LETTER TO EDITOR

Phnx Med J. July 2022, Volume 4 No 2
DOI:10.38175/phnx.1031828

Carbon Monoxide Poisoning and Sequels of Cardiac Function

Karbon Monoksit Zehirlenmesi ve Kardiyak Fonksiyonun Sekelleri

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Dear editor,

Carbon monoxide can cause chemical anemia by binding to hemoglobin and shifts the oxyhemoglobin curve to the left, preventing oxygen from being released into the tissues (1). Elevated levels of cardiac markers such as troponin may be seen when myocardial damage occurs. Electrocardiographic or echocardiographic changes can be monitored. To explain this damage in the myocardium, the toxic effect of carbon monoxide binding to myoglobin is also mentioned in addition to the oxidative stress of carboxyhemoglobin (2). Carbon monoxide-bound myoglobin is not capable of delivering sufficient oxygen to the myocardium (2). Another plausible explanation of tissue hypoxia is binding carbon monoxide to cardiac myoglobin causes myocardial depression, hypotension, and arrhythmias. Carbon monoxide creates myocardial damage through many molecules (3) and this damage to the myocardium may be permanent. For example, within the last few years, a 21-year-old male patient was referred to our clinic with emergency medical services because of loss of consciousness. On anamnesis, it was learned that the family of a patient who had no illness story stayed in barracks and burned fire in the barracks to warm up. Glasgow Coma Score of the patient was 15 (eye 4, verbal 5, and motor 6). The vital parameters were within normal limits. Patient who was not able to protect the respiratory tract, occasionally had orotracheal intubation. There was no ischemic change on electrocardiography. Common brain edema was observed on cranial computed tomography. Troponin I level was measured at 1.937 ng/mL (normal value below 0.0262n/mL), and blood biochemical values within normal range. On the echocardiography, the left ventricle was assessed as globally hypokinetic. The troponin I observations of the patient are summarized in the figure. The patient was extubated on the fourth day after the follow-ups in our clinic. During the clinical follow-up period, the patient was given 300 mg of clopidegrel and acetylsalicylic acid 300 mg per day by nasogastric tube. After extubating consciousness and perception were evaluated as normal. On the echocardiography performed on the fifth day, ejection fraction was 45%. The patient was discharged from our clinic after total of 5 days of follow-up.

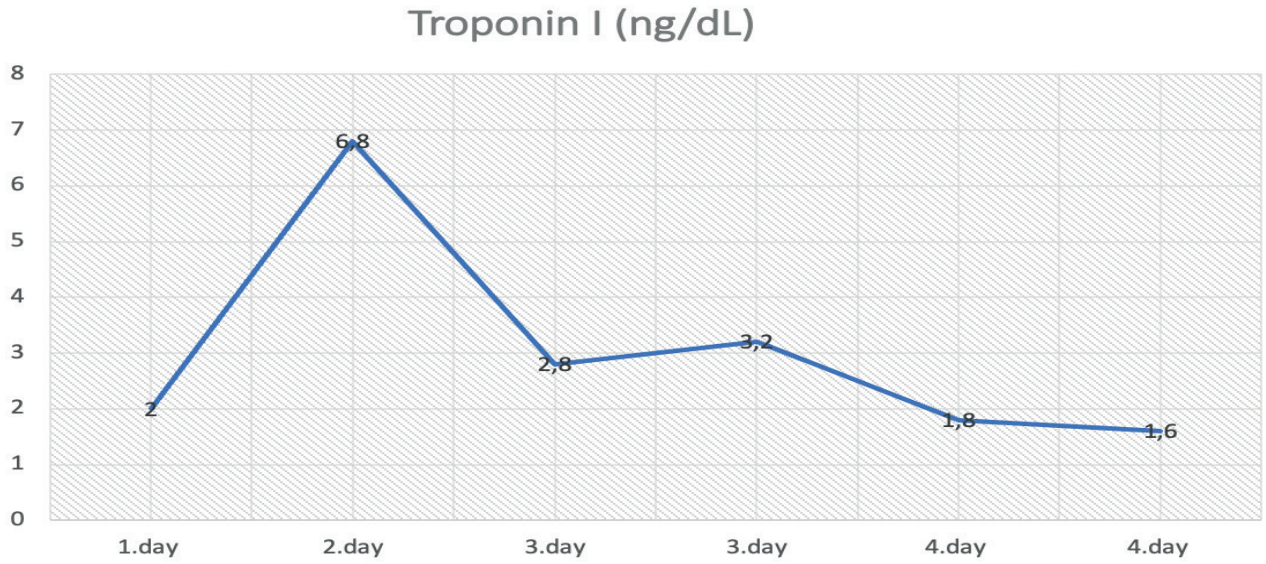

Figure: The patient's troponin I levels

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Cite as: Özdemir S, Alper B, Alp H, Aksel G, Doğanay F. Carbon Monoxide Poisoning and Sequels of Cardiac Function. Phnx Med J. 2022;4(2):94-95.
Received: .04.12.2021 **Accepted:** 03.02.2022

Conflict of interest: Authors declare no conflict of interest.
Ethic: The study does not require ethics committee approval. There is no such thing as any blood, saliva, violation of the rights of the patient, etc.
Case was presented as a poster presentation in 4th Intercontinental Emergency Medicine Congress, 4rd International Critical Care and Emergency Medicine Congress, at May 18- 21, 2017, in Antalya

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