Z Rechtsmed (1983) 91 : 139–143

Zeitschrift für
Rechtsmedizin

An Acute Fatal Occupational Cadmium Poisoning by Inhalation

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Summary. A 43-year-old male smelter was admitted to a hospital on account of severe dyspnea about 2 days after exposure to brownish-yellow smoke produced by melting of "copper" scrap. On admission pronounced hypoxemia was revealed, and an oxygen-enriched gas was administered after intubation. Although inspired oxygen concentration was gradually increased, hypoxemia progressed and he died on day 11 in hospital.

The principal autopsy finding was chiefly confined to the lungs. Both lungs were heavy (the left weighing 1,470 g; the right 1,710 g) and firm to the touch. Histologically, no normal alveoli were found throughout the entire lung. Some alveolar spaces were occupied by pneumocytes, others by organized exudate with fibrosis. Interstitial fibrosis was present. Patchy areas of inflammatory cell infiltrations as well as intra-alveolar hemorrhages were observed. On the basis of the above findings a diagnosis of diffuse alveolar damage was made.

Based on the available evidence (presence of cadmium in the "copper" scrap, feature of the smoke, clinical signs with latent time, and high cadmium concentration of the lung), the diffuse alveolar damage was considered to have been caused by inhaled cadmium. The pulmonary change of the present case was more advanced in pathologic stage in comparison with those reported in the literature.

Key words: Cadmium poisoning by inhalation – Pulmonary fibrosis – Alveolar damage by cadmium poisoning

Zusammenfassung. Ein 43jähriger Schmelzer wurde dem beim Schmelzen von „Kupferabfall" entstehenden bräunlich-gelben Rauch ungefähr 5 Stunden lang ausgesetzt. Nach 2 Tagen trat eine schwere Dyspnoe ein. Bei Aufnahme in ein Krankenhaus wurde eine schwere Hypoxämie entdeckt. Nach der Intubation wurde ein sauerstoffreiches Gasgemisch verabreicht. Die Sauerstoffkonzentrationen der Einatmungsluft wurden allmählich gesteigert, jeoch schritt der Grad der Hypoxie fort. Der Kranke starb 12 Tage nach dem Unfall.

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Die wesentlichen Sektionsbefunde waren auf die Lunge beschränkt. Beide Lungenflügel waren schwer (die linke wog 1470 g und die rechte 1710 g) und derb. Histologisch wurden nirgends normale Alveolen gefunden. Mehrere Alveolarräume enthielten abgelöste Alveolarepithelien oder organisiertes Exsudat. Unter Berücksichtigung der oben genannten Befunde lautete die Diagnose „diffuse alveoläre Schädigung".

Aufgrund des vorhandenen Kadmiums im „Kupferabfall", der Eigenschaft des Rauches, dem klinischen Bild und der hohen Kadmiumkonzentration in der Lunge wurde die Ursache der Lungenveränderungen dem Kadmiumdampf im Rauch zugerechnet. Verglichen mit den Lungenveränderungen, die in ähnlichen Fällen beschrieben wurden, waren die Veränderungen in diesem Falle in weit fortgeschrittenerem Stadium.

Schlüsselwörter: Kadmiumvergiftung, Inhalation - Lungenfibrose, alveoläre Schädigung durch Kadmiumdämpfe

There have been several reports of acute accidental cadmium poisoning by inhalation [1-3, 6, 8], the mortality of which has been estimated to be at approximately 16% [7].

The present case, concerning an acute fatal poisoning by cadmium fume produced by melting of cadmium-contaminated copper scrap (waste copper fuse), showed pulmonary pathologic changes which had not been reported previously in the literature and were considered worth publishing.

Case Report

The task of M.I. (the victim, a 43-year-old male employee in a copper alloy manufacture) and his 40-year-old co-worker on the day in question was to smelt copper scrap in a furnace and to cast it in a mould. Three "charges", a "charge" being a process consisting of smelting and casting, were to be carried out. The first "charge" was started at about 8:30 a.m. Half an hour after 850 kg of material was put into a furnace, brownish-yellow smoke began to develop. As the working place was ventilated only poorly, the two workers were exposed to the smoke. Since the smoke per se was not exceedingly irritating, causing no immediate symptoms, they continued to work until the end of the second "charge", at which time they began to feel unwell. The third "charge" was not done. By that time, they had been exposed to the smoke, although intermittently, for about 5 h. Besides feeling general fatigue with nausea, M.I. complained of a pain in his throat. The next day his condition remained unchanged. Two days after the accident, he began to develop breathlessness and consulted a neighboring physician, who sent him to a hospital in the neighborhood.

