SCIENTIFIC LETTER

Type 2 Acute Myocardial Infarction Secondary to Carbon Monoxide Poisoning: Case Report

Infarto agudo de miocardio tipo 2 secundario a intoxicación por monóxido de carbono: reporte de un caso

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Carbon monoxide (CO) poisoning is a medical emergency of global importance, with a spectrum of clinical presentations ranging from mild and nonspecific symptoms to severe multiorgan involvement and death. Cardiovascular complications are one of the leading causes of morbidity and mortality in patients with CO poisoning and can occur both in the acute phase and in the long term.

We present the case of a 40-year-old male patient with a history of smoking and dyslipidemia who was admitted to our service on January 18, 2025, with loss of consciousness on the previous day secondary to carbon monoxide poisoning, evidenced by a carboxyhemoglobin (COHb) level of 55% on admission.

The admission electrocardiogram (ECG) showed atrial fibrillation rhythm with rapid ventricular response, with ST-segment depression in DI, DII, DIII, aVF, and V3 to V6, and ST-segment elevation in aVR. Ultrasensitive troponin (UST) levels were 593.6/1459/1173 ng/L. The admission echocardiogram showed a left ventricular ejection fraction (LVEF) of 40% with generalized hypokinesia. The condition was interpreted as type 2 acute myocardial infarction (AMI) secondary to CO poisoning, and the patient was admitted to the Coronary Care Unit for monitoring and treatment with oxygen therapy.

A brain and chest computed tomography scan showed no abnormalities, and oxygen therapy with a hyperbaric chamber was administered. The patient was admitted to the intensive care unit in a hemodynamically stable condition, asymptomatic for angina and dyspnea, with no signs of fluid overload or low cardiac output. After hyperbaric chamber therapy, an ECG was performed on the same day, showing sinus rhythm at 75 beats/minute, with no signs of acute or sequelae ischemia, and decreased COHb to 1.2%.

On January 20, 2025, a Doppler echocardiogram revealed a left ventricle with slightly increased wall thickness, no motility disorders, and preserved LVEF (68%). Subsequently, an invasive coronary angiography was performed via the right radial artery, which did not disclose any significant lesions. Laboratory tests during hospitalization included a complete blood count with hematocrit 48.7, hemoglobin 16.4 g/dL, platelets 225 000/mm^3 and leukocytes 8530/mm^3. The lipid profile showed HDL cholesterol 44 mg/dL, LDL cholesterol 130 mg/dL, total cholesterol 195 mg/dL, and triglycerides 180 mg/dL. Glycated hemoglobin (HbA1c) was 5.9%. Serology tests for hepatitis B, hepatitis C, and HIV were nonreactive. Given the patient's favorable clinical course and the absence of criteria for hospitalization, it was decided to discharge him from hospital on January 21, 2025.

Carbon monoxide poisoning can cause a variety of acute and chronic cardiovascular complications, even in patients with no history of heart disease. It exerts its toxicity mainly through tissue hypoxia, mitochondrial dysfunction, and oxidative damage, significantly affecting the myocardium and the vascular system. (1,2)

Acute myocardial injury is observed in approximately 37% to 53% of patients with acute CO poisoning, evidenced by elevated cardiac biomarkers (troponin, CK-MB) and electrocardiographic abnormalities, such as ischemic changes in the ST-segment and T wave. Myocardial injury can occur even in young, previously healthy individuals. (3,4) In the case here presented, elevated troponin and initial electrocardiographic abnormalities are consistent with this finding.

Left ventricular dysfunction may manifest as decreased LVEF or alterations in global longitudinal strain, detectable by echocardiography and cardiac

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magnetic resonance imaging. (4,5) In our patient, left ventricular function was preserved at the time of the echocardiogram.

Carbon monoxide can induce supraventricular and ventricular arrhythmias, including tachycardia, bradycardia, and, in severe cases, ventricular fibrillation. (1,2) The rapid ventricular response to atrial fibrillation initially observed in our patient is an example of this complication.

Cases of AMI secondary to CO poisoning have been documented, attributable to both hypoxia and endothelial dysfunction, and coronary vasospasm. (2,5). The present case is a clear example of type 2 AMI induced by poisoning. The absence of significant coronary lesions on coronary angiography supports this classification.

Cohort studies have shown that patients with a history of CO poisoning have a significantly increased risk of major cardiovascular events, including AMI, heart failure, and stroke, compared with the general population (adjusted HR ≈ 2). (6)

The risk of mortality from cardiovascular causes remains high after poisoning, even after normalization of biomarkers and initial cardiac function. Cardiac magnetic resonance imaging can detect persistent myocardial fibrosis several months after poisoning, which is associated with subclinical dysfunction and potential risk of future heart failure. (5)

In severe cases, poisoning can cause cardiovascular collapse, severe hypotension, and cardiac arrest, especially with massive or prolonged exposure. (1,2)

In conclusion, CO poisoning is a clinical entity that requires high suspicion and comprehensive management, given its potential serious cardiovascular complications. The case presented here illustrates the manifestation of a type 2 AMI in the context of CO poisoning, highlighting the importance of cardiac monitoring and complementary studies for proper diagnosis and treatment. Long-term follow-up of these patients is essential due to the increased risk of cardiovascular events and mortality.

Conflicts of interest

None declared.

(See conflicts of interest forms on the website).

Ethical considerations

Not applicable

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