



## Development of Atrial Fibrillation Associated with Carbon Monoxide Poisoning

### Karbonmonoksit Zehirlenmesi ile İlişkili Atriyal Fibrilasyon Gelişimi

Ali Bağcı<sup>1</sup>

<sup>1</sup>Isparta City Hospital, Isparta, Turkey.

#### Abstract

**Objective:** Carbon monoxide (CO) poisoning is one of the most important toxicological global causes of morbidity and mortality. Carbon monoxide poisoning causes myocardial damage and arrhythmias by impairing the transport of oxygen. In the literature, there have been few cases of CO poisoning-induced atrial fibrillation (AF) reported. In this case, we report a successful conversion of sinus rhythm into a female patient by providing high flow oxygen (to intubate) to the atrial fibrillation caused by CO toxicity.

**Case:** We report a 53-year-old female patient who had atrial fibrillation due to carbon monoxide poisoning and was given sinus rhythm after removal of hypoxia.

**Conclusion:** The patient was intubated and hypoxia was removed after high flow oxygen. After 12 hours, the patient was extubated on the improvement of the blood gas parameters. Normal sinus rhythm was detected on the control ECG.

**Key Words:** Atriyal Fibrilasyon, Karbonmonoksit Zehirlenmesi, Yüksek Akımlı Oksijen.

#### Özet

**Amaç:** Karbonmonoksit (CO) zehirlenmesi, dünyada morbidite ve mortaliteye neden olan en önemli toksikolojik nedenlerden biridir. Karbonmonoksit zehirlenmesi, oksijenin taşınmasını bozarak miyokardiyal hasara ve aritmilere neden olur. Literatürde, az sayıda CO zehirlenmesi kaynaklı atriyal fibrilasyon (AF) vakası bildirilmiştir. Bu vakada bir kadın hastada CO toksitesinin neden olduğu atriyal fibrilasyonu yüksek akımlı oksijen sağlayarak yani entübe ederek sinüs ritmine başarılı dönüşümünü bildirdik.

**Olgu:** Karbonmonoksit zehirlenmesi sonucu atriyal fibrilasyon gelişen ve hipoksi giderildikten sonra sinüs ritmi sağlanan 53 yaşında kadın hastayı sunduk.

**Sonuç:** Hasta entübe edilmiş olup yüksek akımlı oksijen sonrası hipoksi giderilmiştir. Kan gazı parametrelerinin düzelmesi üzerine ekstübe edilmiştir. Kontrol EKG’de normal sinüs ritmi tespit edilmiştir.

**Anahtar Kelimeler:** Atriyal Fibrilasyon, Karbonmonoksit Zehirlenmesi, Yüksek Akımlı Oksijen.

#### Introduction

Carbon monoxide (CO) poisoning is one of the most important toxicological global causes of morbidity and mortality. Carbon monoxide poisoning is one of the major public health hazards which may go unnoticed as this is a tasteless, odorless and colorless gas (1). The central nervous system and cardiovascular system, which are more sensitive to hypoxia, are affected by this poisoning. Carbon monoxide happens myocardial damage and arrhythmias by reducing the transport of oxygen (2). In the literature, there have been few cases of CO poisoning-induced atrial fibrillation (AF) reported (2, 3). We hereby report an AF caused by CO toxicity in a female patient and successful conversion to sinus rhythm with the providing high-flow oxygen (to intubate).

#### Case Report

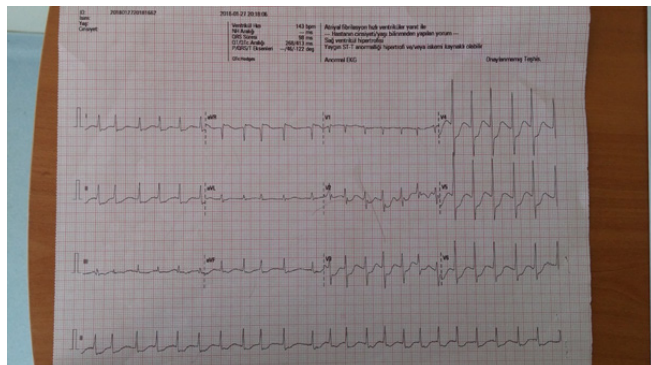
In this case, a 53-year-old female patient was brought to the emergency room with the complaint of loss of consciousness. She had no past medical history of any chronic diseases. Arterial blood gas was studied on the information that the

patient was found in a stove room. Arterial blood gases showed pH: 7.24 FCOHb: 12.4% pCO<sub>2</sub>: 40 mmHg pO<sub>2</sub>: 60 mmHg. Electrocardiography showed atrial fibrillation with a rapid ventricular response and diffuse ST depression (Figure 1). The patient was intubated and supplied 100% O<sub>2</sub>. After 12 hours, the patient was extubated on the improvement of the blood gas parameters. Normal sinus rhythm was detected on the control ECG (Figure 2). Patient’s first troponin level was 1.5ng/mL, and the highest value was 3.5ng/mL. The patient underwent coronary angiography. Normal coronary arteries were detected. The patient was discharged after 48 hours of observation.

