

Misdiagnosis of Carbon Monoxide Intoxication in a Patient with Known Coronary Artery Disease – A Case Report and Review of Cardiovascular Effects of Carbon Monoxide Poisoning

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ABSTRACT

This is a report of a patient who had documented coronary artery disease and was admitted with chest pain, nausea, vomiting and headache. She was immediately taken to coronary angiography and underwent percutaneous coronary intervention with stent implantation. After the operation, she was coincidentally diagnosed to have Carbon Monoxide (CO) poisoning. We discuss if percutaneous intervention (PCI) was an overtreatment and briefly review the mechanisms of the cardiovascular effects of CO toxicity which is an insidious pathology and diagnosed only if it is suspected.

Mal Diagnóstico de Intoxicación por Monóxido de Carbono en un Paciente Diagnosticado con Enfermedad de la Arteria Coronaria – Reporte de un Caso y Estudio de los Efectos Cardiovasculares del Envenenamiento por Monóxido de Carbono

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RESUMEN

Este es un reporte de una paciente con enfermedad de la arteria coronaria documentada, quien fuera ingresado con dolor de pecho, náuseas, vómitos y dolor de cabeza. A la paciente se le hizo inmediatamente una angiografía coronaria y se le realizó una intervención coronaria percutánea con implantación de stent. Después de la operación, se le diagnosticó por coincidencia envenenamiento por monóxido de carbono (CO). Analizamos si la intervención percutánea (IPC) fue un sobretratamiento y examinamos brevemente los mecanismos de los efectos cardiovasculares de la toxicidad por CO que puede ser insidiosa y diagnosticada sólo si se tiene sospecha.

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INTRODUCTION

Carbon monoxide is a by-product of the incomplete burning of hydrocarbon oils, coal, wood, other fuels or motor vehicle exhaust gases. Carbon monoxide causes cardiac toxicity in patients with or without coronary artery disease by several mechanisms like carboxyhaemoglobin formation leading to tissue hypoxia, cytochrome toxicity, binding to myoglobin,

peroxynitrite formation and probably endothelial dysfunction and vasospasm.

Case Report

A 54-year old female patient was admitted to hospital with squeezing chest pain, nausea, vomiting and headache. Her electrocardiography revealed ST segment depression in the first three precordial leads and biphasic T waves in the inferior leads. She had the history of left anterior descending artery-left internal mammary artery (LAD-LIMA), (right coronary artery) – saphenous vein bypass graft operation in 1995 and she had been hospitalized in 2002 for unstable

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angina pectoris and underwent coronary angiography which revealed that the bypass grafts were non-functional. She had undergone percutaneous coronary intervention and two stents were placed in the right coronary artery and the circumflex artery. She was admitted to the hospital two days previously with chest pain again and coronary angiography showed that the stents were patent and there was a non-critical lesion in the ostial region of the right coronary artery (Fig. 1). It was

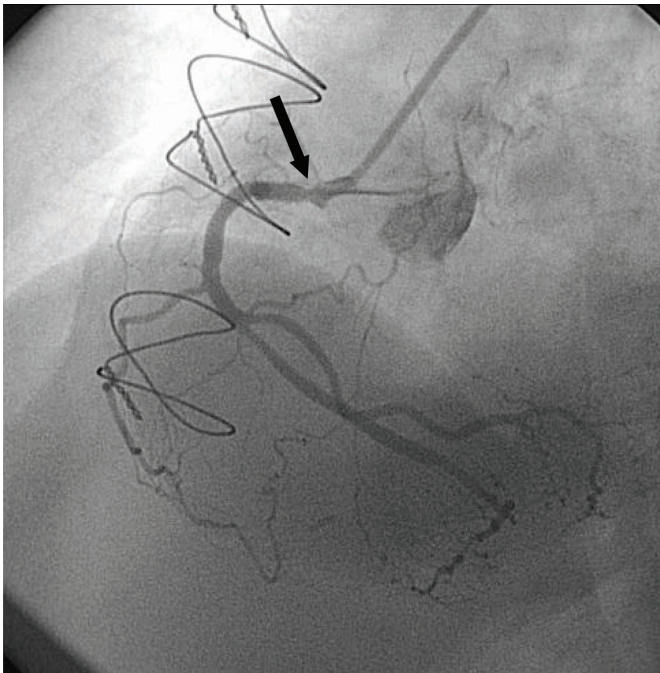


Fig. 1: Coronary angiography two days before the emergency department admission, showing non-critical stenosis at the right coronary artery.

