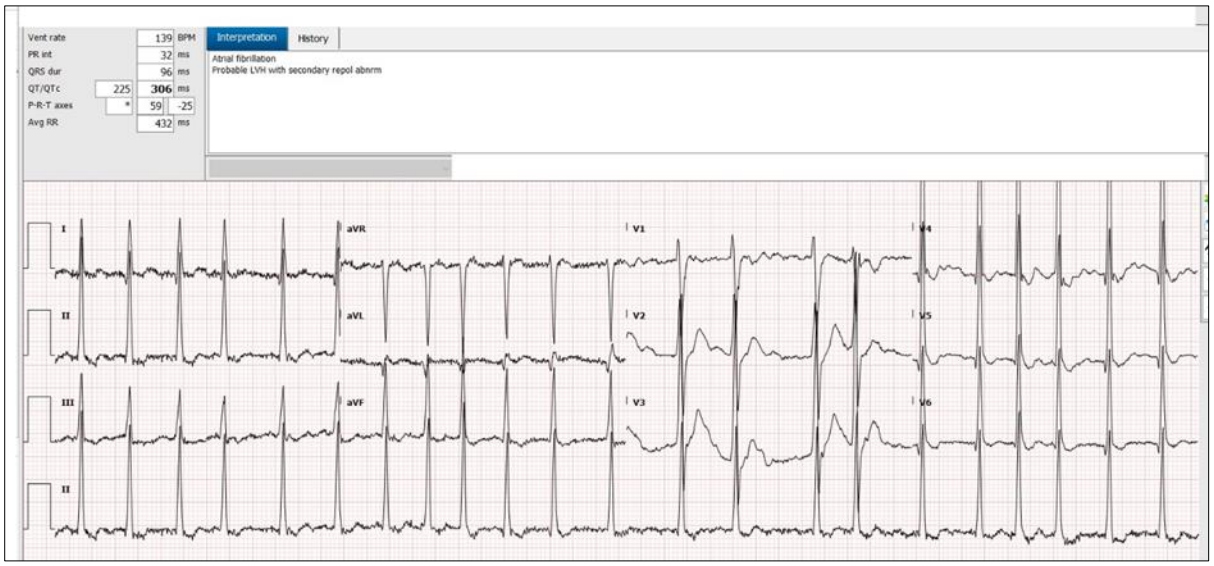

Figure 1 ECG showed Atrial Fibralltion

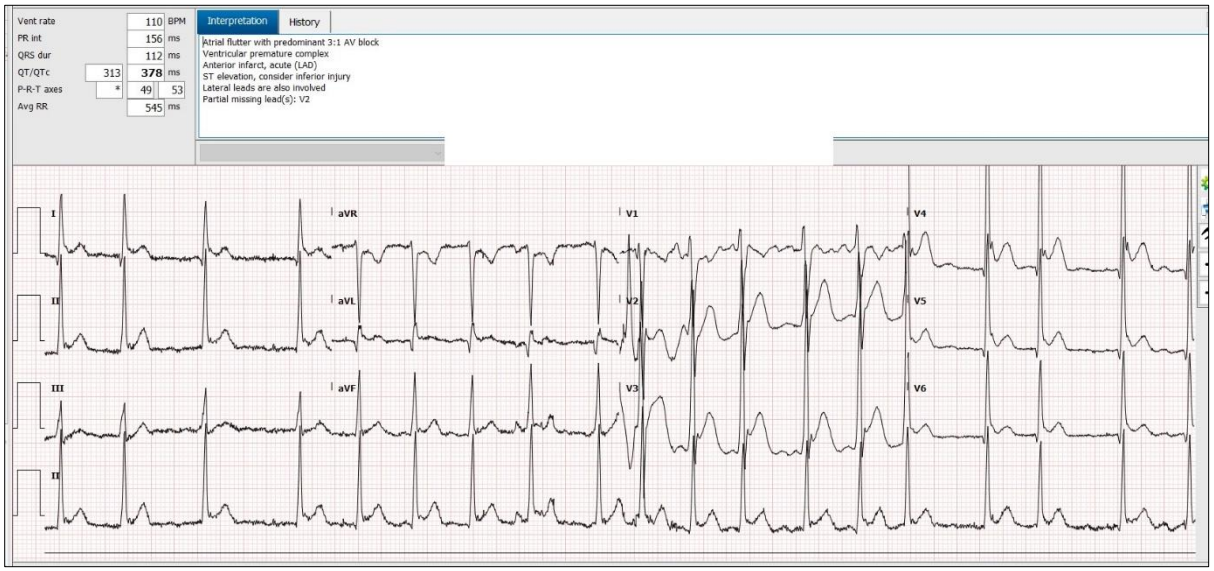

Figure 2 ECG showed transient ST elevation in the anterolateral leads

3. Discussion

The presented case highlights the potentially severe consequences of carbon monoxide (CO) poisoning on the cardiovascular system, as evidenced by the patient's altered level of consciousness, atrial fibrillation with fast ventricular rate, and transient ST elevation on the electrocardiogram (ECG). Carbon monoxide is a colorless, odorless gas that can be produced by incomplete combustion of fuels such as gas, coal, or wood. Inhalation of high levels of CO can lead to tissue hypoxia, including cardiac ischemia, due to its strong affinity for hemoglobin, forming carboxyhemoglobin (COHB)(16).

