Biochemical risk assessment and invasive strategies for acute coronary syndromes without ST-segment elevation

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Citation for published version (APA):

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Pitfalls in cardiology: myocardial ischaemia in carbon monoxide poisoning

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Neth Heart J. 2006;14:183-6
ABSTRACT

Carbon monoxide poisoning is a major cause of morbidity and mortality worldwide. Immediate and proper treatment is crucial for prognosis and is merely dependent on accurate diagnosis. However, correct diagnosis can be difficult due to the aspecific symptomatology in CO poisoning. In this report a case is discussed in which a patient with accidental CO poisoning presented primarily with syncope, extensive ECG changes and elevated troponin levels. In addition, a limited review of the current literature is provided.
INTRODUCTION

One hundred and ten years ago, the British physiologist and philosopher Haldane (1860-1936) published a report on the causes of death resulting from mine explosions and fires. In this article, the action of gases that caused suffocation and the pathological effects of carbon monoxide (CO) present after a mine explosion were described.

Nowadays, despite the great care taken to minimise CO exhaustion, accidental CO poisoning is still a common and major cause of illness and death, on a worldwide scale. 2

Although it is generally known that CO poisoning can cause myocardial ischemia, physicians can be misled by a seemingly nonspecific presentation. 3, 5

The following case report presents a patient admitted with episodes of syncope combined with extensive changes on the ECG and elevated troponin serum levels.

Case report

A 26-year-old Caucasian male presented with syncope. In the hours before presentation he had fainted several times, with complete loss of consciousness. The duration of these episodes was unknown. The patient complained of nausea, had vomited, and apparently been incontinent of urine during one of the episodes. There were no anginal symptoms, nor did he experience palpitations. He did not smoke, use alcohol or drugs.

On physical examination the blood pressure and temperature were within normal range, but the pulse rate was high, exceeding 100 beats/min. No abnormalities were found on auscultation of the heart and lungs or examination of the abdomen and extremities.

The ECG showed a sinus tachycardia, normal QRS and inverted T-waves in leads II, III, aVF and lead V3-V6 (figure 1). Additional laboratory testing showed an elevated troponin I serum level of 0.97 ug/L (reference: <0.48 ug/L) with normal CK and CK-MB concentrations. Further analyses revealed a highly elevated carboxyhaemoglobin level of 25.5% by co-oximetry (reference: <1.5% in nonsmokers) of an arterial gas sample with an oxyhaemoglobin level of 73%.
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FIGURE 1
ECG on admission, showing sinus tachycardia and negative T waves in leads II, III, aVF and lead V3-V6.

The patient was admitted to the coronary care unit and treated with 100% oxygen by high-flow mask. No additional medication was given. No further syncope or arrhythmias were observed.

Echocardiography showed a normal cardiac function without any regional left or right ventricular wall motion abnormalities. An ECG taken 12 hours after admittance had normalised (figure 2). Repeated arterial blood sampling at this time showed no remaining evidence of carbon monoxide.

Additional neurological screening, including computed tomography of the brain, showed no evidence for a neurological cause of syncope and ruled out a cerebral cause of the ECG abnormalities. The patient quickly recovered. A bicycle exercise test one day later was normal.

The fire department discovered a poorly functioning heating system in the patient’s appartement as the cause for the CO poisoning. The heater was dismantled and replaced.
DISCUSSION

The presentation and symptoms of patients with CO poisoning are frequently nonspecific and often lead to a misdiagnosis resembling viral illnesses. As a result, the incidence of acute CO poisoning is most likely to be underestimated. Although central nervous system findings usually dominate the clinical presentation, significant and occasionally fatal cardiac abnormalities have been reported. In a recent registry involving household-related CO poisoning the mortality rate was 2.6% (20/811).

As CO has a higher affinity for haemoglobin than oxygen, tissue oxygenation is impaired, with the central nervous system and the heart being the most susceptible target organs due to their high oxygen needs. While the cardiac toxicity may be partially explained by myocardial hypoxia, a direct toxic effect of the gas on myocardial mitochondria could play an even greater role.

Among haemodynamic changes occurring after acute CO exposure, tachycardia is a common finding, usually considered as a compensatory response to systemic hypoxia and cardiac dysfunction.
ECG abnormalities have frequently been observed with acute CO intoxication in humans and animals. These include disorders of impulse formation, such as premature atrial and ventricular complexes, atrial fibrillation as well as ventricular fibrillation. In addition, disorders of impulse conduction have been described. Atrioventricular dissociation and lesser degrees of atrioventricular heart block as well as infranodal block have been observed in acute CO intoxication. Repolarisation disturbances in acute CO poisoning include ischaemic ST-T changes and QT interval prolongation. However, no clear correlation has been found between carboxyhaemoglobin levels and ECG alterations. The pathological substrate of myocardial toxicity from CO intoxication is characterised by areas of necrosis, haemorrhages, and muscle fibre degeneration scattered in the subendocardial region, especially in the septum and papillary muscle. Conventional markers of myocardial necrosis (CK-MB, aspartate aminotransferase, lactate dehydrogenase), possess low specificity as an indicator of cardiac necrosis in patients with conditions such as skeletal muscle necrosis or multiple organ failure, which may occur in patients with severe CO intoxication. Therefore, more specific markers such as troponin T and I have successfully been utilised for the diagnosis of CO-related myocardial necrosis. The clinical use of cardiac neurohormones, such as brain natriuretic peptide (BNP) and its splitting product NT-proBNP, has yet to be evaluated in CO poisoning. However, it seems likely that in response to the ischemia caused by CO intoxication, BNP and NT-proBNP will be released into the circulation. As in acute coronary syndrome, BNP and NT-proBNP may even prove to be a superior risk predictor than troponin in CO intoxication. As the mortality and morbidity in CO intoxication are considerable, early diagnosis and treatment is crucial. While pulse oximetry cannot distinguish between carboxy- and oxy-haemoglobin, peripheral saturation is most likely to be normal in CO poisoning. Co-oximetry analysis of arterial blood should be the method of choice in determining the presence of potential life-threatening levels of carboxyhaemoglobin. Prompt treatment with 100% oxygen by a high-flow device should be initiated when the diagnosis of CO poisoning is suspected. In severe cases, treatment with hyperbaric oxygen can be considered. High levels of oxygen decrease the CO half-life and thereby prevent further damage.
Conclusion

CO poisoning should be considered when signs of cardiac ischemia are accompanied with aggravated constitutional symptoms such as malaise, nausea, vomiting and/or neurological symptoms. Furthermore, a thorough cardiac evaluation should to be performed in patients presented with evidence of CO intoxication, as CO-induced myocardial necrosis can occur in the absence of cardiac symptoms.
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