Available online at www.elixirpublishers.com (Elixir International Journal)

Cardiovascular Toxicology

Elixir Cardiovascular Toxicology 109 (2017) 47962-47964

ISSN: 2229-712X

Unknown etiology of a common disease: NSTEMI related to CO poisoning: case report and review of literature.

El ouazzani Jamal, Beghi Mustapha, Bazid Zakaria, Ismaili Nabila and El ouafi Noha

Department of Cardiology, Mohammed VI University Hospital, 60049 Oujda, Morocco.,

ARTICLE INFO

Article history:

Received: 7 June 2017;

Received in revised form:

2 August 2017;

Accepted: 12 August 2017;

Keywords

Carbon monoxide poisoning,
Acute coronar syndrome,
Oxygen therapy,
Carboxyhemoglobin.

ABSTRACT

Carbon monoxide (CO) poisoning is the leading cause of death by poisoning in western countries. Certainly the neurological symptoms are the most common, yet the cardiovascular signs are considerable too and may be responsible of a significant morbidity and mortality. Therefore health professionals must be aware of its diagnosis, treatments and prognostic implications. We report here a case of a patient with a non ST segment elevation acute coronary syndrome with positive troponin related to carbon monoxide poisoning.

Introduction

Carbon monoxide poisoning is the leading cause of death by poisoning in western countries [1]. This painless and colorless gas generated by the incomplete combustion of carboxylic compounds, has a 240 times higher affinity for hemoglobinthan of the oxygen [2-3]. Certainly the neurological symptoms are most common, yet the cardiovascular signs are considerable too and may be responsible of a significant morbidity and mortality. We report here a case of a patient with a non ST segment elevation acute coronary syndrome with positive troponin related to carbon monoxide poisoning.

Methods

The information used to prepare the clinical case were obtained by a meticulous examination of the patient's medical file, while the literature's documents were picked after a careful review of medical database: "sciencedirect", "pubmed" and "Google scholar".

Case report

We report the case of a 35 years old female patient without any cardiovascular risk factors neither a medical history. The woman was found unconscious for an undetermined duration, by a member of her family in a bathroom that contains a Geyser, which prompted the family to consult at emergency room of our hospital. At her admission, the patient was somnolent but responded to verbal and painful stimulus, and had a retro sternal chest pain, of which the characteristics were difficult to specify due to the patient's neurological status. Her heart rate was at 110 beats/ min, the blood pressure at 165/95 mmHg, respiration rate at 30 breaths/min, and the oxygen's saturation in ambient air was at 85%. An electrocardiogram was performed revealing a sinusal tachycardia at a frequency of 110 beats/ min, ST-segment depression in lead V4, V5, V6, DI and DII associated with ventricular premature beats, QT corrected interval was normal at 446 ms (fig1).

At her admission, the patient benefited from conditioning with oxygen at 100% by a high concentration mask, after its maintain for 120 minutes, the neurological examination became normal while the chest pain and the ECG's disorders persisted.

The biological balance sheet found a carboxyhemoglobin (HBCO) level at 17%, an Ultra-sensitive troponin I was in kinetic of increase (1st to 6 times the normal level and the 2nd to 77 times). The blood count was normal as well as the rate of creatinine and plasma urea.

She was admitted then, at the cardiac intensive care unit for the management of the acute coronary syndrome without ST-segment elevation and a positive troponin associated with CO poisoning.

At the cardiac intensive care unit, she received a treatment combining the oxygen for carbon monoxide poisoning and the acute coronary syndrome's treatment including: double platelet anti-aggregation (CLOPIDOGREL AND ASPIRIN), Heparin therapy at curative dose, an Angiotensin-converting-enzyme inhibitor (ACE inhibitor), a statin and a beta blocker.

24 hours after admission, the patient did not suffer and the electrocardiogram (fig2) and echocardiography performed did not find any abnormalities with a homogeneous contractility, and a left ventricular ejection fraction estimated at 60%. The coronary angiography showed anangiographically normal coronary arteries (fig3). The patient was discharged after 5 days of hospitalization without any neurological or cardiac sequelae. Followed-up in consultation for six months did not find any recurrences of neurological or cardiac symptomatology.