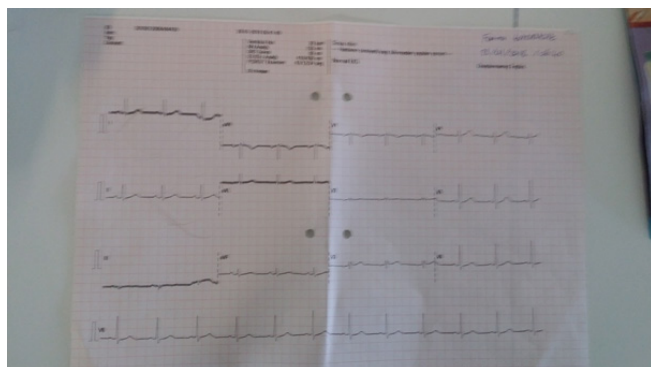
#### Discussion

The affinity of carbon monoxide to hemoglobin is over than oxygen. Furthermore, CO reduces the oxygen carrying capacity of hemoglobin after binding to hemoglobin in red blood cells (4). As a result of this, increased blood concentration of CO happens to reduced tissue oxygen transport (4). A change in cardiac markers and ECG may occur because of insufficient

oxygen in myocardial tissue (5). Myocardial damage can be explained by respiratory dysfunction in myocardial cells, directs toxic damage in the coronary arteries, COHb-associated hypoxia, thrombogenic effect and smooth muscle relaxation in the vascular wall (6).



**Figure 1.** ECG on first application



**Figure 2.** Post-extubation ECG

In previous studies, ECG changes and the myocardial damage have been reported due to the CO poisoning (3, 7-9). Cakir et al (8) found AF with rapid ventricular response and an elevation in cardiac markers. Szponar et al (9) were observed an increase in cardiac markers and ECG changes (eg, AF) in two young patients with unconsciousness brought to the emergency department. Carnevali et al (3) showed that there was a relationship between severity of poisoning and cardiac involvement. In addition, Carnevali et al (3) determined AF and sinus tachycardia episodes. Similarly, in our case cardiac markers were elevated and the rhythm was atrial fibrillation with rapid ventricular response. However, sinus tachycardia was not observed in our case.

## Conclusion

Carbon monoxide intoxication has an important impact on both individuals and the community due to emerging clinical outcomes. It should be remembered that in such cases, the patient needs to provide the rapid oxygenation, various arrhythmias can develop, and rhythm must be monitored.

## References

1. Chou C-H, Lai C-H, Liou S-H, Loh C-H. Carbon monoxide: an old poison with a new way of poisoning. *Journal of the Formosan Medical Association*. 2012; 111(8): 452-5.
2. Akdemir HU, Güngörer B, Çalışkan F, Çolak Ş, Güzel M. Atrial fibrillation related to carbon monoxide poisoning in a female patient. *The American journal of emergency medicine*. 2014; 32(9): 1154. e3-. e5.
3. Carnevali R, Omboni E, Rossati M, Villa A, Checchini M. Electrocardiographic changes in acute carbon monoxide poisoning. *Minerva medica*. 1987; 78(3): 175-8.
4. Varol E, Ozaydin M, Aslan SM, Dogan A, Altinbas A. A rare cause of myocardial infarction: acute carbon monoxide poisoning / Miyokard infarktusunun nadir bir sebebi: Akut karbon monoksit zehirlenmesi. *The Anatolian Journal of Cardiology (Anadolu Kardiyoloji Dergisi)*. 2007; 7(3): 322-4.
5. Ryoo S, Sohn C, Kim H, Kwak M, Oh B, Lim K. Intracardiac thrombus formation induced by carbon monoxide poisoning. *Human & experimental toxicology*. 2013; 32(11): 1193-6.
6. Szponar J, Kołodziej M, Majewska M, Zaleski K, Lewandowska-Stanek H. Myocardial injury in the course of carbon monoxide poisoning. *Przegląd lekarski*. 2012; 69(8): 528-34.
7. Cha YS, Cha KC, Kim OH, Lee KH, Hwang SO, Kim H. Features and predictors of myocardial injury in carbon monoxide poisoned patients. *Emerg Med J*. 2014; 31(3): 210-5.
8. Cakir Z, Aslan S, Umudum Z, Acemoglu H, Akoz A, Turkyılmaz S, et al. S-100 $\beta$  and neuron-specific enolase levels in carbon monoxide-related brain injury. *The American journal of emergency medicine*. 2010; 28(1): 61-7.
9. Szponar J, Majewska M, Drelich G, Kostek H, Tchórz M, Górska A. Myocardial infarction secondary to carbon monoxide poisoning--a study of two cases. *Przegląd lekarski*. 2011; 68(8): 527-9.