

Chronic carbon monoxide poisoning. A report of two cases

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Abstract

If acute carbon monoxide poisoning is a well-known emergency situation, this is not the case for chronic poisoning. The missed diagnosis of acute CO poisoning is a well-known problem but the identification of a chronic poisoning is very challenging. Knowledge and awareness of chronic poisoning is less defined and probably there is a great number of patients with undiagnosed chronic CO poisoning. It is possible that in case of missed diagnosis because of non-specific symptoms, chronic CO poisoning could be responsible for significant morbidity. We describe the case of a married couple who were rescued almost simultaneously, to show this clinical condition.

Introduction

If acute carbon monoxide poisoning is a well-known emergency situation,^{1,2} this is not the case for chronic poisoning.³ The latter has a very variable spectrum of symptoms, often less overt and consequently more difficult to report, because they are not always recognizable.³⁻⁵ It is likely that chronic carbon monoxide intoxication is much more frequent than one might imagine, and

that it can sometimes escape even the most experienced clinicians. Here we describe the case of a married couple who were rescued almost simultaneously. They suffered from different symptoms, which could have deceived a doctor and delayed the diagnosis.

Case Report

The first patient was a 44-years-old man, with a silent history, taken to our Emergency Department at night because of diarrhoea, fatigue, and mildly diffused chest pain. He was alert, without fever and he had normal vital signs (150/80 mmHg arterial pressure, 90 bpm, 99% arterial saturation) and a normal ECG (Figure 1a). His neurological physical assessment was normal and the only important objective element was his skin, which appeared to be extensively reddened (particularly his face, chest and both hands). Along with this patient, we evaluated his wife, a 40-years-old woman without a significant past medical history: she referred recent onset of toothache, diffuse arthralgia, non-specific chest pain. Also, her vital parameters were normal (120/60 mmHg arterial pressure, 98 bpm, 99% arterial saturation), but she appeared confused even in the absence of signs of neurological deficit, fever and other objectives signs. Her ECG was also normal (Figure 1b).

In both cases the triage nurses were alerted by the smell of burnt wood, and decided to perform a venous blood gas examination: in the man, the carbon monoxide level resulted normal (3.3%), while in his wife we detected a slight but significant increase (8.8%, in no smoker woman). The other parameters were normal.

In fact, they lived in an unattended country house, and used an unspecified wood stove for heating, so despite not particularly high carbon monoxide values, chronic exposure seemed plausible. They were both immediately treated with 100% O₂ non-rebreathing masks.

Blood tests were normal except for a high level of cardiac Troponin I: 987 ng/mL in the man, and 1087 in the woman. Cardiac ultrasound, carried out following the discovery of elevated troponin values, showed no abnormalities in cardiac contractility or morphology. After a consultation with the specialist of the bariatric center of the referring hospital, considering the cardiac and neurological involvements, both patients were transferred to start hyperbaric treatment.

They were treated for five days, with a progressive resolution of their clinical conditions, and then discharged to their home. An inspection from fire workers in their home found an irregular indoor home heating system with CO leak.

Discussion

CO is a colorless, odorless, non-irritating gas produced by incomplete combustion of any fuel, due to poor oxygenation of fire.^{1,2} The pathophysiology of carbon monoxide poisoning is complex and not completely known, in particular in chronic, occult

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poisoning.³ We can consider different pathophysiological aspects: i) the high affinity of carbon monoxide to haemoglobin (up to 300 times than oxygen), and its stability, lead to peripheral tissue hypoxia in the whole body;^{1,2} ii) the carbon monoxide can bind any kind of heme-containing proteins, like cytochromes, myoglobin, and guanylyl cyclase: CO can reduce cellular activity in different ways, and can induce direct myocardial toxicity;⁶⁻¹¹ iii) there are increasing suggestions of an important role of nitric oxide: NO level increases in CO exposure in animal model, with a reduction in brain flow and systemic hypotension;¹²⁻¹⁴

The missed diagnosis of acute CO poisoning is a well-known problem:¹⁵ some retrospective analysis in acute coronary syndrome or ischemic stroke showed a prevalence of undiagnosed CO poisoning in up to 30% patients,^{11,16} and a strategy to identify any suspected case in the ED is mandatory;^{1,3,15} but the identification of a chronic poisoning is very challenging:^{3,15} knowledge and awareness of chronic poisoning is less defined and probably there is a great number of patients with undiagnosed chronic CO poisoning.

It is possible that chronic CO poisoning is responsible for significant morbidity, especially if diagnosis is not reached because of non-specific symptoms, and some particular issues.^{17,18}

First of all, there is few evidence in literature, and there is no defined role in CO levels in blood sample:¹ in our patients, only the female had a slight increase in CO levels, similar to a level considered normal for smoker. The male had a normal level of CO, and he had no marked neurological complaints. There is no agreement about the exact pathophysiology in chronic, occult poisoning: probably, a continuous, submaximal exposure could lead to cellular bindings, with severe neurological and cardiac toxicity.^{2-5,10,11}

For both patients, we excluded other causes that could lead to their symptom through a careful evaluation. Surely the alteration in both spouses of troponin levels could suggest the diagnosis of Takotsubo syndrome, but we underline that the chest pain was undefined and not typical in both, electrocardiograms were inexpressive and cardiac ultrasound did not show the typical pattern. Moreover, their histories did not show a possible stressful event as