The blood gas analyzed on admission to the hospital revealed a severe hypoxemia (blood P_aO_2 42 mm Hg), and a chest radiograph showed a diffuse shadow. He was immediately transported to the intensive care unit of the hospital, where after intubation his airway was connected to a respirator, and an oxygen-enriched (oxygen concentration about 40%) gas was administered while applying a continuous positive airway pressure (CPAP). Although inspired oxygen concentration was gradually increased (from day 5: to 50%; from day 6: to 70-100%; from day 7: to 80-100%; from day 8 onward: to 100%), hypoxemia progressed. A chest radiograph taken on day 5 showed ground-glass appearance. The patient had frequently a bad cough with slightly viscid, sometimes blood-tinged, sputum which had to be aspirated. His body temperature sometimes increased to as high as 38.3° C. The urine output varied from 850 to 3,000 ml, and macroscopic hematuria was noted occasionally. He died on day 11 in hospital, 12 days after the accident.

His co-worker, who also complained of breathlessness and consulted another physician 3 days after the accident, recovered without serious consequences.

Autopsy Findings

Autopsy carried out 18 h post mortem showed a slightly ill-nourished man, 158 cm in height and 50 kg in weight. The purplish-red lividity was observed in the dorsal aspect of the trunk. There was a bedsore, measuring 1 cm × 3 cm in size, on the lower part of the back. No wound except one due to tracheotomy was found. The principal internal findings were confined to the lungs. Both lungs were heavy, the left weighing 1,470 g and measuring 19 cm × 23 cm × 4.2 cm; the right 1,710 g and 28 cm × 21 cm × 5.5 cm, and firm to the touch. The surface was purplish-red and scattered with minute petechiae. The color of the cut surface was darker than that of the external surface and a moderate amount of fluid was expressed. There was 90 ml of reddish-yellow fluid in the left pleural space. The heart, weighing 350 g, was apparently normal. There was not much free fluid in the peritoneal cavity. The liver, kidney, trachea, esophagus, and the other organs were normal on naked-eye examination.

Histological Findings

Histologically, no normal alveoli were found throughout the entire lung. Some alveolar spaces were occupied by mononuclear cells, which were considered to be pneumocytes, others by organized exudate (Fig. 1). Fibroblasts were proliferated both interstitially and intra-alveolarly (Fig. 2). Patchy areas of infiltrations of leukocytes as well as intra-alveolar hemorrhages were observed. Based on the findings described above a diagnosis of diffuse alveolar damage was

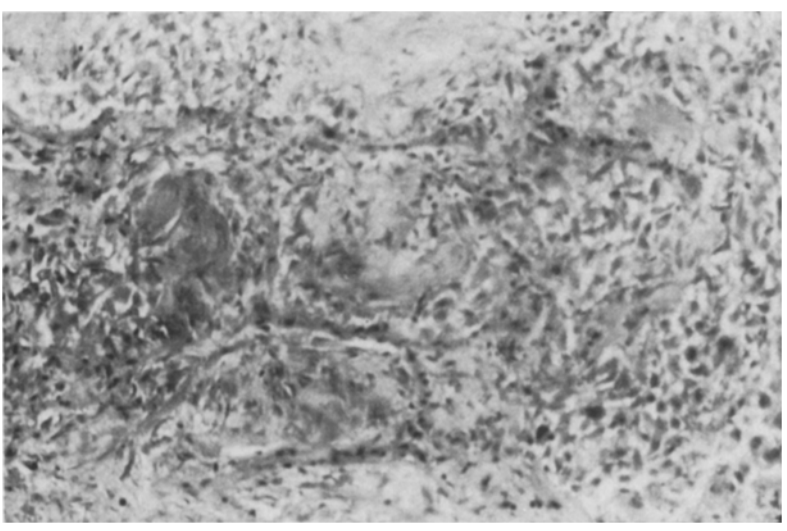

Fig. 1. The lower right alveolus is occupied by mononuclear cells, considered to be pneumocytes. In other alveoli, the exudate is organized. HE, × 100

