ECRONICON OPEN ACCESS

EC CARDIOLOGY
Research Article

Monoxide Poisoning: Case Series of Myocardial Infarction

S Bellali*, J hassari, R Habbal, A El Khaoudi, R Hafiane and M Mouhaoui

Cardiology and Emergency Room Services, CHU Ibn Rochd, Casablanca, Morocco

***Corresponding Author:** S Bellali, Cardiology and Emergency Room Services, CHU Ibn Rochd, Casablanca, Morocco.

Received: April 29, 2019; **Published:** July 29, 2019

Abstract

Cardiac dysfunction including arrhythmias and myocardial ischemia have often been reported in carbon monoxide poisoning; Although the neurologic sequelae of CO poisoning have been well described, the cardiovascular consequences are limited to isolated case reports.

We conducted a two-year cross-sectional prospective study (December 2013 to December 2015), including all patients admitted to the emergency resuscitation department for symptomatic carbon monoxide poisoning. The severity of the intoxication was judged on hemodynamic, neurobiological and respiratory criteria.

18 patients were hospitalized for carbon monoxide poisoning. The average age is 38 years with a clear female predominance (sex ratio to 1/2). The main cardiovascular risk factors found are: hypertension (6 patients), type 2 diabetes (4 patients). CO intoxication was found to be severe in 14 patients (77.7%). ECG abnormalities were found in 55,5% patients (sinus tachycardia in 8patients, negative T waves in 6 patients, complete right limb block in 3patients, and ST depression in DII, DIII and aVF in 3 patient). Echocardiography found increased pulmonary hypertension (HTP) loading pressures in 4 patients, LV systolic dysfunction with segmental kinetic disorders in 6 patients, and cardiogenic shock in 4 patients. The troponin Ic was positive in 10 patients, i.e. 55.56% of the cases, with intervals between 0.87 ng/dl and 27.34 ng/dl. The occurrence of myocardial ischemia has been correlated mainly with young age and the severity of intoxication.

This study, confronted with review of the literature, focuses on the frequency of myocardial ischemia in the context of carbon monoxide poisoning.

Keywords: *Monoxide Poisoning; Myocardial Infarction*

Introduction

Acute CO intoxication is an important entity of great import to public health and remains a leading cause of morbidity and mortality [1]. It is the most common type of fatal air poisoning, and severe intoxication may cause seizure, coma, and death. it can be accidental or intentional (suicide attempt).

Carbon monoxide is a toxic gas that consists of one carbon and one oxygen atoms linked by two covalent bonds and one dative covalent bond, with no unpaired electrons (i.e. singlet state). This reactive chemical compound can form ligands with several minerals and ions, especially iron and copper. Carbon monoxide is an odorless, clear gas, with a density of 0.97 that of air. In most instances, carbon monoxide mixes evenly in turbulent air [2].

His symptomatology is very polymorphic, dominated by neuropsychological manifestations. Unfortunately, myocardial involvement in carbon monoxide poisoning; are underestimated; and has rarely been reported in relation to the large number of intoxications. Our work aims to focus on myocardial ischemia during carbon monoxide poisoning, through a series of patients admitted to the intensive care unit and emergency room of Ibn Rochd University Hospital in Casablanca.

Patients and Methods

We conducted a two-year cross-sectional prospective study (December 2013 to December 2015), including all patients admitted to the emergency resuscitation department for symptomatic carbon monoxide poisoning. The severity of the intoxication was judged on hemodynamic, neurobiological and respiratory criteria. All patients received an ECG, a chest x-ray, a trans-thoracic echocardiogram, and a Troponin Ic dose on admission. A value > 0.06 ng/l represents the threshold of positivity of this enzyme in the hospital laboratory. Clinical and para-clinical data were collected on a pre-established form.

