Cardiology and Angiology: An International Journal

Volume 12, Issue 4, Page 295-301, 2023; Article no.CA.106415
ISSN: 2347-520X, NLM ID: 101658392

Acute Coronary Syndrome Induced by Carbon Monoxide Intoxication: A Case Report

P. M. Mulendele [a*], M. Sidi Boutar [a], B. E. Ovaga [a], M. Njie [a], M. Charfo [a], S. Arous [a,b++], M. G. Benouna [a,b++], A. Drighil [a,b++], L. Azzouzi [a,b++] and R. Habbal [a,b++]

[a] Department of Cardiology P37, Ibn Rochd University Hospital, Casablanca, Morocco.
[b] Faculty of Medicine and Pharmacy, Hassan II University, Casablanca, Morocco.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/CA/2023/v12i4370

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/106415

Case Study

Received: 20/07/2023
Accepted: 25/09/2023
Published: 03/10/2023

ABSTRACT

Carbon monoxide (CO) intoxication is the most frequent etiology of toxicological morbidity and mortality in the world. If the neurological manifestations are in the foreground, then cardiovascular signs of myocardial damage by necrosis are possible, which can result in angina, an acute coronary syndrome followed by myocardial stupor, collapse or even cardiac arrest.

We report a case of CO intoxication inducing an acute coronary syndrome without ST elevation in an elderly diabetic patient with no coronary artery disease history.

Diagnosis: This patient presented a CO intoxication after an accidental exposure; the electrocardiogram showed a depressed ST segment of the inferior and apico-lateral leads; cardiac enzyme troponins gradually increased. Echocardiography showed a preserved systolic function with a disorder of the heart wall motions. Coronary angiography revealed significant lesions of the

---

[++] Professor;
**Corresponding author: E-mail: mulendelepatrick2@gmail.com;*

proximal left anterior descending artery (LAD) and the first marginal evoking a diagnosis of acute coronary syndrome induced by CO intoxication.
Interventions: His management consisted of the administration of hyperbaric oxygen therapy, a platelet aggregation inhibitor (aspirin plus clopidogrel), an anticoagulant (low molecular weight heparin) and myocardial support (Bisoprolol). Coronary angiography and the placement of two active stents were performed after 24hrs onset diagnosis.
Results: The patient had a good clinical outcome under hyperbaric oxygen therapy, complete revascularization of the angiographic lesions which showed improvement in cardiac function and a slight improvement of heart wall motion on echocardiography performed on the 5th day. After 6 days, the patient had recovered well and was discharged from the hospital without chest discomfort, dizziness or headache.
Lecture: This case suggests that the symptoms of carbon monoxide intoxication are complex and diverse. This can manifest as a primary hypoxic symptom or cause an exacerbation of underlying diseases due to hypoxia. Therefore, patients with carbon monoxide intoxication should actively seek a comprehensive cardiac examination to ensure early diagnosis. Whenever necessary, coronary angiography and stent implantation should be performed to improve the patient's chances of survival.

Keywords: Carbon monoxide intoxication; acute coronary syndrome without ST elevation; coronary angiography.

1. INTRODUCTION

CO intoxication is the most common etiology of toxicological morbidity and mortality worldwide [1,2]. It can occur intentionally or accidentally, through exposure to any source of combustion, including automobile exhaust, faulty heating systems, hot water production, cooking or smoking [3]. If the neurological manifestations are in the foreground, and the neurological sequelae are the long-term seriousness of the intoxication, the cardiovascular signs of myocardial damage by necrosis are possible, which can result in angina, acute coronary syndrome followed by myocardial stunning, collapse or even cardiac arrest make it life-threatening, and have already been reported [4,5,6,7].

We report a patient with non-ST-segment elevation myocardial infarction induced by accidental carbon monoxide intoxication.

2. CASE REPORT

A 77-year-old woman, who called the firefighters for chest discomfort which results to a trauma after falling on her pelvis bone few minutes later after respiratory discomfort and chest discomfort. Upon arrival at the incidence site, the firefighters noted: a CO level of 550 ppm and a faulty boiler. She reported symptoms of nausea, vomiting and dizziness since the day before.

The somatic examination finds a patient hemodynamically stable, polypneic with desaturation, regular heart sounds, no murmur, discreet bilateral crackles at the bases; no signs of right heart failure.

The ECG reveals a regular sinus rhythm with a discrete repolarization disorder type ST segment depression at the inferior and apico-lateral leads (Fig. 1).