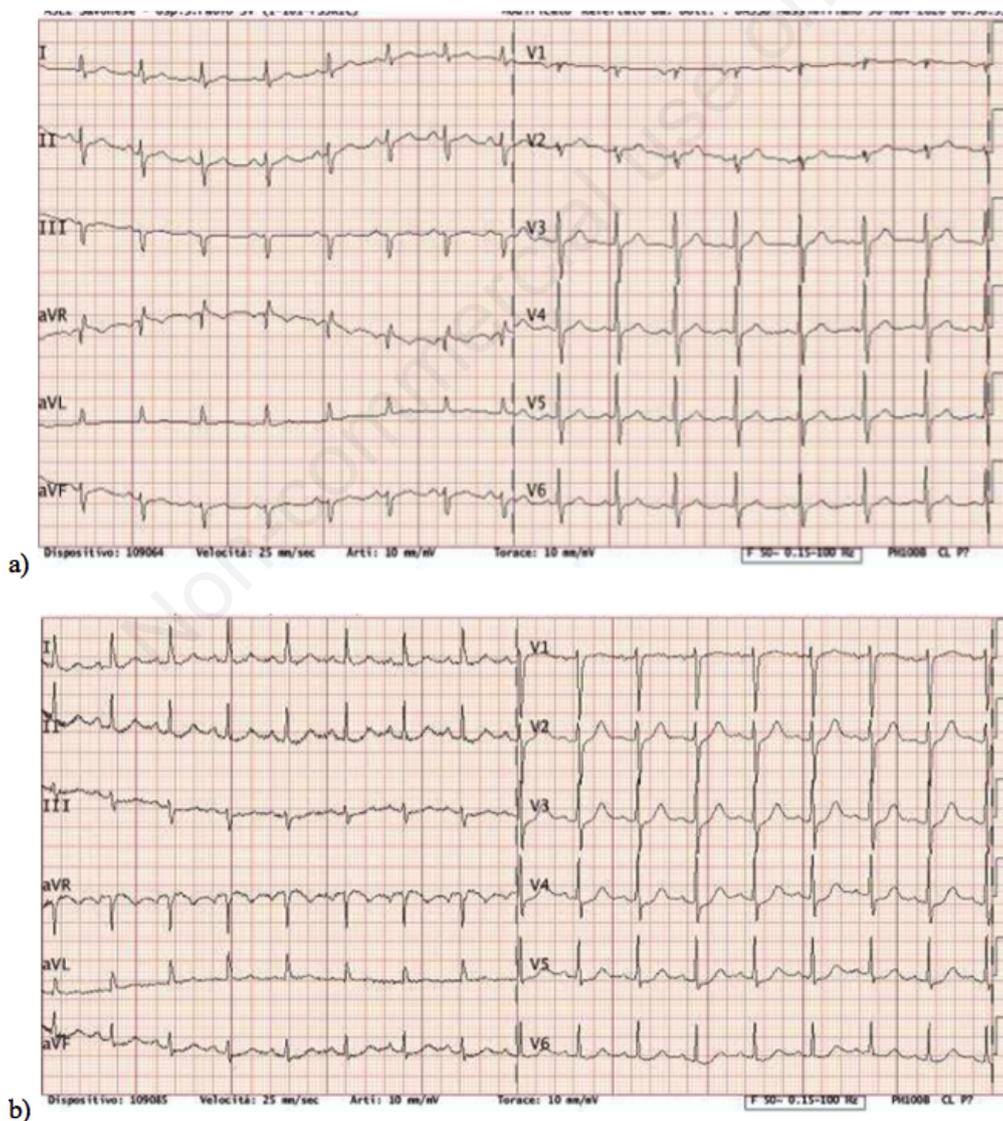


Figure 1. The EKG of our patients: a) the male patient, b) the female patient.

it is often described in Takotsubo syndrome. The hyperbaric treatment was immediately started and showed a progressive and dramatic improvement in neurological symptoms and reduction of troponin levels. This further supports the diagnosis of chronic CO poisoning.

But how is it possible to suspect a CO poisoning in patients with normal CO levels?

A high index of suspicion is very important.^{1,3} First of all, a cluster of patients living together sharing the same neurological or cardiological symptoms, must lead to a suspected CO poisoning. Secondly, if present, the typical skin coloration (see Figure 2, as in our male patient) is a possible indicator, although is emerged in post-mortem cases. Further, an evaluation of the type of domestic heating is important, although not always clear.

In our cases, the evaluation of both patients, with acute cardiac injury in both of them led us to the correct diagnosis. But what would happened if the patients had been evaluated at different times and by different physicians?

The cluster of symptoms in correlated patients or sharing the same for living space is mandatory: but in case of single patients, diagnosis could be missed.³

Conclusions

Is it possible to think of CO poisoning for all patients with an acute ischemic complaint evaluated in an ED? All authors agree that venous CO levels are as diagnostic as arterial ones,¹ but this could lead to an increase in sanitary costs without a real impact on health results. Probably the use of oxygen and carbon monoxide oximeter could make diagnosis of ED poisoning^{19,20} easier, but this is useless in case of chronic, severe exposure with normal or quite to normal CO blood levels.



Figure 2. Skin coloration of male patient.

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