Results

Over 2years of study, 18 patients were hospitalized for carbon monoxide poisoning. The average age is 38 years with a clear female predominance (sex ratio to 1/2). The main cardiovascular risk factors found are: hypertension (6 patients), type 2 diabetes (4 patients) and smoking (4 patients). CO intoxication was found to be severe in 14patients (77.7%). ECG abnormalities were found in 55,5% patients (sinus tachycardia in 8patients, negative T waves in 6 patients, complete right limb block in 3 patients and ST depression in DII, DIII and aVF in 3 patient). Chest X-ray showed hilar opacity of bilateral alveolar type suggestive of PAO in 4 patients. Echocardiography found increased pulmonary hypertension (HTP) loading pressures in 4 patients, LV systolic dysfunction with segmental kinetic disorders in 6 patients, and cardiogenic shock in 4 patients. The troponin Ic was positive in 10 patients, i.e. 55.56% of the cases, with intervals between 0.87 ng/dl and 27.34 ng/dl. The occurrence of myocardial ischemia has been correlated mainly with young age and the severity of intoxication. Death occurred in 1/3 of the patients and they all had acute myocardial ischemia judged on the positivity of the troponins.

Discussion

Epidemiology

Carbon monoxide poisoning is the leading cause of toxic death worldwide.

- In France, there are 5,000 to 8,000 intoxications per year, including 2,500 hospitalizations and 400 deaths, with a significant peak in winter [3].
- In Great Britain, 200 admissions for intoxications by CO [4].

Although rarely reported, myocardial ischemia is a common consequence of moderate to severe CO intoxication. In fact, a prospective American study of 230 patients showed that 37% of patients had myocardial infarction (judged by high levels of cardiac enzymes) [5]. This type of complication can be misdiagnosed in the emergency departments. Other studies have found that myocardial ischemia was more common in patients with common young age, elevated carboxyhaemoglobin (COHB), and altered state of consciousness upon admission. Risk factors for heart disease or coronary heart disease have been excluded [6].

In our study, Troponins Ic were positive in 55.56% of patients, this relative importance of the incidence of myocardial ischemia can be explained by the young age of patients, as well as the severity of CO intoxication.

Physiopathology

Carbon monoxide is an odorless, colorless gas with a strong affinity for 3 hemoproteins: haemoglobin, myoglobin and cytochrome a3 [7].

- The binding of CO to haemoglobin forms carboxyhaemoglobin HBCO which is unable to transport O_2 to tissues causing tissue hypoxia, with displacement to the left of the dissociation curve of Hb-O_2 [8].
- Its binding with myoglobin forms the carboxyhaemoglobin responsible for a decrease in cardiac output and worsening of tissue hypoxia [9].
- The fixation of CO to cytochrome a3 prevents the normal functioning of the mitochondrial respiratory chain, which leads to anaerobic metabolism showing lactic acidosis, therefore myocardial pain [10].

In addition, restoration of intracellular respiration after myocardial hypoxia may cause reperfusion injury through the production of free radicals which lead to myocardial sideration and cardiac dysfunction [11].

Myocytes are unable to contribute to effective contractility but continue to maintain metabolic viability. This explains the reversibility of myocardial ischemia after adequate management, and, in the same way, the absence of significant coronary lesion apart from true acute coronary syndrome with ST segment elevation [12].

Symptoms

During CO intoxication, myocardial ischemia often goes unnoticed explained by lack of specificity of cardiovascular symptoms. It is unfortunately diagnosed only in 25 to 30% of cases. Screening for myocardial infarction is often motivated by the presence of typical chest pain, or orientation symptoms [13].

In the various series published, the search for cardiovascular events was not systematic and the diagnosis of myocardial infarction was mainly motivated by the occurrence of dyspnoea and cardiogenic shock [14].

- Respiratory: Pulmonary edema occurs in 10 - 30% of cases and may be due to either hypoxia, direct effect of CO on alveolar membranes, left heart failure (myocardial injury), or inhalation secondary to consciousness disorders [10].
- Hemodynamically, it is difficult to differentiate between a cardiogenic and hypovolemic shock pattern that is usually due to the rhabdomyolysis often associated with CO intoxication, which highlights the value of performing an exam echocardiographic assay with Troponin dosing.

In our series, the search for cardiovascular events was systematic and dominated by:

- Tachycardia: 8 cases.
- Cardiogenic shock: 4 cases.
- Pulmonary edema: 4 cases.

Paraclinique

EKG

EKG abnormalities are frequently encountered after exposure to CO. These changes are often transient but may persist for days or weeks [15]. They are also non-specific but evocative.