Biological assessment showed; HbCO at 9% in a non-smoker patient, highly sensitive cardiac enzyme troponin increased gradually (raised to 279ng/l for the first dosage then to 1056ng/l after the 2nd dosage); creatinine at 127umol/l with respiratory acidosis on venous gazometry (Ph=.29, paCO2 46.2, whilst the other biological parameters were normal.

Transthoracic echocardiography (TTE) findings concluded; heart chambers of normal size, disorder heart wall motion; akinesia of the inferosepto wall of the heart at its basal and median segments and hypokinesia of the septo-apical wall. The systolic function of the left ventricle was preserved, LVEF of 50% by Simpson biplane method. Normal filling pressure and absence of valvular disease.

The diagnose of non-ST segment elevation myocardial infarction (NSTEMI) induced by carbon monoxide intoxication was retained. The patient responded well after high concentration oxygen therapy with a controlled HbCO of 3.1%. He also received treatment of a dual anti-platelet therapy with aspirin and clopidogrel, an

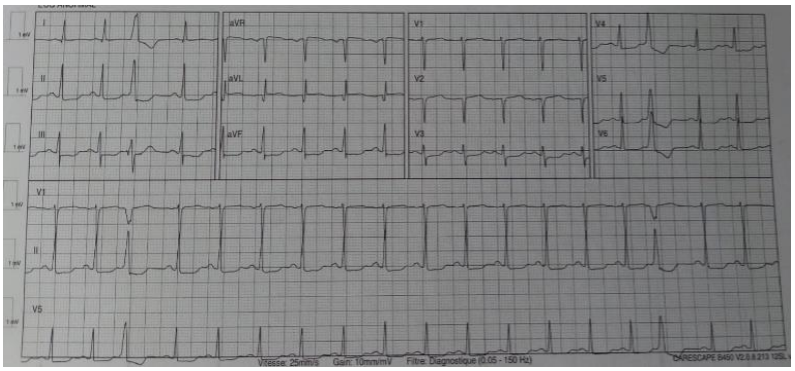

Fig. 1. Electrocardiogram (ECG): showing a non-ST segment elevation myocardial infarction at the apico-lateral and inferior leads corresponding to territories of coronary disease arteries

anticoagulant (low molecular weight heparin) and a myocardial support (Bisoprolol).

Infront of the clinical manifestation of chest discomfort associated with electrical changes, elevation of troponin and impaired heart wall motion, coronary angiography was performed; noting a left dominance coronary artery. The common trunk was of normal length, without stenosis. The anterior descending artery of normal size. This artery gives off a diagonal branch. The proximal left anterior descending artery (LAD) presents a total occlusive stenosis (90-99%) more than 20mm long, slightly calcified, irregular, encompassing the origin of the first diagonal. This lesion extends to the middle portion of the left anterior descending artery (LAD). The reference diameter was 3 mm at this level. There was a slowing down of the contrast product in this artery (TIMI 2). The circumflex artery was of a normal diameter. This artery gives off a single significant marginal branch. The ostium of the first marginal has a very tight stenosis (70-90%) 10 to 20 mm long, slightly calcified and type B2 (Fig 2.). Coronary flow was normal (TIMI 3). The right coronary artery was small and dominated and free from stenosis on all of its segments. The conclusion of the angiography was: bitroncular lesions; significant long stenosis of the proximal LAD, encompassing the origin of the first diagonal (bifurcation lesion) and significant stenosis of the ostium of the first marginal.

During angiography, the first marginal ostium was treated by implanting an active stent (Fig. 3). The proximal LAD was treated by implantation of an active stent. POT LAD 1 distal by balloon NC3.0X8mm (Fig. 4).

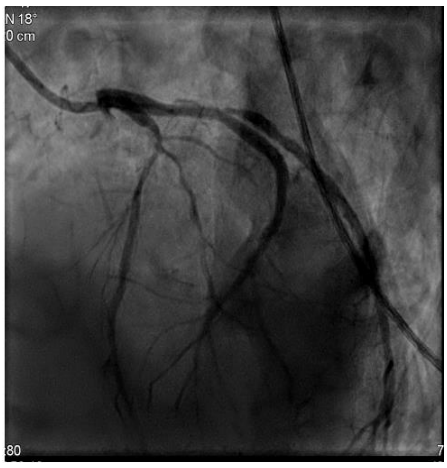

Fig. 2. Coronary angiography: Reveals a tight stenosis of the ostium of the first marginal (70-90%) measuring 10mm to 20 mm long, slightly calcified and type B2

