CASE REPORT

Fatal chemical pneumonitis due to cadmium fumes

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Acute exposure to high concentrations of cadmium fumes may cause acute chemical pneumonitis with a possibly fatal outcome. The etiologic diagnosis of acute cadmium intoxication from inhaled fumes may be difficult and can be confused with other forms of acute respiratory failure. We report on a case of a fit 53 year-old man who was exposed to cadmium fumes after flame-cutting an alloy containing around 10% of cadmium for a period of 60–75 minutes. He developed severe chemical pneumonitis and died 19 days after exposure.

Key words: Cadmium; chemical pneumonitis; poisoning; heavy metals.

INTRODUCTION

Cadmium is a by-product of the zinc and lead industry. It is mainly used in metal plating and in the production of batteries, pigments, plastic stabilizers and some alloys. The metal melts at 320°C and boils at 767°C but when heated it readily oxidizes in air and may ignite, producing yellowish-brown cadmium fumes which tend to settle as fine dust on cold objects nearby.

We report a fatal case of acute respiratory insufficiency after the inhalation of cadmium fumes.

CASE REPORT

A 53 year-old male who was referred to our Intensive Care Unit (ICU) from another hospital centre where he had been diagnosed as having acute respiratory insufficiency secondary to pneumonia.

The patient, a nonsmoker, had no particular medical antecedents and no history of drug abuse. For 23 years he had worked in the assembly, installation and repair of heating boilers.

On the day of his admission to the first hospital he had experienced a series of respiratory symptoms, consisting of a dry cough and thoracic pain with pleuritic features. This occurred after the patient had cut an unknown alloy with a butane torch for a duration of 60–75 min. He had been repairing a vat which had been used to treat aluminium profiles. The bottom of the vat was covered with an alloy melting at 72°C. The patient filled the vat with hot water after having removed the entire deposited mass and he then cut it with a butane torch. He did not use any type of personal respiratory protection and there was no extraction system in the premises.

Four hours after finishing the work he presented progressive dyspnoea and had to be admitted into hospital. On the third post-inhalation day his clinical condition worsened, and he became hypoxemic and a chest X-ray revealed a bilateral interstitial pattern. He was transferred to the ICU. The week previous to admission, he displayed cough, fever and chest pain. With the suspicion of a respiratory infection, antibiotic treatment was started using cefotaxime, erythromycin and rifampin. Hemocultures, urine-cultures and sputum cultures, bronchoalveolar lavage (BAL) and bronchoaspiration (BAS), were considered normal. Confronted with bad clinical evolution, corticoid treatment was added on the fifth day without a favourable response. On the seventh day orotracheal intubation was required because of clinical and functional deterioration, and the patient was transferred to our centre.

Physical examination on admission showed an arterial pressure of 120/60 mmHg; diffuse crepitations were heard by auscultation over both lungs. Radiological examination revealed bilateral 'white lungs'. The most important analytical results were: leucocytes 15.700/mm³, platelets 274.000/mm³, Quick’s time 50%, partial thromboplastin time = 50 s (control = 33 s) and serum...
creatinine 1.13 mg/100 ml. Artificial ventilation was carried out with a frequency of 20 breaths per minute, FiO₂ of 0.6 and positive end-expiratory pressure (PEEP) of 10 cm H₂O; PₐCO₂ was 60 mmHg and PₐO₂ of 58 mmHg.

The clinical evolution was unfavourable with progressive hypoxemia with PₐO₂ of 60–70 mmHg and hypercapnia with PₐCO₂ of 65–75 mmHg despite FiO₂ of 1, PEEP of 15 cm of H₂O and minute volume of 16–18 litres. Pulmonary artery pressure was elevated with mean pulmonary arterial pressure between 35–55 mmHg, pulmonary capillary wedge pressure < 15 mmHg, cardiac output of 8 l/min and systemic vascular resistance (SVR) of 600 dyne/s/cm⁵.