The most common signs are: sinus tachycardia, flattening or inversion of the T wave, ST segment depression, and the occurrence of ventricular extrasystoles [16]. It has been shown experimentally that the ventricular fibrillation threshold becomes low after exposure to CO as explained by the heterogeneous repolarization of the ventricles, as assessed by the QT segment dispersion which has been shown to increase during CO poisoning [17]. This suggests close electrocardiographic monitoring of patients with ESV, given the considerable risk of sustained ventricular tachycardia.

In our series, the EKG showed 8 cases out of 18 sinus tachycardia, 6 cases of repolarization disorder judged on the inversion of the T wave, 3 cases of sub-shift of ST in inferior, and a 3cases of block right bronchus.

Chest X ray

The chest x-ray may show diffuse opacities in both lungs, related to pulmonary congestion [11]. In the context of neurological impairment, pulmonary pneumopathies may be revealed by chest x-ray.

In our series, the chest X-ray showed images in favor of pulmonary oedema in 4 patients.

Echocardiography

May show more or less significant alteration of left ventricular ejection fraction (LVEF), with global or segmental hypokinesia and moderate systolic dysfunction of the right ventricle. Signs of left ventricular overload with increased filling pressures and HTP/PAH may also be present [18].

These abnormalities are often rapidly reversible and the examination must be done early, especially if the oxygen therapy was started early.

A study of patients with myocardial involvement in CO poisoning distinguishes two groups: the first group includes young patients with a limited number of cardiovascular risk factors, but with severe intoxication (abnormal GCS), the TTE showed global ventricular dysfunction which can be explained by the presence of myocardial ischemia. In the second group, having an advanced age with cardiovascular factors, and 50% of whom have a normal GCS, the TTE illustrates hypokinesia, suggesting that CO masks the underlying coronary diseases by creating an incompatibility between the requests and the contributions of oxygen to the myocardium [19].

In our series, the TEE; which has been systematically performed in all patients; showed:

- LV systolic dysfunction with segmental kinetic disorders in 6 patients.
- Increased filling pressures with PAH in 4 patients.
- Low cardiac output in 4 patients.

The cardiac enzymes

The various studies carried out on patients suffering from CO intoxication, showed changes in serum levels of myocardial enzymes. Early dosing may be valuable in the diagnosis of myocardial involvement and remains reliable if performed in the days following intoxication [20]. However, their specificity is debatable depending on clinical signs and other organs affected.

The majority of authors agree to classify Troponin as the most specific marker of cardiac distress [21], this has been well demonstrated in a study of autopsies of CO poisoning victims, where Troponin has been elevated in cardiac and pericardial blood, with a close correlation with the histological lesions of diffuse cardiotoxicity [22]. The increase in other enzymes has been well documented in rhabdomyolysis following CO poisoning, with or without myocardial involvement [23].

In our series, the diagnosis of myocardial involvement was retained 55,5% on the troponin Ic level assay, performed systematically in all patients. The level of MB CPK is unreliable due to constant rhabdomyolysis with a greater increase in CPK.

Perfusion scintigraphy

The comparison of myocardial perfusion scintigraphy (99m Tc-MIBI SPECT) with the clinical, electrical and biochemical evaluation of necrosis, gave a significant value to this examination in the evaluation of myocardial ischemia during CO poisoning. The images obtained by this technique evaluate the locations and extension of the lesion, which makes this technique more sensitive than other methods used in assessing cardiac toxicity [24].

Despite its sensitivity, this examination is not necessary in the population without cardiovascular risk factors, or if the ECG and cardiac biomarkers are normal [16].

Coronary-Angiography

Coronary angiography is an invasive examination that remains the gold standard in the investigation of myocardial ischemia.

CO exposure promotes acute thrombotic events by fibrinogen binding [9], increased platelet clearance and polycythaemia, and promotes coronary vasospasm [25]. SCA can occur with CO intoxication with extreme myocardial infarction with ST elevation. However, in 80% of cases, myocardial ischemia caused by CO poisoning is mainly due to tissue hypoxia resulting in myocardial stunning; often reversible; apart from any anatomically significant injury.

Thus, the use of coronary angiography should be reserved for ST segment elevation SCA, and the occurrence of documented myocardial ischemia in a known stable coronary patient [26].

In our series, coronary angiography has not been performed. No cases of SCA ST + were reported, and no patients were known to be coronary.