decided to treat her conservatively and any percutaneous intervention was withheld. So when she presented to the Emergency Department again with angina pectoris and the predefined electrocardiographic changes, she was taken to the catheter laboratory to see if the stenosis judged to be non-critical two days previously had become critical indeed. She had coronary angiography within 10 minutes of hospital arrival and a stent was placed in the ostial right coronary artery (Fig. 2). After the operation, we were informed by the laboratory personnel that the arterial blood gas analysis which was performed because it was part of another ongoing trial, showed 30% of COHb level. At that moment, the diagnosis was changed, making the decision of percutaneous intervention very doubtful. Then we considered hyperbaric oxygen therapy but since she had undergone percutaneous intervention and had a femoral sheath, and she was on heparin therapy, we decided to keep her instead of transferring to a centre where she could receive hyperbaric oxygen, considering the risk of bleeding. On normobaric oxygen 100% for several hours, she recovered quickly and was discharged in 4 days.

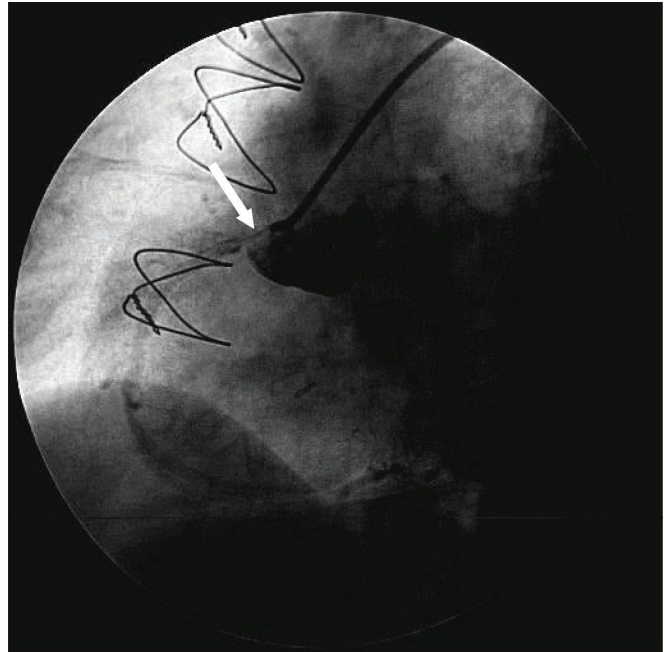


Fig. 2: Coronary angiography at the index admission, showing the ostial lesion at the right coronary artery.

DISCUSSION

Carbon monoxide binds to haemoglobin (Hb) rapidly and the oxygen-carrying capacity of blood decreases resulting in tissue hypoxia (1). Another mechanism which has a minor role in CO poisoning is toxicity to cytochromes but the amount of CO required to poison cytochromes is much more than the lethal dose. Also, CO binds to intracellular myoglobin in the myocardium and impairs oxygen supply to the mitochondria. This negatively affects oxidative phosphorylation and the energy source of heart muscle (2, 3).

In CO toxicity, ultramicroscopic lesions have been reported but relative roles of general tissue hypoxia and specific CO toxicity due to CO binding to cytochromes is unknown.

Chest pain, rhythm disturbances and increased level of cardiac enzymes are very frequent (4). Myocardial injury with ischaemic electrocardiographic (ECG) changes and elevated cardiac biomarkers were found in 37% of 230 patients with a moderate to severe CO poisoning with 5% in hospital mortality (5). Chest pain as a symptom of myocardial ischaemia occur with or without underlying coronary artery disease (6). Features of ischaemia as well as tachycardia, bradycardia, ventricular ectopic beats and conduction abnormalities can be easily detected on ECG.

Clinical findings of CO poisoning are very nonspecific: headache, weakness, confusion and nausea were encountered frequently. Headache is the most common symptom occurring in approximately 80% of patients. Initially, tachycardia and tachypnoea develop to compensate for cellular hypoxia.

Patients with underlying heart and lung diseases decompensate quickly even with mild hypoxia (7, 8).

As previously mentioned, CO poisoning leads to myocardial infarction in patients with and without coronary artery disease. Factors such as inadequate myocardial perfusion and increased thrombotic tendency might also contribute to decreased oxygenation.

Acute thrombosis which is triggered by CO might be the cause of myocardial infarction in patients with underlying coronary artery disease (9, 10).

