A case of first degree AV block in carbon monoxide poisoning patient

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Carbon monoxide (CO) intoxication is one of the most common types of poisoning. Cardiac manifestations of CO poisoning are limited to case reports of Electrocardiogram (ECG) changes, myocardial dysfunction and myocardial ischemia.

A 22 year old Saudi male was admitted to the emergency department with decreased level of consciousness after being involved in a fire at home. ECG showed first degree AV block. To the best of our knowledge, this case report is the first CO poisoning case in the literature presented with first degree AV block.

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Introduction

Carbon monoxide (CO) intoxication is one of the most common types of poisoning and the leading cause of death by poisoning in the United States. CO is a tasteless, odorless, and nonirritating but highly toxic gas [1,2]. Sources of CO poisoning include any source of combustion such as motor vehicle exhaust fumes, charcoal and poorly functioning heating system. In Saudi Arabia, the main sources of CO poisoning are charcoal and motor gasoline [3–5]. Carbon monoxide mediated toxicity results from number of factors. CO binds to hemoglobin with affinity 200–250 times that of oxygen which results in competitive binding to hemoglobin, reducing oxygen delivery leading to profound tissue hypoxia. In vitro, CO binds to cytochrome-C oxidase of the electron transport chain resulting in asphyxiation at the cellular level. These mechanisms are believed to cause neurological and myocardial injury [2,3,6,7].

The clinical symptoms of CO poisoning are non-specific and can mimic variety of common disorders. The severity ranges from mild flu-like symptoms to coma and death. Because of their higher metabolic rate, the brain and the heart are the most susceptible organs to CO toxicity. Description of the cardiovascular...
lar manifestations of Co poisoning in humans have been limited to case reports [1,3,4].

Case report

A 22 year old Saudi male, who is a smoker, brought to ER by an ambulance with decreased level of consciousness. He was involved in a fire at home. After rescuing his family members he collapsed inside his home and was rescued later by the firemen. On arrival to ER he was found to have signs of inhalation injury and encephalopathy with a Glasgow coma scale (GCS) of 5/15. The pupils were equally reactive. He had normal equal breath sounds bilaterally with coarse crackles. No signs of burns have been noticed. He was intubated because of low GCS. Initial ECG revealed first degree AV block with heart rate of 87 beats per minute and PR interval was 208 ms (Fig. 1). The PR interval in the third ECG done 6 h later was 244 ms (Fig. 2). Subsequent ECG revealed sinus bradycardia (Fig. 3). Creatinin kinase (CPK) was 1119 IU/L (60–400) on arrival, however, troponin I was normal. Complete blood count (CBC), biochemistry and electrolytes were normal. The arterial blood gas (ABG) showed carboxyhemoglobin (COHgb) level of 8.7% and hyperbaric oxygen therapy was started. Within 24 h, the patient was extubated and COHgb level returned to normal range within 2 days. The first degree AV block and bradycardia had recovered, and ECG returned to normal (Fig. 4). Creatinin kinase also returned to normal level. Multidisciplinary team was involved in this patient management including internal medicine, Intensive care unit, cardiology, pulmonology and neurology. Patient was given appointment for echocardiography 2 weeks after discharge for follow up but unfortunately he did not show up.

Figure 1.
Discussion

Available literature on cardiovascular consequences of CO poisoning is limited to isolated case reports of ECG changes, myocardial dysfunction and myocardial ischemia [3]. Patients with underlying cardiac conditions are at risk of death from arrhythmias and fatal heart attacks. However myocardial ischemia can occur without underlying coronary artery disease [1,7]. Patients who are young with few cardiac risk factors are more likely to have stunned myocardium secondary to CO poisoning and global left ventricular dysfunction that is usually transient. In older patients with cardiac risk factors, CO poisoning tends to unmask underlying coronary artery disease by creating supply demand mismatch [3].

The most important factors for myocardial injury or dysfunction in patients with CO poisoning are carboxyhemoglobin level and duration of CO exposure. Usually cardiac abnormalities recover rapidly but permanent or persistent damage may rarely occur [8]. Myocardial injury in CO poisoning can be demonstrated by elevated cardiac biomarkers and ischemic ECG changes. Decreased left ventricular function, right ventricular dysfunction and arrhythmias can be observed in patients with CO poisoning [9]. One local study looked at complications of CO poisoning showed that cardiac arrhythmias presented in 29% of patients [5]. However, we did not find a case of first degree AV block induced by CO poisoning in the literature. Although myocardial ischemia may be associated with arrhythmias such as AV block, however, in this patient,
it is also possible that CO poisoning could have induced autonomic dysfunction that lead to increased vagal tone and it is well known that increasing vagal tone can prolong atrioventricular node conduction time and ultimately produce full AV block [10].

Myocardial injury is a common consequence of CO poisoning causing increased risk of mortality therefore, patients with CO poisoning should be screened for myocardial injury [2,11]. Henry et al., studied mortality risk in patients with moderate to severe CO poisoning and found that among 85 patients who sustained myocardial injury from CO poisoning, 38% were dead within 7.6 years [2].

In a study done by Satran et al., myocardial injury with ischemic ECG changes were found in 37% of 230 patients with moderate to severe CO poisoning with 5% in-hospital mortality, therefore patients admitted to the hospital with CO poisoning should have a baseline ECG, serial cardiac biomarkers and patients with abnormal cardiac biomarkers should have an echocardiogram [1,3].

Tissue hypoxia is the major outcome of CO intoxication, therefore Oxygen is the natural antidote and is considered the cornerstone of therapy. Hyperbaric oxygen therapy is the treatment of choice for patients presenting with syncope, coma, seizure, focal neurological deficit, arrhythmias or COHgb level more than 25% (15% in pregnancy) [1,3].

Although first degree AV block in this patient resolved, permanent or persistent myocardial damage may have occurred. Myocardial stunning may be responsible for cardiac dysfunction [8]. Interestingly that the natural history of first degree AV block is not as benign as previously believed, therefore additional follow up with routine echocardiogram is needed [12].
Conclusion

To our knowledge, this is the first reported case in the literature of carbon monoxide poisoning found to have first degree AV block that resolved after treatment of CO intoxication.

Myocardial injury and arrhythmias including different degrees of AV block should be suspected in patients with CO poisoning and these patients should have serial ECG and cardiac biomarkers.

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References