Tele:
E-mail address: j.elouazzani.cardio@gmail.com

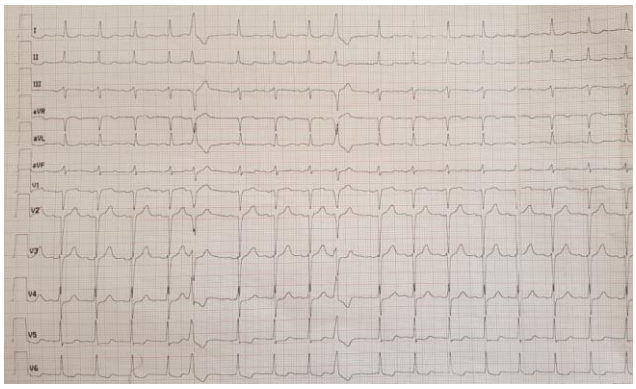

Figure 1. A Electrocardiogram performed on admission to the emergency room revealing ST-segment depression in lead V4, V5, V6, DI and DII associated with ventricular premature beats and a normal QT corrected interval at 446 ms.

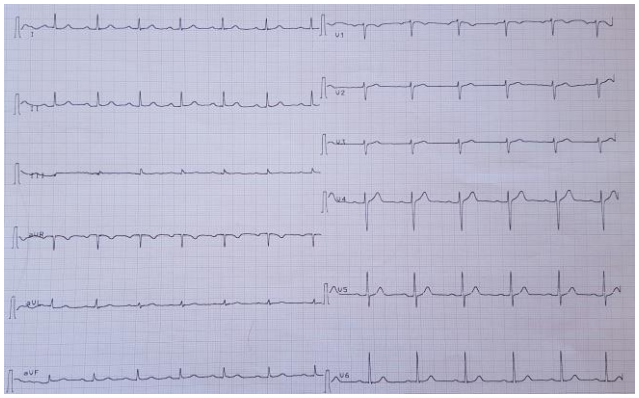

Figure 2. Electrocardiogram 24 hours after admission to the emergency room.

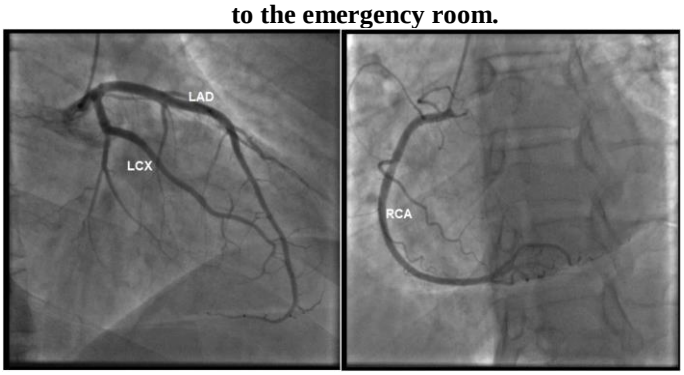

Figure 3. Coronary angiography showing normal epicardial coronary arteries.

LAD: left anterior descending coronary artery
LCX: left circumflex artery
RCA: right coronary artery

Discussion

Carbon monoxide's toxicity is known throughout history as the major cause of death by involuntary poisoning, and in the Greek and Roman history as a method of criminal execution [2]. In the united states, the Centers for Disease Control and Prevention (CDC) had revealed that the incidence of CO intoxication is about 23.2 per 1 million people per year. It's also responsible for nearly 15 000 consultations at the emergency department and for 500 death per year [4]. A prospective study has shown that myocardial injury is not rare in moderate to severe CO poisoning, and it occurs in almost 37% [5].

The main physiopathological mechanism of this intoxication is summarized in the carbon monoxide's capacity to link to hemoglobin with a higher affinity than that's of oxygen, what induces the production of Carboxyhemoglobin instead of Oxyhemoglobin which is essential for tissue's oxygenation [3].the Myocardial injury is common in moderate to severe poisoning, this is explained by the increased oxygen requirements and the low metabolic reserves of myocardial tissue [3,6].Patients with the previous heart disease particularly coronary heart disease have a higher risk of infarct and arrhythmia compared to patient free of heart disease [8].the heart injury spectrum is wide. Including mainly acute coronary syndromes with or without ST-segment elevation, conduction disorders, ventricular and supraventricular arrhythmias, as well as acute heart failure and cardiogenic shock [8,9,10].