Coronary spasm is another mechanism leading to acute coronary syndrome in CO intoxication (11, 12). In an animal study, it was shown that calcium sensitivity was increased by CO exposure, vasoreactivity was also changed with a decrease in the response to acetylcholine (ACh) or nitric oxide (NO) donor (13). Carbon monoxide exposure was also shown to cause lipid peroxidation (4). Nitrosative stress due to peroxynitrite formation but without NO formation may occur as in cerebral CO toxicity and this is thought to explain the increase in cardiac calcium sensitivity. Also, endothelial dysfunction, due to CO intoxication triggering coronary vasoconstriction, is a proposed mechanism (13).

In the treatment, oxygen which reduces the half-life of COHb is used. Hyperbaric oxygen is more effective; in this respect it also prevents lipid peroxidation and accelerates regeneration of inactivated cytochrome oxidase. It inhibits neutrophil adherence to the walls of ischaemic vessels which decreases free radical production, vasoconstriction and tissue destruction. In less severe patients, normobaric 100% oxygen, for at least 6 hours, is thought to be adequate therapy. Usually for patients with more than 25% COHb levels, syncope and seizures, hyperbaric oxygen therapy is preferred (14, 15).

In a patient who was known to have coronary artery disease and whose coronary anatomy had recently been shown by coronary angiography, the recurrence of chest pain was thought to be due to any lesion which had been previously judged to be non-critical so she was taken immediately to the catheter laboratory. She also complained of nausea, vomiting and headache but these nonspecific symptoms could have been seen also in coronary artery disease.

However it is more probably CO intoxication by the multiple mechanisms mentioned above including vasospasm that caused the non-critical stenosis to result in chest pain. So, oxygen therapy instead of percutaneous coronary intervention could have been a more appropriate therapeutic choice.

We concluded that in a patient with non-specific symptoms such as headache, nausea, vomiting especially if he/she has risk factors for CO poisoning like living in places where heating facilities are inappropriate, this insidious pathology should be kept in mind since the diagnosis can be made only if it is suspected.

REFERENCES

1. Kao LW, Nanagas KA. Carbon monoxide poisoning. *Emerg Med Clin North Am* 2004; **22**: 985–1018.
2. Brown SD, Piantodosi CA. In vivo binding of carbon monoxide to cytochrome c oxidase in rat brain. *J Applied Physiol* 1990; **68**: 604–10.
3. Brown SD, Piantodosi CA. Reversal of carbon monoxide-cytochrome c oxidase binding by hyperbaric oxygen in vivo. *Adv Exp Med Biol* 1989; **248**: 747–54.
4. Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med* 1998; **339**: 1603–8.
5. 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**: 1513–60.
6. Allred EN, Bleecker ER, Chaitman BR, Dahms TE, Gottlieb SO, Hackney JD et al. Effects of carbon monoxide on myocardial ischemia. *Environ Health Perspect* 1991; **91**: 89–132.
7. Handa PK, Tai DY. Carbon Monoxide poisoning: a five-year review at Tan Tock Seng Hospital, Singapore. *Ann Acad Med Singapore* 2005; **34**: 611–4.
8. Choi IS. Carbon monoxide poisoning; systemic manifestations and complications. *J Korean Med Sci* 2001; **16**: 253–61.
9. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after exposure to carbon monoxide. *Chest* 1990; **97**: 491–4.
10. Aronow WS. Effect of carbon monoxide on cardiovascular disease. *Prev Med* 1979; **8**: 271–78.
11. Lange RA, Hillis LA. Toxins and the heart. Zipes DP, Libby P, Bonow RO, Braunwald E. *Heart Disease a Textbook of cardiovascular medicine*. 7th ed. Philadelphia. Elsevier Saunders 2005 1731–40.
12. Varol E, Ozaydin M, Aslan Suleyman, Dogan A, Altunbas A. *Anadolu Kardiyol Derg* 2007; **7**: 322–3.
13. Wattel F, Favory R, Lancel S, Nevriere R, Mathieu D. Carbon monoxide and the heart: unequivocal effects? *Bull Acad Natl Med* 2006; **190**: 1961–74.
14. Tibbles PM, Perotta PL. Treatment of Carbon monoxide poisoning: a critical review of human outcome studies comparing normobaric oxygen with hyperbaric oxygen. *Ann Emerg Med* 1994; **24**: 269–76.
15. Tibbles PM, Edelsberg JS. Hyperbaric oxygen therapy. *N Engl J Med* 1996; **334**: 1642–8.