Acute kidney failure complicating carbon monoxide poisoning

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Abstract

Background. Carbon monoxide, albeit common, is rarely associated with renal failure. We report a case of CO-associated kidney failure requiring short-term dialysis.

Case report. A 33-year-old male was found unconscious in a bathroom equipped with a propane-gas heater. The duration of exposure to carbon monoxide was unknown. The patient was transported to a regional hyperbaric centre; the carboxyhaemoglobin concentration in the blood on admission was 38.3%. After 60 minutes of exposure to hyperbaric oxygen, he regained consciousness and was transferred to the toxicology department. Mild rhabdomyolysis with acute kidney failure was diagnosed and despite two subsequent hyperbaric sessions, haemodialysis was necessary. The kidney failure resolved within two weeks, and the patient made a full recovery.

Discussion and conclusions. Carbon monoxide mainly affects the central nervous system and the myocardium; renal failure may occur due to rhabdomyolysis and hypoxia. Therefore, all CO-poisoned patients should be closely monitored for their renal function.

Key words: complications, kidney failure; gases, carbon monoxide, poisoning

Carbon monoxide (CO) is a colourless, odourless and non-irritating gas. CO is the commonest cause of inhalatory intoxications worldwide and the third leading cause of all intoxications in Poland [1, 2]. Moreover, it is the main cause of intoxication-associated deaths in Great Britain and accounts for 50,000 admissions to emergency departments per year in the United States [3, 4]. One of the sources of CO is incomplete combustion of carbon compounds contained in natural gas. The majority of accidental intoxications result from malfunctions of household devices and/or faulty ventilation and air conditioning systems.

We report a case of CO intoxication complicated by acute kidney failure requiring short-term haemodialysis.

CASE REPORT

A 33-year-old male was found unconscious in a bathroom equipped with a propane heater. The family members failed to estimate the duration of his exposure to carbon monoxide (most likely several hours). The symptoms of respiratory failure were observed, therefore endotracheal intubation was performed and ventilation with 100% oxygen initiated; due to suspected CO intoxication, the patient was immediately transported to a regional centre for hyperbaric therapy. On admission, the blood concentration of carboxyhaemoglobin (COHb) was 38.3%. During the first hyperbaric session (60 min of respiration with 100% oxygen at 2.5 atm.) the patient regained consciousness. After the session, COHb decreased to 1.9%. The patient’s general condition improved, his respiration was efficient; he was extubated and transferred to the toxicology department.

On admission, the patient was fully conscious, with proper verbal-logical contact and memory deficits covering several hours. The examinations did not demonstrate respiratory or circulatory insufficiency; neither neurological features of focal nervous system damage nor clinical manifestations of rhabdomyolysis were observed. The detailed history taking revealed the history of arterial hypertension treated irregularly.

The laboratory tests performed on admission disclosed: serum creatinine 150.2 μmol L⁻¹, ALAT 105 U L⁻¹, ASPAT 103 U L⁻¹, CPK 1917 U L⁻¹. The serum concentration of troponin I was elevated to 1.7 ng mL⁻¹, yet no features of myocardial ischaemia were found on ECG. The only abnormality of acid-base balance of arterial blood was high oxygen pressure — 300 mm Hg (40 kPa).

Due to rhabdomyolysis, intensive supply of crystalloids and 8.4% sodium bicarbonate was started to alkalinate the urine. The oxygen therapy was continued, 5 L min⁻¹. The
next two hyperbaric sessions were carried out during the subsequent days of hospitalisation.

Despite the standard therapy used, the renal function deteriorated, i.e. an increase in serum creatinine to 842.4 μmol L⁻¹ at initially normal diuresis. The abdominal ultrasound scan was normal. The CPK activity was continuously monitored; its highest value (2500 U L⁻¹) was found on hospitalisation day 2. Since the arterial pressure was high, hypertensive treatment was instituted. Moreover, due to elevated indices of nitrogen kidney damage and reduced diuresis, haemodialysis were initiated on hospitalisation day 5, every other day. On hospitalisation day 11, the patient was transferred to the department of nephrology because of the lack of improvement in renal functions. The serum concentration of creatinine was 839.8 μmol L⁻¹ and of urea 39.1 mmol L⁻¹. Additionally, the serum levels of phosphates and uric acid were elevated.