Faced with this negative clinical evolution, antibiotic treatment was suspended for 24 hours and on the tenth post-inhalation day, new hemocultures, urine cultures, tracheal aspiration, serologies, BAL, and checks for viruses, mycobacteria, fungi and parasites were carried out, all with negative results.

The composition of the alloy cut was analyzed using X-ray fluorescence techniques and revealed bismuth 50%, lead 26%, tin 13%, cadmium 10% and 1% of impurities (iron, zinc, copper and nickel).

Fifteen days after the fume inhalation, the concentration of cadmium in the blood and urine was analyzed by atomic absorption spectrometry with graphite oven in STPF conditions (platform tubes at a stabilized temperature).¹ ²

The blood and urine concentration of cadmium was: 0.34 μg/100ml (control = 0.11 μg/100ml) and 17.6 μg/g creatinine (control = 0.2 μg/g creatinine). According to the American Conference of Government Industrial Hygienists (ACGIH) 1993–94, the maximum concentrations permitted for workers who are exposed to cadmium are (after a working day) 5 μg/l in blood and 5 μg/g creatinine in urine.

On the 15th day after the fume inhalation, the patient presented refractory hypoxaemia and severe hypercapnia (PₐCO₂ of 100mmHg), with haemodynamic instability requiring the administration of high doses of inotropic drugs (dopamine and noradrenaline) and multi-organ failure. He died on the nineteenth day after the intoxication.

At autopsy the main pathologic findings were found in the lungs which showed a typical picture of diffuse alveolar damage with beginning interstitial and intra-alveolar fibrosis. Concentrations of cadmium were 823 ng/g in the liver, 3,571 ng/g in the kidney and 1,143 ng/g in the lung.

**DISCUSSION**

Cadmium may be absorbed through inhalation or ingestion. After the inhalation of cadmium fumes, 10–50% may be absorbed, depending on particle size and chemical composition. Gastrointestinal absorption is usually less than 10% but may be increased when iron, protein, calcium or zinc deficiencies are present.

Cadmium mainly accumulates in the kidneys and the liver, organs which account for roughly 70% of the total body burden. The half-life of cadmium is about 6 weeks for the fast compartment of cadmium concentrations in blood.³

Excretion is primarily through the kidney, but elimination of cadmium accumulated in an organism is very slow with a half-life of 20–40 years.

Acute exposure to moderate concentrations (200–500 (μg/m³) of cadmium fumes can cause the symptomatology of 'metal fume fever' (metallic taste, fever, malaise, joint pains, cough, sore throat, chest tightness and fatigue) and the syndrome lasts for one to two days. However a very intense exposure may cause pulmonary edema and pneumonitis,⁴ with death due to respiratory failure.⁵-¹² Pulmonary fibrosis in a survivor of a single exposure to cadmium fumes has been reported.¹³

Chronic cadmium exposure by inhalation has been reported to cause emphysema, anemia, renal insufficiency, slight liver damage, anosmia, yellowing of teeth, shortness of breath and fatigue.¹⁴ ¹⁵

Our case was initially diagnosed as pneumonia which caused an acute respiratory insufficiency. The negative microbiological results and the occupational history directed our inquiries to the diagnosis of chemical pneumonitis due to cadmium fumes, since the other components have not been associated with lung injury.¹⁶ However, this diagnosis did not alter the course of the disease.

The cadmium concentrations found in the blood and urine of the patient are similar to those found by Taylor et al.,¹⁰ although the latter were determined two days after intoxication, whilst we carried out these measurements 2 weeks after exposure. Preventive measures such as technological improvements in the workplace, worker education about the potential hazards of the substances they work with, appropriate ventilation, and, if necessary, use of personal protective equipment, are of primary importance in these cases.

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**REFERENCES**


2. Ony CN, Chua LH, Lee BL, Ong HY, Chia KS. Electrothermal atomic absorption spectrometric. Determination of cadmium...