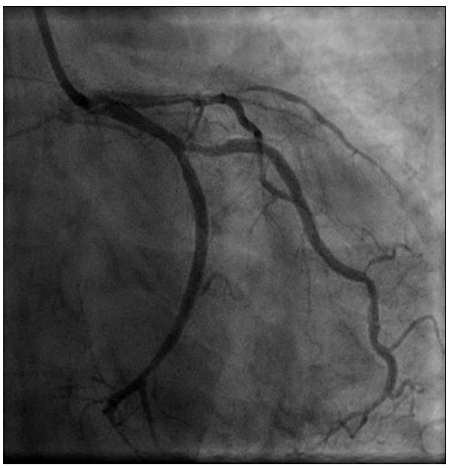

Fig. 3. Coronary angiography: shows an improved blood flow after an active stent implantation at the proximal left anterior descending artery

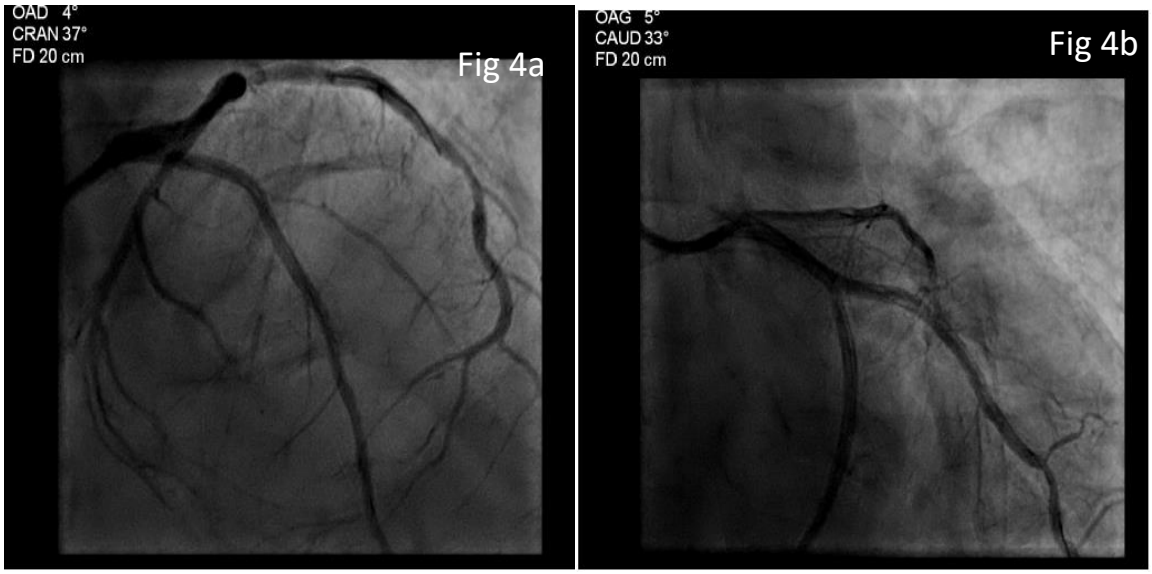

Fig. 4 (a&b). Coronary angiography: a-shows angioplasty of the first marginal by balloon b-recovered blood flow after balloon implantation

The patient responded well clinically under hyperbaric oxygen therapy and underwent complete revascularization by implantation of two active stents and was discharged from the hospital after 6 days of hospitalization without recurrent chest discomfort, dizziness or headache.

3. DISCUSSION

Carbon monoxide intoxication is the leading cause of toxicological deaths worldwide [1,2]. This colorless, odorless, tasteless, non-irritating gas, not perceptible to humans, is produced during any incomplete combustion of carbonaceous compounds (fuel, natural gas, wood, coal, butane, gasoline, petroleum, propane) due to insufficiency of oxygen. With a density close to that of air, carbon monoxide diffuses rapidly in the atmosphere and is easily absorbed in the lungs, it reaches the bloodstream and essentially binds to hemoglobin, to form a stable molecule, carboxyhemoglobin, due to its affinity 200 to 250 times greater than that of oxygen and hemoglobin [8]. CO intoxication can cause tissue damage of varying severity, neurological, respiratory and also cardiac [3,9]. The severity of intoxication depends on the amount of carbon monoxide (CO) fixed by hemoglobin. Chronic CO intoxication, induced by inhaling low concentrations over repeated periods of time, causes mild symptoms that can be relieved quickly. Acute or subacute intoxication secondary to exposure to high concentrations of CO over a short period of time can damage several organs such as the brain, heart, lungs, kidneys thus fatal [3,9].