Evolution and prognosis

The evolution of patients with cardiovascular complications is variable. Several studies have shown reversibility of symptoms and haemodynamic stabilization after resuscitation [27].

The delay in improving cardiac function after adequate treatment is estimated to be between one week and one month [14].

However, myocardial involvement is considered a predictor of long-term mortality in patients with moderate to severe CO poisoning [19]. by the considerable rate of mortality: 38% mortality in 85 patients with myocardial involvement compared to 15% in 145 patients without myocardial involvement [28].

For patients who survive after intoxication without improvement in left ventricular dysfunction, with underlying coronary disease or risk factors for coronary heart disease, it is desirable to refer them to the cardiologist for further assessment including angiography and revascularization [25].

Treatment

Management takes precedence over the removal of the source and the removal of the victim from the toxic atmosphere, without endangering the rescue team.

Oxygen therapy in these two forms: normobaric and hyperbaric; is recommended in emergency, allowing dissociation of carboxyhaemoglobin.

Normobaric oxygen therapy at 100% fiO_2 is always requested immediately for a minimum of 12 hours. It can be used by high concentration mask or mechanical ventilation [29].

Hyperbaric oxygen therapy is generally accepted as a treatment for moderate to severe CO intoxication, indications according to the American consensus include coma, loss of consciousness, COHB > 40%, signs of cardiac ischemia, arrhythmia, or any history of coronary disease with COHB > 20% [19].

Limited studies exist with regard to amelioration in cases of myocardial involvement under hyperbaric oxygen therapy and their prevention by this specific treatment [30].

In experimental, in dogs, put on OHB with or without thrombolytics, it was noted a limitation of the infarction following an occlusion of the coronary arteries, this beneficial effect seems to be dependent on the time elapsed for the use of the treatment: more the treatment is early, the better the result [19].

In CO intoxication with documented coronary ischemia, medical treatment of coronary syndrome should be combined with hyperbaric oxygen therapy, performed under electrocardiographic surveillance. In the absence of ST elevation, coronary angiography is delayed [31]

Conclusion

This study, confronted with review of the literature, focuses on the frequency of myocardial ischemia in the context of carbon monoxide poisoning.

Given the severity of the cardiac involvement, its silent character during moderate to severe CO intoxication, as well as the non-specificity of the electrocardiographic signs, recommending a systematic screening of the myocardial ischemia by the troponin Ic assay.

Although ACS can occur in the absence of underlying coronary artery disease, coronary angiography should be considered in view of ST elevation, as well as non-improvement of systolic function after adequate specific therapy.

Bibliography

1. Wei-Chih Liao. "Outcome and prognostic factors of patients treated in the intensive care unit for carbon monoxide poisoning". *Journal of the Formosan Medical Association* 118.4 (2018): 821-827.

2. Giuseppe Lippi., *et al*. "Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbon monoxide poisoning". *Clinical Biochemistry* 45.16-17 (2012): 1278-1285.

3. SY Donati., *et al*. "Intoxication au CO". *EMC - Anesthésie-Réanimation* 2.1 (2005): 46-67.

4. Ivan Blumenthal. "CO poisoning". *Journal of the Royal Society of Medicine* 94.6 (2001): 270-272.

5. lindell K Weaver., *et al*. "Hyperbaric Oxygen for Acute Carbon Monoxide Poisoning". *The New England Journal of Medicine* 347.14 (2002): 1057-1067.

6. Aslan S., *et al*. "The investigation of ischemic myocardial damage in patients with carbon monoxide poisoning". *Kardiyoloji Dergisi* 5.3 (2005): 189-193.

7. Armin Ernest and Joseph D Zibrak. "CO poisoning". *The New England Journal of Medicine* 340.16 (1999): 1290-1392.

8. Syeven Doherty. "History, pathophysiology, clinical presentation and role of HBO in acute co poisoning". *Emergency Medicine* 12.1 (2000): 55-61.

9. JC Raphael., *et al*. "Intoxication aiguë par le monoxyde de carbone". *Réanimation Urgences* 1.5 (1992): 723-735.

10. James A Raub., *et al*. "Carbon monoxide poisoning - a public health perspective". *Toxicology* 145.1 (2000): 1-14.

11. Yoav Yanir., *et al*. "Cardiogenic shock complicating acute co poisoning despite neurologic and metabolic recovery". *Annals of Emergency Medicine* 40.4 (2002): 420-424.