Carbon monoxide (CO) poisoning can lead to a range of cardiovascular manifestations. These include Cardiogenic shock, bradycardia, tachycardia, cardiomegaly, disturbances in atrioventricular conduction, T-wave and ST-segment changes on the electrocardiogram, acute myocardial infarction, angina pectoris, ventricular fibrillation, atrial fibrillation and premature ventricular contractions(13).

One of the most commonly observed ECG features in CO poisoning is the flattening or biphasic changes in T-wave morphology, often followed by variable degrees of T-wave inversion(12). The duration of ECG abnormalities typically varies from minutes to several hours in each patient(17). The ECG finding of our patient also showed ST segment elevation. Additionally, there have been reported cases of prolonged ECG abnormalities and symptomatic sequelae lasting approximately two years. For example, a research documented the case of a 35-year-old male patient with myocardial disease resulting from acute CO poisoning, who experienced progressive and persistent ECG abnormalities and related symptoms over this extended period(18).

Myocardial injury is a commonly observed consequence of carbon monoxide (CO) poisoning, and it significantly increases the risk of mortality in affected individuals. Therefore, it is of paramount importance to conduct thorough screening for evidence of myocardial injury in patients who have experienced CO poisoning(18).

A study conducted aimed to assess the mortality risk in patients with moderate to severe CO poisoning. The researchers examined a cohort of 85 patients who had sustained myocardial injury as a result of CO poisoning. The findings were striking, as they revealed that 38% of these patients had succumbed to mortality within an average duration of 7.6 years(5).

Myocardial injury or dysfunction is primarily influenced by two key factors: the level of carboxyhemoglobin and the duration of CO exposure. In general, cardiac abnormalities associated with CO poisoning tend to recover rapidly, but in rare cases, permanent or persistent damage may occur(19). Elevated cardiac biomarkers and ischemic changes on the electrocardiogram (ECG) can indicate myocardial injury in CO poisoning(20). Additionally, patients with CO poisoning may exhibit decreased left ventricular function, right ventricular dysfunction, and arrhythmias(21). According to a local study focusing on CO poisoning complications, cardiac arrhythmias were present in 29% of the patients(22).In our patient, levels of troponin levels were found to be elevated indicating a cardiac injury and the patient also developed atrial fibrillation with fast ventricular rate.

The exact incidence of atrial fibrillation (AF) in relation to carbon monoxide (CO) poisoning is not well-established. In a study involving 2,579 Korean patients with acute CO poisoning, AF was observed in a total of eight cases, representing a prevalence of 0.3 percent(23). Although there have been several reported cases of AF secondary to CO poisoning, only one published case report documents the successful treatment of an 82-year-old female using hyperbaric oxygen therapy(21). A case of a 42-year-old female with AF caused by CO poisoning was also reported where the rhythm was restored to sinus rhythm with normobaric oxygen therapy(23). Our patient also developed atrial fibrillation.

In managing CO poisoning, the primary focus should be on correcting tissue hypoxia. High-flow oxygen therapy is essential for CO poisoning management as it reduces the half-life (t1/2) of carboxyhemoglobin (CO-Hb) to four to six hours(24). Hyperbaric oxygen therapy can further decrease the t1/2 to 15-30 minutes(25). Our patient has also been treated administration of high-flow oxygen to displace CO from hemoglobin, which resulted in a significant reduction in the COHB level and improvement of the altered level of consciousness. Continuous cardiac monitoring and supportive care were provided to ensure the patient's hemodynamic stability. The patient's discharge in a stable condition with sinus rhythm indicates a successful response to the treatment.

Following discharge, the patient was advised to follow up with a primary care physician for further evaluation and monitoring of cardiac function. This is crucial to assess any long-term effects of CO poisoning on cardiac function and to detect any potential arrhythmias or cardiac complications that may manifest at a later stage.

In addition, it is important for the patient to be educated about the risks and symptoms associated with carbon monoxide poisoning to prevent future exposures. Adequate ventilation during cooking activities and the installation of carbon monoxide detectors in living spaces are essential preventive measures.

4. Conclusion

This case highlights a 21-year-old male who presented with an altered level of consciousness after exposure to carbon monoxide while cooking in a closed room. Despite the transient ST elevation observed on the ECG, subsequent investigations, including echocardiography and coronary angiogram, revealed normal cardiac function and no evidence of coronary artery disease. The patient responded well to high-flow oxygen therapy, leading to a decrease in the COHB level and resolution of symptoms. Timely recognition and appropriate management of carbon monoxide poisoning are crucial in preventing adverse cardiac events and ensuring a favorable outcome for the patient.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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