In the department of nephrology, intermittent haemodialysis sessions were continued and intensive treatment was carried out. The repeated abdominal US examination with Doppler option showed unevenly increased echogenicity of the renal parenchyma, partially blurred sinus-parenchyma border and hypoechochogenic pyramids (features of oedema). The Doppler examination of renal arteries was normal. The parameters of vascular resistance in segmental arteries were also normal. The urine test results demonstrated low specific weight (1.005 g µL⁻¹) and protein concentration of 75 mg dL⁻¹. ECG did not show any abnormalities; echocardiography disclosed the cardiac cavities of proper sizes, with good contractility, and proper diastolic and systolic functions of the myocardium. The left ventricular ejection fraction was 65% and the interventricular septum thickness 1.0 cm. The fundus examination did not reveal any pathology.

During the subsequent days of hospitalization, the renal function gradually improved. After about 2 weeks, the renal replacement therapy was discontinued. The patient was discharged from hospital after approximately 3 weeks of hospitalization. On discharge, the serum concentration of creatinine was 176.8 μmol L⁻¹ and of urea 12.9 mmol L⁻¹. The urine test did not show the presence of proteins; the activity of creatinine kinase, an indicator of rhabdomyolysis, was not high. Additionally, to prevent acute renal failure, forced diuresis was used, the urine alkalised and electrolyte abnormalities stabilised. The renal failure was most likely caused by CO toxic effects on the vascular endothelium, which resulted in the release of reactive chemical compounds with activation and/or adhesion of neutrophils. This in turn caused progressive damage to blood vessels, which led to enhanced lipid peroxidation [9].

The differential diagnosis of acute renal failure in CO-intoxicated patients should consider the diseases, which alone may exacerbate chronic renal failure, such as diabetes mellitus, arterial hypertension or atherosclerosis, particularly

**DISCUSSION**

Depending on the air concentration of carbon monoxide and duration of exposure, CO intoxication can induce mild or severe clinical symptoms. The exposure to low levels of CO mainly causes headaches, dizziness, muscle pains, and neuropsychological disturbances. In contrast, the exposure to its high levels leads to confusion, loss of consciousness or even death [5].

Elevated concentrations of COHb in blood confirm the diagnosis of CO intoxication. It should be stressed, however, that the highest correlation between increased clinical symptoms and levels of COHb is observed immediately after cessation of CO exposure. Thus, in practice, enhanced clinical manifestations are found at low concentrations or lack of COHb in blood.

Carbon monoxide intoxications mostly induce neurologic and neuropsychological disturbances. Additionally, the toxic effects of CO may affect the cardiovascular and respiratory systems, muscles, liver and kidneys. The incidence of CO intoxication-associated complications has not been precisely known. According to the French study encompassing 385 patients intoxicated with CO, the commonest complications involve the central nervous system and the gastrointestinal tract [6]. The renal failure cases due to CO intoxication reported in literature were associated with severe rhabdomyolysis and myoglobinuria [7].

The major cause of tissue and organ damage following CO intoxication is hypoxia, which develops due to the formation of carboxyhaemoglobin and a leftward shift of the haemoglobin dissociation curve. The haemoglobin binding affinity for CO is over 200 times higher than its affinity for oxygen. CO binds haemproteins impairing the mitochondrial function, which leads to enhanced oxidative stress, formation of oxygen reactive species, neuron necrosis and apoptosis. Impaired cellular respiration induces the stress response, activating the hypoxia-inducible factor 1 alpha. Moreover, destructive effects of CO result from its negative impact on many hypoxia-independent metabolic pathways, which damages the nervous tissue and the myocardium [8].

In our case, hyperbaric therapy was decided due to high concentrations of carboxyhaemoglobin, short time between exposure and provision of help, and loss of consciousness. Despite the therapy instituted and quickly reduced carboxyhaemoglobin levels, the patient developed acute renal failure, which in this case should not be linked only with muscle damage and myoglobin release since the activity of creatinine kinase, an indicator of rhabdomyolysis, was not high. Additionally, to prevent acute renal failure, forced diuresis was used, the urine alkalised and electrolyte abnormalities stabilised. The renal failure was most likely caused by CO toxic effects on the vascular endothelium, which led to enhanced lipid peroxidation [9].
in the elderly. Furthermore, the drugs used should be taken into account, which increase renal damage. The essential part of management of patients with acute renal failure is the most meticulous examination to disclose the conditions promoting this complication, e.g. fever or water-electrolyte imbalance resulting from vomiting, diarrhoea, etc. [10].

In the present case, acute renal failure developed dynamically. Within a few post-intoxication days, haemodialysis was required. Fortunately, the renal function quickly improved, which enabled the discontinuation of renal replacement therapy. Further months of observation and treatment should evidence whether the patient sustained permanent renal failure.

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Received: 14.10.2011
Accepted: 21.01.2012

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