For their proper functioning, the nervous system (brain) and the cardiovascular system (heart) require high levels of oxygen thus they are very sensitive to hypoxia.

In moderate to severe CO intoxication, neurological damage is prominent and well known and myocardial damage is also common [9,10]; Myocardial infarction, with or without ST segment elevation, coronary spasm and arrhythmias have been reported and constitute an independent predictive factor of vital prognosis in seriously affected victims of CO intoxication [4-6]. These myocardial lesions generally occur between 1 to 7 days after intoxication [10] and can even appear during mild intoxication.

The toxic mechanisms of carbon monoxide are complex. Hypoxia is the main mechanism explaining lesions, in particularly the myocardium, secondary to carbon monoxide intoxication. The affinity of CO binding to hemoglobin is more than that of the affinity of hemoglobin for oxygen, thereby shifting the hemoglobin dissociation curve to the left [11]. Carbon monoxide reduces the ability of hemoglobin to carry oxygen and also decreases its ability to deliver it to tissues. In addition,

carbon monoxide binds to myoglobin to form non-functional carboxymyoglobin with decreased cardiac output further aggravating tissue hypoxia explaining the causes of myocardial lesions. The binding of CO with cytochrome-c oxidase (a terminal enzyme of the electron transport chain), directly interferes with cellular respiration by setting up anaerobic metabolism by inhibiting the synthesis of ATP leading to tissue hypoxia, lactic acidosis and apoptosis [12].

CO can cause damage to the endothelium by endothelial deposition of peroxynitrite and induce coronary artery spasm, accelerate platelet aggregation and slow blood flow-induced thrombosis [13].

Acute and subacute intoxication are manifested by trivial and variable functional symptoms and may be asymptomatic.

CO intoxication; especially in severe cases can cause asthenia, dizziness as well as changes in mood (anxiety, agitation, irritability), behavioral (confusional syndrome) or visual disturbances, dyspnea on exertion and loss of consciousness at the slightest effort [3]. ACS can occur during CO intoxication with extreme myocardial infarction with or without ST segment elevation, in patients with or without cardiovascular risk factors [5,6,7]. Carbon monoxide intoxication can destabilize a stable plaque in unknown coronary subject and can be a triggering factor. Therefore, in elderly patients with carbon monoxide intoxication, the possibility of myocardial infarction is relatively high especially in the presence of cardiovascular risk factors as seen in our patient. In this context, any patient presenting with signs of carbon monoxide intoxication must have a cardiac evaluation, including an electrocardiogram, a cardiac ultrasound, the dosage of highly sensitive cardiac troponin and, depending on the results; a coronary angiogram should be performed to facilitate an accurate diagnosis [10]. Our patient coronary angiogram confirmed a significant stenosis of the proximal LAD and the ostium of the first marginal.

It should be noted that the degree of carbon monoxide intoxication may not be directly proportional to the degree of myocardial damage. In most cases, a more severe degree of carbon monoxide intoxication results in severe myocardial damage and elevated cardiac enzyme troponin values. However, in rare cases, mild carbon monoxide intoxication can also lead to severe myocardial damage. The case reported here is an example.

Treatment includes hyperbaric oxygen therapy after rescuing from intoxication site. Recognized for many years [14], oxygen therapy could be normobaric or hyperbaric. Compared to standard oxygen therapy, hyperbaric oxygen therapy allows the rapid dissociation of carboxyhemoglobin and promotes the elimination of carbon monoxide. In addition, hyperbaric oxygen reduces the secretion of inflammatory factors, increases mitochondrial function, inhibits lipid peroxidation and reduces leukocyte adhesion to damaged microvascular walls [15]. Henry et al. [16] recommended that patients should inhale 100% oxygen until the symptoms resolve and drop in the level carboxyhemoglobin to 5% to 10%. If the cardiovascular system is affected by intoxication, it is recommended that the carboxyhemoglobin should fall below 2%.; our patient has responded well under high concentration oxygen therapy with a controlled HbCO of 3.1%.

The role of hyperbaric oxygen in the management of carbon monoxide intoxications remains controversial, although physiological data and some randomized trial data suggest potential benefit [17,18]. Experiments in non-human animals demonstrated that administration of hyperbaric oxygen reduced infarct size in the ischemic rabbit heart during reperfusion [19]; however, other studies have reported that hyperbaric oxygen had no beneficial effect on infarct size in dogs [20,21]. Currently, there are no other reports of hyperbaric oxygen therapy for carbon monoxide intoxication associated with myocardial infarction. Further clinical studies are needed.

