

Fatal cadmium-induced pneumonitis

by Kristin Seidal, MD,¹ Nils Jörgensen, MD,¹ Carl-Gustaf Elinder, MD,² Bengt Sjögren, MD,³ Marie Vahter, PhD⁴

SEIDAL K, JÖRGENSEN N, ELINDER C-G, SJÖGREN B, VAHTER M. Fatal cadmium-induced pneumonitis. *Scand J Work Environ Health* 1993;19:429–31. A previously relatively healthy 78-year-old man was exposed to cadmium fumes during brazing with cadmium-containing silver solder. He developed severe chemical pneumonitis and died 25 d after exposure.

Key terms: case report.

Cadmium is a soft, silvery, and ductile metal. Its production and consumption have increased considerably since the beginning of this century. Cadmium is used worldwide in electroplating, in batteries, and as pigments and plastic stabilizers (1).

Hard solders mainly contain copper and zinc, but cadmium is sometimes added to lower the melting point. The most commonly used hard solders of this type contain 15–25% cadmium (2). Cadmium is the most toxic metal in cadmium-containing hard solders. Exposure to this metal has been associated with fever, leukocytosis (3), and pulmonary edema in exposed brazers during short-term exposure (4). Long-term brazing has resulted in increased excretion of beta₂-microglobulin in urine as an expression of tubular kidney lesion (5).

In this case report we describe one brazer who developed chemical pneumonitis and respiratory insufficiency after brazing with cadmium-containing hard solder.

Case report

A 78-year-old nonsmoking man sought medical attention on 14 November 1986 because of continuous cough during the previous 3 d. He had otherwise been healthy, except for mild symptoms of angina pectoris without pulmonary symptoms. Sotalol hydrochloride (80 g daily), a beta-receptor blocker, was prescribed. At the time he was retired but had previously worked as a log floater and tool maker. Nothing exceptional was discovered in his physical

examination. His temperature was 38.3°C, his white blood cell count was $14\,600 \cdot \text{mm}^{-3}$, and the sedimentation rate was $75 \text{ mm} \cdot \text{h}^{-1}$. A chest X-ray revealed alveolar infiltrates on the base of the right lung. These findings were interpreted as pneumonia, and the patient was given penicillin V, 2 g orally per day.

Two days later, on 16 November, the patient returned with a sore throat and dyspnea. His lips were cyanotic and the mucous membranes in his throat were red and dry. At this point he explained that his cough appeared 3 h after brazing on stainless steel. He was now admitted to the hospital with the suspicion of having toxic pneumonitis.

It was revealed that, on 11 November, our patient and a friend had been manufacturing an apparatus for the illegal production of alcohol in his garage. While working, the patient had kept the metal pieces directly in front of him, without using any respiratory protective equipment. His friend had been brazing for about 15 min with a silver solder, later analyzed to contain 20–30% cadmium. The friend used respiratory protection during the brazing operation and was consequently less exposed. The friend developed cough, shivering, and fever, but felt well the next morning and did not consult his physician.

When admitted to the hospital on 16 November, the patient was initially treated in the intensive care unit. His partial pressure of arterial oxygen was 3.9 kPa, and he needed a Bird-mask the first 2 d, but his respiration did not have to be assisted in any other manner. He used his accessory breathing muscles and had tachypnea and inspiratory rhonchi at auscultation. He was treated with oxygen and bronchodilators. He was not treated with steroids, as 5 d had passed since the exposure. Benzylpenicillin (3 g) was given intravenously three times a day during the next 4 d.

On 20 November his obstructive symptoms became worse and an intravenous infusion containing 4 mg of betamethasone and 15 ml of theophyllamine was given twice daily during the next 5 d. The steroid treatment was increased to 8 mg of betamethasone,

¹ Department of Lung Medicine, Central Hospital, Karlstad, Sweden.

² Department of Renal Medicine, Huddinge Hospital, Huddinge, Sweden.

³ Department of Occupational Medicine, National Institute of Occupational Health, Solna, Sweden.

⁴ Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden.

Reprint requests to: Dr K Seidal, Department of Lung Medicine, Central Hospital, S-651 85 Karlstad, Sweden.

given intravenously four times, without any significant effect.

The patient's condition deteriorated slowly, and he died on 6 December due to respiratory insufficiency. During the course of the illness his serum creatinine levels were 85, 124, 109 $\mu\text{mol} \cdot \text{l}^{-1}$; in other words they were generally within the reference interval of 55–115 $\mu\text{mol} \cdot \text{l}^{-1}$. Spirometry was not performed.

Autopsy

In the autopsy the patient's lungs were large and firm with the light microscopic appearance of extensive bronchopneumonia and pronounced peribronchial, perivascular, and interstitial fibrosis, a finding which may suggest that some of the changes were not caused by this acute intoxication. The patient's heart was slightly enlarged with no signs of recent or previous infarction. The coronary arteries were moderately arteriosclerotic. The kidneys were of normal size, somewhat granulated on the surface but with normal color and consistency. Microscopy was not performed for the kidney. The liver was macroscopically normal.