The most frequent extra-cardiac symptoms of the intoxication are often neurological, ranging from headache to syncope and coma, without an obvious correlation between the HBCO level and the neurological outcome of the intoxication [11.12]. Thoracic pain is the most frequent clinical symptom of cardiac origin, with an obvious relation between the response and CO poisoning degree [13].

The diagnosis of CO poisoning is based on Carboxyhemoglobin blood dosage. The high positive levels have a high positive predictive value while the low levels do not definitively exclude the exposure [14], but we retain a HBCO value higher than 10% in non-smokers and than 15% in smokers to confirm CO poisoning diagnosis [1].

The diagnosis of the cardiac involvement during poisoning is not always apparent, since patients come usually with consciousness disorders and we don't always think to eliminate heart involvement. Electrocardiographically, many abnormalities have been reported, repolarization disorders are the most widespread and may persist for 3 to 7 days, or for a lifetime [1], ventricular and supra-ventricular arrhythmias are also common [14]. The evaluation and vigilant supervision of QT interval should be systematical, because of the risk of its prolongation that can cause fatal arrhythmias and contraindicate the use of any drugs prolonging this interval [15].

As for the biological level, assessment of cardiospecific troponins is being now the main biomarker allowing diagnosis and evaluating cardiac involvement severity, whether it's of toxic or ischemic origin, so that it would be particularly indicated in any CO poisoning, and mostly after the advent of highly sensitive new immunoassay methods that enable us to identify reliably and precociously the presence of a myocardial injury [16, 17].

V.DAVUTOGLU et al had shown that plasmatic dosage of NT-proBNP can contribute to the precocious diagnosis and the assessment of the severity of cardiotoxicity in this context [18]. Concerning the other cardiac damage biomarkers as CK, CK-MB, aspartate aminotransferase and alanine aminotransferase, seem to be inappropriate for assessing heart involvement because of their low sensibility and poor correlation with data from Single photon emission computed tomography (SPECT) and ECG [19].

The oxygen therapy is the main pillar of CO poisoning treatment. In theory, it reduces pulmonary elimination's half-life from 3-4 hours to 30-90 minutes in the presence of 100% normobar oxygen and to 15-23 minutes with hyperbar oxygen at 2.5 atm and 100% oxygen by accelerating the dissociation of Carboxyhemoglobin [14]. Practically it's not that simple, and the studies results are controversial especially concerning the interest of hyperbaric oxygen therapy; some randomized essays suggest a potential benefit of this method particularly in the prevention of neurological sequelae[20], while others have not shown significant difference mainly in the short term mortality

between normobaric and hyperbaric-oxygen therapy [21,22].That's why hyperbaric-oxygen therapy's indications are still a debatable topic, and are presently restrained to loss of consciousness, neurological deficit, myocardial ischemia, metabolic acidosis and in the case of a Carboxyhemoglobin level upper than 25%[23].

The oxygen therapy should be maintained until the patient becomes asymptomatic and the Carboxyhemoglobin level is less than 10% or even 5%, and about 2% in case of pulmonary or cardiac involvement [24].

In our case, the suggested extended exposure to CO, the loss of consciousness, rising biomarkers of myocardial necrosis, ECG's modifications pushed us to class the intoxication as severe and to take into consideration the hyperbaric oxygen therapy but due to its unavailability, we used the normobaric-oxygen therapy by high concentration mask with close supervision. This oxygen therapy was maintained for 36 hours until cardiac and neurological symptoms extinction and Carboxyhemoglobin's level normalization.

Conclusion

The heart involvement following carbon monoxide poisoning is usually under diagnosed even neglected, that's why health professionals must be sensitized about its diagnostic, therapeutics and prognostic implications.

In spite of its severity and high frequency, few studies were interested in carbon monoxide poisoning and particularly in the heart involvement, which explains the lack of proofs and guidelines concerning diagnostic and treatment modalities, and the need for further randomized studies in order to establish clear and precise recommendations for a better management.

Conflict of interest

The authors declare that there are no conflict of interest

Source of funding

None

Acknowledgment

Authors acknowledge the scholars whose articles are cited and included in references of this case report. The authors are also grateful to authors/editors/publishers of all those articles and journals from where the literature for this article has been reviewed and discussed.

Reference

[1] S-Y. Donati, M. Gainnier, O. Chibane-Donati. Intoxication au monoxyde de carbone; EMC Anesthésie-réanimation 2005: 1-15.