4. CONCLUSION

Carbon monoxide intoxication can destabilize a previously stable plaque in unknown coronary subjects and can be a triggering factor. It is extremely important to examine the influence of CO intoxication on ischemia and myocardial damage. Diagnosis and treatment should be made at an early stage of myocardial damage to reduce the long-term mortality rate.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Centers for Disease Control and Prevention (CDC). Carbon monoxide--related deaths-United States, 1999-2004. MMWR Morb Mortal Wkly Rep. 2007;56 :1309-12.
2. Mott JA, Wolfe MI, Alverson CJ, Macdonald SC, Bailey CR, Ball LB, Moorman JE, Somers JH, Mannino DM, Redd SC. National vehicle emissions policies and practices and declining US carbon monoxide-related mortality. JAMA. 2002 ;288 :988-95.
3. ERNST, Armin et ZIBRAK, Joseph D. Carbon monoxide poisoning. New England journal of medicine. 1998;339:1603-1608.
4. MARIUS-NUNEZ, Andres L. Infarctus du myocarde avec artères coronaires normales après exposition aiguë au monoxyde de carbone. Poitrine. 1990;97: 491-494.
5. Dziewierz A, Ciszowski K, Gawlikowski T, Rakowski T, Kleczyński P, Surdacki A, Dudek D. Primary angioplasty in patient with ST-segment elevation myocardial infarction in the setting of intentional carbon monoxide poisoning. J Emerg Med. 2013 ;45 :831-4.
6. Gao Y, Yang J, Ma L, Zhang Y, Li Z, Wu L, Yang L, Wang H. Non-ST elevation myocardial infarction induced by carbon monoxide poisoning: A case report. Medicine (Baltimore). 2019;98: e15151.
7. Capilla E, Pons F, Poyet R, Kerebel S, Jego C, Louge P, Cellarier GR. Syndrome coronaire aigu avec dysfonction ventriculaire gauche lors d'une intoxication au monoxyde de carbone [Acute coronary syndrome with impaired left ventricular function in a carbon monoxide poisoning]. Ann Cardiol Angeiol (Paris). 2016;65:45-7.
8. Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. J Neurol Sci. 2007; 262:122-30.
9. Lippi G, Rastelli G, Meschi T, Borghi L, Cervellin G. Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbon monoxide poisoning. Clin Biochem. 2012;45:1278-85.
10. Satran D, Henry CR, Adkinson C, Nicholson CI, Bracha Y, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. J Am Coll Cardiol. 2005;45:1513-6.
11. Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. J Neurol Sci. 2007;262:122-30.
12. ALONSO, Jose-Ramon, CARDELLACH, Francesc, LÓPEZ, Sònia, et al. Carbon monoxide specifically inhibits cytochrome c oxidase of human mitochondrial respiratory chain. Pharmacology & toxicology. 2003;93:142-146.
13. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. Chest. 1990;97:491-4.
14. Haldane J. The Relation of the Action of Carbonic Oxide to Oxygen Tension. J Physiol. 1995;18:201-17.
15. Weaver LK. Hyperbaric oxygen therapy for carbon monoxide poisoning. Undersea Hyperb Med. 2014;41:339-54.
16. Henry CR, Satran D, Lindgren B, Adkinson C, Nicholson CI, Henry TD. Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning. JAMA. 2006;295:398-402.
17. Weaver LK, Hopkins RO, Chan KJ, et al. Oxygène hyperbare pour l'intoxication aiguë au monoxyde de carbone. N Engl J Med. 2002;347:1057–1067
18. Thom SR, Taber RL, Mendiguren II, Clark JM, Hardy KR, Fisher AB. Séquelles neuropsychologiques tardives consécutives à une intoxication au monoxyde de carbone : Prévention par traitement à l'oxygène hyperbare. Ann Emerg Med. 1995;25: 474–480
19. Sterling DL, Thornton JD, Swafford A, Gottlieb SF, Bishop SP, Stanley AW, Downey JM. Hyperbaric oxygen limits infarct size in ischemic rabbit myocardium in vivo. Circulation. 1993;88:1931-6.
20. Mogelson S, Davidson J, Sobel BE, Roberts R. The effect of hyperbaric oxygen

on infarct size in the conscious animal. Eur J Cardiol. 1981;12:135-46.

21. Henry CR, Satran D, Lindgren B, Adkinson C, Nicholson CI, Henry TD. Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning. JAMA. 2006;295:398-402.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/106415