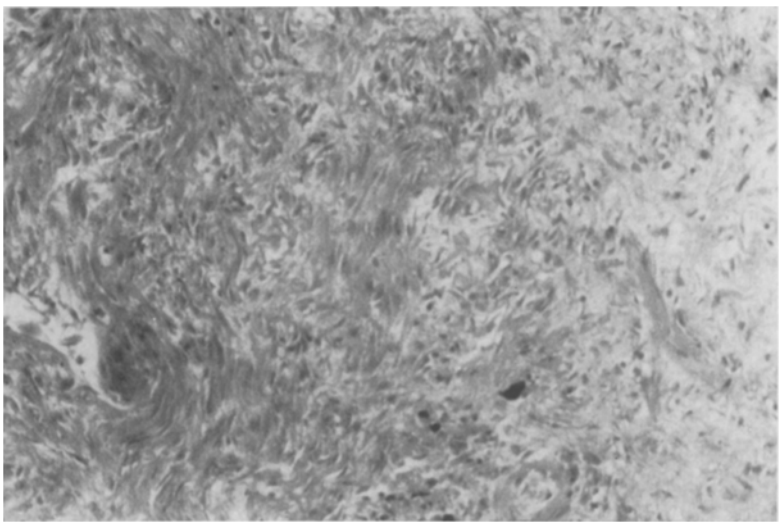

Fig. 2. Fibrosis occurs both interstitially and intra-alveolarly. The normal structure is destroyed. HE, ×100

made [4]. In the liver fatty degeneration was present around the central vein. The renal tubular epithelia showed degenerative change, almost all cells having lost nuclei. A small number of casts were present in the tubular lumen. Heart muscles showed small foci of sarcolytic and fibrotic change.

Toxicologic Findings

The cadmium concentration of the right upper lobe of the lung was determined atomic-absorption-spectroscopically to be 1.06 μg/g of wet specimen. This value was about 30% higher than that reported by Yamada et al. on adult Japanese (p. 59 in the collection of summaries of the communications presented at the 57th Conference of the Medico-legal Society of Japan).

Discussion

The marked pathologic change of the lung, diagnosed as diffuse alveolar damage and responsible for severe hypoxemia, was considered to have been caused by some injurious agents present in the brownish-yellow smoke. From the feature of the smoke (having been brownish-yellow and not exceedingly irritating) and the clinical signs with latent time, cadmium was once suspected as a causative agent. Cadmium concentrations in the blood (6 μg/ml) and urine (332 μg/ml), taken on days 2 and 5 in hospital, respectively, have been interpreted as showing possible exposure. The presence of cadmium in the copper scrap, which was later confirmed by a X-ray microanalyzer, and high cadmium concentration in the lung definitely showed that cadmium was present in the smoke in question. According to the reconstruction experiments conducted on the copper scrap later on, evolved fume contained high concentrations of cadmium.

Diffuse alveolar damage can occur to a variety of injurious agents including cadmium, and from pulmonary pathology alone no inference can be drawn as to the injurious agent [4]. However, on the basis of the above evidence it can be concluded safely that the lung damage was caused by inhaled cadmium. The victim has probably also been exposed to fumes of copper and zinc, the pulmonary toxicity of which is, however, considered to be less than that of cadmium. As a characteristic feature of the pulmonary histopathology of acute cadmium poisoning by inhalation, hyperplasia of pneumocytes has been mentioned and illustrated in some reports [1, 2,6]. In the present case, it was interpreted that hypertrophied pneumocytes were shed from the alveolar walls and accumulated in the alveolar spaces.

The pulmonary changes in the present case were at an advanced stage, differing from those reported in previous literature [1, 2, 6] in that alveolar exudate had undergone extensive organization and even fibrosis, and that interstitial fibrosis had occurred. The difference in stage can be explained chiefly by the longer survival time of the present case in comparison with reported cases, the survival of which ranged from 5 to 8 days [1, 2, 6]. According to Paterson [6], rats survived for 10–18 days after exposure to large doses of cadmium and showed early pulmonary fibrosis.

High concentrations of oxygen administered to the patient for a considerably long period may also have to be taken into consideration [5]. However, contribution of high oxygen to the present pathologic change is difficult to assess accurately.

According to the manager of the firm, an accident of the kind described in this paper had never happened before. However, the two workers had not been informed duly at that time either of the copper scrap's being plated with cadmium or of the possible production of very toxic cadmium fume. The above, combined with the poor ventilation of the working place, was considered to have brought about the fatal accident.

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Received June 26, 1983