[2] LD. Prockop, RI. Chichkova. carbon monoxide intoxication: an updated review. jneurolsci 2007;262(1–2): 122–30.

[3] G. Lippi, G. Rastelli, T. Meschi, L. Borghi, G. Cervellin . Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbon monoxide poisoning. Clinical Biochemistry 45 (2012) 1278–1285.

[4] Centers for Disease Control, Prevention (CDC). Carbon monoxide exposures–United States, 2000–2009. MMWR Morb Mortal Wkly Rep 2011;60:1014–7.

[5] D. Satran, CR Henry, C Adkinson, et al. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. JACC. 2005;5:1513-6.

[6] D. Rastelli, S. Callegari, C. Locatelli, G. Vezzani . Myocardial injury in carbon monoxide poisoning. G ItalCardiol (Rome) 2009;10:227–33.

[7] G. Lippi, M. Plebani, G. Cervellin. Cocaine in acute myocardial infarction. AdvClinChem 2010;51:53–70.

[8] A. Ernst, JD. Zibrak. Carbon monoxide poisoning. N Engl J Med. 1998;339:1603-1608.

[9] MW. Diltoer, IO. Colle, I. Hubloue, et al. Reversible cardiac failure in an adolescent after prolonged exposure to carbon monoxide. Eur J Emerg Med. 1995;2:231-235.

[10] Y. Yanir, A. Shupak, A. Abramovich, SA. Reisner, A. Lorber. Cardiogenic shock complicating acute carbon monoxide poisoning despite neurologic and metabolic recovery. Ann Emerg Med 2002;40:420–4.

[11] I. Blumenthal. Carbon monoxide poisoning. J R Soc Med 2001;94:270–2.

[12] S. Henz, M. Maeder. Prospective study of accidental carbon monoxide poisoning in 38 Swiss soldiers. Swiss Med Wkly 2005;135:398–408.

[13] RS. Koskela, P. Mutanen, JA. Sorsa, M. Klockars. Factors predictive of ischemic heart disease mortality in foundry workers exposed to carbon monoxide. Am J Epidemiol 2000;152:628–32.

[14] G. Lippi, G. Rastelli, T. Meschi, L. Borghi, G. Cervellin. Pathophysiology, clinics, diagnosis and treatment of heart involvement in carbon monoxide poisoning. Clinical Biochemistry 45 (2012) 1278–1285.

[15] B. Yelken, B. Tanriverdi, F. Cetinbas, D. Memiş, N. Süt. The assessment of QT intervals in acute carbon monoxide poisoning. Anadolu Kardiyol Derg 2009;9:397–400.

[16] E. Unal, A. Yazar, B. Oran. The importance of troponin-I as a predictor of cardiac injury caused by carbon monoxide poisoning. InhalToxicol 2007;19:587.

[17] D. Lichtarska, R. Feldman. Troponin positive acute coronary syndromes in the course of acute carbon monoxide poisoning as the factor exposing primary coronary heart disease previously undiagnosed. PrzeglLek 2011;68:510–4.

[18] V. Davutoglu, N. Gunay, H. Kocoglu, NE. Gunay, C. Yildirim, M. Cavdar, et al. Serum levels of NT-ProBNP as an early cardiac marker of carbon monoxide poisoning. Inhal Toxicol 2006;18:155–8.

[19] G. Lippi, M. Montagnana, GL. Salvagno, GC. Guidi. Potential value for new diagnostic markers in the early recognition of acute coronary syndromes. CJEM 2006;8:27–31.

[20] LK. Weaver, RO. Hopkins, KJ. Chan, S. Churchill, CG. Elliott, TP. Clemmer, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002;347:1057–67.

[21] NB. Hampson, NM. Hauff. Risk factors for short-term mortality from carbon monoxide poisoning treated with hyperbaric oxygen. Crit Care Med. 2008;36:2523-7.

[22] DN. Juurlink, NA. Buckley, MB. Stanbrook, GK. Isbister, M. Bennett, MA. McGuigan. Hyperbaric oxygen for carbon monoxide poisoning. Cochrane Database Syst Rev 2005;1:CD002041.

[23] NB. Hampson, CA. Piantadosi, SR. Thom, et al. Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning. Am J Respir Crit Care Med. 2012;186:1095-101.

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