

# Carbon Monoxide and Cyanide Intoxication: An Association to Remember

## Intoxicação por Monóxido de Carbono e Cianeto: Uma Associação a Recordar



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### ABSTRACT

Carbon monoxide and cyanide are toxins that induce cellular hypoxia; both can be produced in the context of domestic fires and may have a synergistic effect. We present the case of a man, victim of a house fire, who was initially diagnosed as having carbon monoxide poisoning, but that afterwards also presented signs and symptoms compatible with cyanide poisoning. He was successfully treated with an antidote. We want to highlight this relatively frequent association of poisonings and the need for urgent empirical treatment.

**Keywords:** Carbon Monoxide Poisoning/therapy; Cyanides/poisoning

### RESUMO

O monóxido de carbono e o cianeto são toxinas que induzem hipóxia celular; ambos podem ser produzidos em contexto de incêndios domésticos e podem exercer um efeito tóxico sinérgico. Apresentamos o caso de um homem, vítima de incêndio doméstico, que foi inicialmente diagnosticado como intoxicação por monóxido de carbono, mas que posteriormente apresentou também sinais e sintomas compatíveis com intoxicação por cianeto, tendo sido tratado com sucesso com antídotos dirigidos. Pretendemos alertar para esta associação de intoxicações relativamente frequente e para a necessidade de tratamento empírico urgente.

**Palavras-chave:** Cianeto/intoxicação; Intoxicação por Monóxido de Carbono/tratamento

### INTRODUCTION

Carbon monoxide (CO) and cyanide (CN) are toxins that have in common the ability to bind iron ions. This common characteristic means that they interfere with the oxygen (O<sub>2</sub>) carrying capacity of blood and/or with aerobic cellular respiration, leading to cellular hypoxia and eventually the death of the intoxicated individual; when combined, they may have a synergic effect.<sup>1-3</sup>

CO poisoning is common in victims of fires. The role of CN intoxication is less known, perhaps because this intoxication is relatively rarer. CN may be produced from several sources, the most frequent being the combustion of synthetic materials like plastic, foam and paint, and some natural fibers such as paper, wool and silk; as such, CN may play an important role in the morbimortality of victims of enclosed space fires, such as houses or cars.<sup>1,3,5</sup>

The early diagnosis of CN intoxication is vital, because there are life-saving antidotes, such as hydroxocobalamin, which can be used.

### CLINICAL CASE

We describe the case of a 78-year-old male, without relevant previous history, who was a victim of a house fire. Assisted at the scene by the emergency team (VMER - *Viatura Médica de Emergência e Reanimação*) who identified mental status change with a Glasgow coma scale of 10, and signs of possible airway burn (burnt nasal cilia and eyebrows, lip edema and soot in the tongue and airway). He was intubated at the scene and transported to our emergency room (ER).

On admission to the ER the patient was sedated and the airway burn was confirmed through videolaryngoscopy, which showed edema of the glottis, soot in the airway and erythema of all the structures. Despite remaining hemodynamically stable, he had mottled skin, prolonged capillary refill time and increased lactate level of 3.8 mmol/L. His blood work showed slight rhabdomyolysis, with creatinine kinase of 271 U/L (reference values (RV) 30 - 200 U/L) and myoglobin of 626 ng/mL (RV < 146.9 ng/mL), and carboxy-hemoglobin (COHb) of 18.7% (reference interval in non-smokers 1% - 3%)<sup>6</sup>; the electrocardiogram and brain computerized tomography showed no relevant changes.

He was diagnosed with CO intoxication and hyperbaric oxygen therapy (HBO) was prescribed (two sessions of 90 minutes each, separated by 15 hours, with 100% O<sub>2</sub> at the pressure of 2.5 absolute atmospheres), due to the mental status change at presentation, according to current guidelines.<sup>7</sup>

The patient was admitted to the intensive care unit (ICU), where he evolved favorably in the first hours, with resolution of the hyperlactacidemia, being extubated after 48 hours. In the third day of admission he developed agitation, hypertension, tachycardia, facial flushing and a resurgence of the hiperlactacidemia, without an obvious cause. Because of this we raised the hypothesis of concomitant intoxication of other inhaled substances, namely CN, and decided to start empirical treatment with single dose, intravenous hydroxocobalamin 5 g and sodium thiosulphate 12.5 g. The patient presented complete resolution of all symptoms and signs

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and normalization of the arterial lactate; he developed generalized red-orange erythema and chromaturia, both benign side effects of the intravenous injection of hydroxocobalamin.

During his admission in the ICU, the patient also presented chemical pneumonitis, without need for reintubation, and delirium, solved after 24 hours of dexmedetomidine perfusion and benzodiazepine prescription.

Afterwards he had a favorable evolution and was discharged to the general ward after five days.

## DISCUSSION

Acute poisoning by CN has non-specific symptoms and signs, usually with an early presentation, many of which are common to CO intoxication.<sup>1</sup> Contrary to CO intoxication, which can be easily diagnosed through the concentration of COHb, the determination of CN blood levels, while possible, is complicated by the instability of the molecule and the delay and unavailability of the tests in most laboratories.<sup>1,5,8</sup> As such, many patients with simultaneous intoxication may go unnoticed, demanding a high level of clinical suspicion in the contexts where CN exposure is likely.<sup>1,4,5</sup>

CN links non-competitively to the cytochrome-c oxidase, the last enzyme in the electron transport chain, stopping it and leading to the exclusively anaerobic production of adenosine triphosphate. A severe lactic acidosis is, therefore, expected and serum lactate levels are useful to corroborate the diagnosis of intoxication.<sup>3,9</sup> Lawson-Smith *et al* propose that CN intoxication be suspected in every patient with context (exposure to a fire and with smoke inhalation injuries) who have two or more of the following criteria: neurologic dysfunction, soot in the mouth or phlegm and arterial blood sampling with metabolic acidosis with a lactate level above 8 mmol/L.<sup>5</sup>

The proposed treatment for CN intoxication is based on decontamination, resuscitation and support.<sup>2</sup> There are many possible antidotes available, with hydroxocobalamin being the most frequently used in Europe, due to its favorable safety profile; some authors suggest its empirical use in pre-hospital and emergency settings, in cases of high clinical suspicion.<sup>2,8</sup> Sodium thiosulphate has an equally favorable safety profile, but a slower onset of action, which is the

reason why its use as a single agent is not usually recommended; its association with hydroxocobalamin is not completely established but has biological plausibility, since both act at different points in the CN metabolism and may have a synergistic effect; some published cases have showed good results.<sup>10</sup>

In the presented case, the context of domestic fire and airway burn, combined with the initial mental status changes and soot in the airway should have raised the suspicion of CN intoxication which was, however, not raised. We believe that the later and less severe presentation could be explained by the use of HBO to treat the CO poisoning, since HBO seems to mobilize CN from the tissue to the circulation, facilitating its metabolism and excretion.<sup>11</sup> HBO is not currently recommended as therapeutic option for CN poisoning.

With this clinical case we intend to raise awareness of this association of intoxications and make clinicians ponder the early and empirical treatment with the antidote in cases of domestic fire victims with red-flag symptoms.

## PROTECTION OF HUMANS AND ANIMALS

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and to the Helsinki Declaration of the World Medical Association.

## DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

## PATIENT CONSENT

Obtained.

## CONFLICTS OF INTEREST

All authors report no conflict of interest.

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