12. Doran Zahger., *et al*. "Severe Reversible Left Ventricular Dysfunction Induced by carbon monoxide intoxication". *American Journal of Emergency Medicine* 20.6 (2002): 572-573.

13. Hampson NB., *et al*. "Symptoms of acute carbon monoxide poisoning do not correlate with the initial carboxyhemoglobin level". *Undersea and Hyperbaric Medical Society* 39.2 (2012): 657-665.

14. Gandini C., *et al*. "Cardiac damage in pediatric carbon monoxide poisoning". *Journal of Toxicology: Clinical Toxicology* 39.1 (2001): 45-51.

15. Mary Jo C Grant and Bonnie Clay. "Accidental carbon monoxide poisoning with severe cardiorespiratory compromise in 2 children". *American Journal of Critical Care* 11.2 (2002): 128-131.

16. Hubalewska-Hola A., *et al*. "Clinical and scintigraphic (99mTc-MIBI SPECT) heart evaluation in young acutely carbon monoxide poisoned patients". *Przegląd Lekarski* 60.4 (2003): 226-232.

17. CSA Macmillan., *et al*. "Reversible increase in QT dispersion during co poisoning". *Acta Anesthesiologica Scandinavia* 45.3 (2001): 396-397.

18. David L., *et al*. "Transient cardiac dysfunction in acute carbon monoxide poisoning". *The American Journal of Medicine* 117.8 (2004): 623-625.

19. Daniel Sartan., *et al*. "Cardiovascular manifestations of moderate to severe co poisoning". *Journal of the American College of Cardiology* 45.9 (2005): 1513-1516.

20. James R Roberts., *et al*. "Successful Heart Transplantation From a Victim of Carbon Monoxide Poisoning". *Annals of Emergency Medicine* 26.5 (1995): 652-655.

21. Wocka-Marek T., *et al*. "The usefulness of troponin I in the diagnosis of cardiac damage in acute carbon monoxide poisoning". *Medycyna Pracy* 53.2 (2002): 119-123.

22. Bao-Li Zhu., *et al*. "Postmortem cardiac troponin T levels in the blood and pericardial fluid. Part 1. Analysis with special regard to traumatic causes of death". *Legal Medicine* 8.2 (2006): 86-93.

23. Zhonghua Lao Dong., *et al*. "Changes of myocardial enzymes in patients with acute carbon monoxide poisoning". 21.1 (2003): 51-53.

24. Pach J., *et al*. "Usefulness of rest and forced perfusion scintigraphy (SPECT) to evaluate cardiotoxicity in acute carbon monoxide poisoning". *Przegląd Lekarski* 58.4 (2001): 297-300.

25. Richard Pullinger. "Lesson of the week: something in the air: survival after dramatic, unsuspected case of accidental co poisoning". *British Medical Journal* 312.7035 (1996): 897-898.

26. Tritapepe L., *et al*. "Functional and ultrastructural evidence of myocardial stunning after acute carbon monoxide poisoning". *Critical Care Medicine* 26.4 (1998): 797-801.

27. Michael J Bentley., *et al*. "Successful cardiac transplantation with methanol or carbon monoxidepoisoned donors". *The Annals of Thoracic Surgery* 71.4 (2001): 1194-1197.

28. Henry CR., *et al*. "Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning". *Journal of the American Medical Association* 295.4 (2006): 398-402.

29. Robert Malbosc. "Intoxications aiguë et chronique par le monoxyde de carbone: aspect analytique et interprétation des oxycarbonémies". *Revue Française des Laboratoires* 2000.323 (2000): 19-25.

30. Sorodoc L., *et al*. "A rare cause of non-Q myocardial infarction-acute carbon monoxide poisoning". *Revista Medico-Chirurgicala a Societatii de Medici si Naturalisti din Iasi* 108.4 (2004): 782-785.

31. WR Harvey and P Hutton. "Carbon monoxide: chemistry, role, toxicity and treatment". *Current Anaesthesia and Critical Care* 10.3 (1999): 158-163.

Volume 6 Issue 8 August 2019
©All rights reserved by S Bellali.*, et al.*