Cadmium concentrations

The concentration of cadmium in the patient's blood was about 530 $\text{nmol} \cdot \text{l}^{-1}$ at autopsy. His lungs contained 1.6 μg of cadmium per gram of wet weight (range of four samples 0.76–3.6 $\mu\text{g} \cdot \text{g}^{-1}$), the kidney cortex 23 $\mu\text{g} \cdot \text{g}^{-1}$ (range of four samples 20–26 $\mu\text{g} \cdot \text{g}^{-1}$), and the liver 1.9 $\mu\text{g} \cdot \text{g}^{-1}$ (range of four samples 1.8–1.9 $\mu\text{g} \cdot \text{g}^{-1}$). Cadmium was determined with graphite furnace atomic absorption spectrophotometry (6) at the Institute of Environmental Medicine, Karolinska Institute. The cadmium content of the solder was analyzed at the same laboratory.

Discussion

The brazer was exposed to cadmium fumes giving rise to a blood level of 530 $\text{nmol} \cdot \text{l}^{-1}$, a value indi-

cating significant exposure. However, postmortem blood samples should be interpreted with caution. With a half-time of about 100 d for the fast compartment of cadmium concentrations in blood (7), it can be estimated that the blood concentration at a time close to the exposure was about 630 $\text{nmol} \cdot \text{l}^{-1}$. In Sweden the median level of cadmium in blood among occupationally unexposed persons is about 2 $\text{nmol} \cdot \text{l}^{-1}$ for nonsmokers and about 16 $\text{nmol} \cdot \text{l}^{-1}$ for smokers (6). According to Swedish regulations (8), the blood concentration of cadmium should not exceed 100 $\text{nmol} \cdot \text{l}^{-1}$ among occupationally exposed subjects.

The concentration of cadmium in the lungs immediately after exposure was probably higher than at the time of death. A rough figure, based on the estimated half-time in blood, would be about 1.9 g of cadmium per gram of lung tissue. As shown by table 1, the concentration of cadmium in the lungs of our patient, as well as in all other reported cases leading to death, was clearly above the level of occupationally unexposed men.

Lethal doses of cadmium fumes have been calculated to be 2600–2900 $\text{mg} \cdot \text{m}^{-3} \cdot \text{min}^{-1}$ (13), for example, 260 $\text{mg} \cdot \text{m}^{-3}$ during 10 min or 8.6 $\text{mg} \cdot \text{m}^{-3}$ during 5 h, as reported by Beton et al (14). These estimations are based on the measured cadmium concentration, the weight of the lungs, the estimated retention of inhaled cadmium, and the respiratory minute volume. Information was lacking on the weight of the lungs of our patient. The time of soldering was 15 min, but the true exposure time was probably longer, as the patient spent time in the garage after the soldering activity.

Pulmonary fibrosis after a single exposure to cadmium fumes has previously been reported by Townsend (15). In 1963, a welder developed pulmonary edema after welding a cadmium-silver alloy (16). His lung function gradually improved during the first six months. Seventeen years later this man developed severe progressive pulmonary fibrosis (15).

A 30-year-old man worked with silver solder for 1 h in a small, enclosed, unventilated tank. Later that

Table 1. Concentrations of cadmium in lung, kidney, and liver from autopsies.

Study	Lung ($\mu\text{g} \cdot \text{g}^{-1}$ wet weight)	Kidney ($\mu\text{g} \cdot \text{g}^{-1}$ wet weight)	Liver ($\mu\text{g} \cdot \text{g}^{-1}$ wet weight)	Days after exposure
Occupationally unexposed men, 30–79 years of age (1, 9)	0.1–0.7	13–19 ^a	0.5–1.0	
Beton et al 1966 (14)	2.5	5.7	3.2	5
Blejer et al 1966 (4)	4.1	—	5.0	4
Lucas et al 1980 (10)	4.7	31	4.8	5
Patwardhan 1976 (11)	1.5	—	2.3	3.5
Winston 1971 (12)	1.5	5	1	5
Present case	1.6	23 ^a	1.9	25

^a Kidney cortex.

evening he developed dyspnea and cough, but he did not seek medical attention. Pulmonary function tests two weeks later revealed moderate restrictive impairment (total lung capacity 3.3 l) and moderately decreased single breath diffusing capacity. His diffusing capacity returned to normal within two months and his total lung capacity improved continuously but remained below normal (5.0 l versus 6.3 l expected) nearly four years after the exposure (17).

Another man spent two weeks brazing the propellers of ships with a cadmium-containing solder in a confined area with poor ventilation. He developed fibrosing alveolitis, which was treated with prednisolone. His vital capacity was initially 2.8 l, but it improved gradually to 5.5 l after more than a year (18).

Short-term exposure to high levels of cadmium fumes can cause pulmonary edema and pneumonitis. Pulmonary fibrosis is more seldom reported after such exposure. In our case pneumonitis appeared shortly after exposure and the man in question later developed respiratory insufficiency, which was the cause of death one month after exposure. Information on the true exposure was probably delayed due to the illegal character of the patient's activity. An immediate start of steroid treatment is important, and it might have changed the outcome for this patient. This tragic case report emphasizes the need for strict hygienic control measures whenever the highly toxic metal cadmium is handled.

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Received for publication: 19 May 1993