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CASE REPORT

What do we know about carbon monoxide poisoning and cardiac compromise? Treatment and prognosis☆

Rosa Cardiga*, **Margarida Proença, Carolina Carvalho, Luís Costa, Arturo Botella, Filipa Marques, Carolina Paulino, António Carvalho, Cândida Fonseca**

Serviço de Medicina III, Hospital São Francisco Xavier, Centro Hospitalar de Lisboa Ocidental, Faculdade de Ciências Médicas, Universidade Nova de Lisboa, Lisboa, Portugal

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Abstract Carbon monoxide (CO) poisoning is one of the most common types of poisoning and the leading cause of death by poisoning worldwide. Cardiac injury caused by CO poisoning has been little described despite being a predictor of poor prognosis.

We present the case of a healthy 24-year-old woman, admitted to our emergency room due to an episode of lipothymia without loss of consciousness. She reported holocranial headache for the previous two weeks associated with nausea and vomiting.

Laboratory tests revealed blood gas analysis: pH 7.392, pCO_2 32 mmHg, pO_2 101 mmHg, lactate 3.5 mmol/l, HCO_3 20.8 mmol/l; COHb 29.2%; serial troponin I 1.21→5.25→6.13→3.65 μg/l; myoglobin 1378→964→352 μg/l; and NT-proBNP 1330 pg/l. The electrocardiogram showed sinus rhythm, heart rate 110 bpm, and ST-segment depression of 2 mm in V4 and 1 mm in V5. Transthoracic echocardiography revealed a left ventricle with normal wall motion and preserved ejection fraction.

Given the clinical and epidemiological context, myocardial and central nervous system ischemia due to prolonged CO exposure was assumed and normobaric oxygen therapy was immediately started. In view of evidence of injury to two major organ systems the indication for hyperbaric oxygen therapy was discussed with a specialist colleague, who suggested maintaining conservative treatment with oxygen therapy and in-hospital monitoring for 72 h. The patient was discharged on the third day and was still asymptomatic at 400 days of follow-up.

Besides symptoms and signs of central nervous system dysfunction, myocardial damage should also always be considered in the context of CO poisoning. Hyperbaric therapy is still controversial and the lack of objective data highlights the need for new randomized studies.

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* Corresponding author.
E-mail address: rosita.cardiga@gmail.com (R. Cardiga).

PALAVRAS-CHAVE
Intoxicação por monóxido de carbono;
Isquemia do miocárdio;
Oxigénio hiperbárico

Intoxicação por monóxido de carbono com compromisso cardíaco: o que sabemos?

Resumo A intoxicação por monóxido de carbono (MC) é um dos tipos de intoxicação mais frequente e a principal causa de morte por intoxicação no mundo. A lesão cardíaca provocada pela intoxicação por MC tem sido pouco descrita apesar de ser um fator preditor de mau prognóstico.

Apresentamos o caso de uma mulher de 24 anos, saudável, que recorre ao serviço de urgência por lipotimia sem perda de conhecimento. Referia cefaleia holocraniana associada a náuseas e vómitos com duas semanas de evolução. Analiticamente: gasimetria pH 7,392; pCO_2 32 mmHg, pO_2 101 mmHg, lactatos 3,5 mmol/L, HCO_3 20,8 mmol/L, COHb 29,2%; troponina I seriada 1,21→5,25→6,13→3,65 ug/L, mioglobina 1378→964→352 ug/L, NT-ProBNP 1330 pg/L. Eletrocardiograma: ritmo sinusal, frequência cardíaca 110 bpm, depressão do segmento ST de 2 mm em V4 e 1 mm em V5. Ecocardiograma transtorácico: ventrículo esquerdo sem alterações da cinética segmentar e boa função sistólica global.

Associando-se a clínica ao contexto epidemiológico assumiu-se isquemia do miocárdio e do sistema nervoso central por exposição prolongada ao MC. Iniciou-se prontamente oxigenoterapia normobárica. Perante a evidência de compromisso de dois órgãos nobres, foi discutida com o colega especialista nesta área a indicação para tratamento com oxigénio hiperbárico, sugeriu manter tratamento conservador com oxigenoterapia e vigilância em internamento durante 72 h. Teve alta ao terceiro dia e mantém-se assintomática aos 400 dias de follow-up.

Tal como os sintomas e sinais de disfunção do sistema nervoso central, a lesão do miocárdio deve ser sistematicamente equacionada no contexto de intoxicação por MC. A indicação para terapêutica em câmara hiperbárica permanece controversa e com indicações pouco objetivas, a apelar a novos estudos aleatorizados.

Introduction

Carbon monoxide (CO) is a colorless, tasteless, odorless and non-irritant but highly toxic gas.[1] It binds rapidly to hemoglobin with an affinity 210 times higher than for oxygen, leading to the formation of carboxyhemoglobin (COHb) and tissue hypoxia.

CO poisoning is one of the most common types of poisoning; it is a frequent cause of morbidity,[2] and the leading cause of death by poisoning worldwide.[3]

While not a common cause of hospital admission in Portugal, its incidence is not negligible: 5.86/100 000 population over an eight-year period.[4]

The clinical symptoms of CO poisoning are non-specific and can mimic a wide range of conditions, and diagnosis requires a high index of suspicion.[1]

Headache is the most common symptom of CO poisoning, reported in 84% of cases, and 50% of patients present fatigue, nausea, mental confusion and dyspnea.[1] Less frequent symptoms include abdominal pain, visual disturbances, chest pain and seizures.[1]

Tissue hypoxia in CO poisoning affects all organs and systems, and involvement of the major organs worsens prognosis.

The neurological sequelae of CO poisoning have been amply described in the literature, but cardiovascular repercussions have only been presented in occasional case reports.[5,6]

Cardiac injury results from the binding of CO to hemoglobin and myoglobin and inhibition of mitochondrial cytochrome oxidase, resulting in tissue hypoxia and reduced adenosine triphosphate synthesis.[7]

The most common cardiac manifestations are ischemia leading to elevated myocardial necrosis markers, electrocardiographic (ECG) alterations, particularly ST-segment depression, myocardial infarction and conduction disturbances such as atrial fibrillation and ventricular arrhythmias.[8]

The authors present a case of CO poisoning with cardiac compromise with the aim of highlighting the seriousness of this complication, which is frequently overlooked, and the need for thorough investigation and monitoring in all cases of CO poisoning. The indication for hyperbaric therapy is also discussed in this context.

Case report

A 27-year-old woman, with no relevant clinical history or cardiovascular risk factors and not taking any medication, was admitted to the emergency room (ER) due to an episode of lipothymia, during which she was unable to move for around two hours following a bath. She denied loss of consciousness, tonic–clonic movements, loss of sphincter control, chest pain or palpitations. She reported holocranial headache for the previous two weeks, which she quantified as 6 on a scale of 1 to 10, associated with nausea and vomiting.[9]

From an epidemiological standpoint, the patient was accompanied by two other women who lived in the same