Intensive Care Med (1988) 14:17–24

Intensive Care Medicine

Original articles

ARDS after accidental inhalation of zinc chloride smoke

E. Hjortsø[1], J. Qvist[1], M.I. Bud[2], J.L. Thomsen[3], J.B. Andersen[1], F. Wiberg-Jørgensen[4], N.K. Jensen[5], R. Jones[6], L.M. Reid[6] and W.M. Zapol[7]

[1]Department of Anesthesia and [2]Clinical Chemistry, Herlev Hospital, University of Copenhagen, Faculty of Medicine, Herlev, [3]University Institute of Forensic Pathology, University of Copenhagen, Copenhagen, Departments of [4]Anesthesia and [5]Internal Medicine, County Hospital, Hillerød, Denmark, [6]Department of Pathology, Children's Hospital Medical Center, and [7]Department of Anesthesia, Massachusetts General Hospital, Boston, Massachusetts, USA

Received: 7 November 1986; accepted: 24 February 1987

Abstract. Five soldiers were injured by inhalation of hexite smoke ($ZnCl_2$) during military training. Two soldiers, not wearing gas masks breathed hexite for 1 or 2 min, they slowly developed severe adult respiratory distress syndrome (ARDS) over the ensuing 2 weeks. This slow, progressive clinical course has not been previously described. In both patients, an increased plasma zinc concentration was measured 3 weeks after the incident. Intravenous and nebulized acetylcysteine increased the urinary excretion of zinc, and briefly decreased the plasma levels. In an attempt to arrest collagen deposition in the lungs, L-3,4 dehydroproline was administered. Both patients died of severe respiratory failure (25 and 32 days after inhalation). At autopsy diffuse microvascular obliteration, widespread occlusion of the pulmonary arteries and extensive interstitial and intra-alveolar fibrosis was observed. Three soldiers wearing ill fitting gas masks, immediately developed severe coughing and dyspnea. They improved, and 12 months after exposure their lung function tests were nearly normal, but they still had slight dyspnea on exercise.

Key words: Adult respiratory distress syndrome – Zinc chloride toxicity – Acute lung injury

The fumes from smoke ammunition bombs (hexite) consist primarily of zinc chloride ($ZnCl_2$) with an average particle size of 0.1 μm [1]. Because the particles are small, 20% are deposited beyond the respiratory bronchioles, the remainder settle in the tracheobronchial tree [2]. Inhaling this corrosive, heavy metal salt immediately produces coughing and dyspnea, and can cause an acute chemical pneumonitis [3, 4]. Several reports (Table 1) have described fatal ARDS immediately following the inhalation of $ZnCl_2$ smoke [5–7]. The largest incident occurred on Malta during World War II, when a smoke ammunition depot ignited in a tunnel injuring 70 persons; 35 required hospitalization and 10 died within 4 days of what is now recognized as ARDS.

This report describes the clinical course and pulmonary microvascular pathology of two recent and

Table 1. A summary of clinical reports of $ZnCl_2$ smoke exposure

Year	Author (reference)	No. of patients hospitalized	No. of patients died	Days to death
1945	Evans HE [5]	35 (70 exposed)	10	0–4
1954	Pare CMP et al. [11]	1	0	
1961	Johnson FA et al. [26]	3	0	
1963	Milliken JA et al. [6]	2	1	18
1966	Macaulay MB et al. [27]	1	1	11
1974	Schmahl K [3]	11	0	
1979	Schiadt VG et al. [7]	1	1	11
1981	Schenker MB et al. [4]	none (81 exposed)		
1984	Pedersen C et al. [1]	9	0	
1986	Matarese SL et al. [28]	1	0	

fatal cases of $ZnCl_2$ inhalation. In contrast to other reports, ARDS developed slowly in these patients.

Clinical course

During a 1985 training maneuver, 5 soldiers wearing gas masks passed through a $ZnCl_2$ smoke-filled pipeline (30 m long, 1.75 m internal diameter). While traversing the pipe 1 soldier (patient 1) removed his gas mask. The commanding officer (patient 2) re-entered the pipe without a gas mask to guide the soldier out. The exposure time of patient 1 is believed to be approximately 2 min and of patient 2 about 1 min.

Patient 1

A previously healthy 20-year-old male breathed $ZnCl_2$ smoke. Immediately on exposure, he exhibited severe coughing and dyspnea. At hospital admission blood pressure was normal and both pulmonary auscultation and the chest radiograph were unremarkable. Arterial blood gas (ABG) tensions breathing air showed PaO_2 117 mmHg, $PaCO_2$ 23 mmHg and pH_a 7.56. Inhalation therapy with beclomethasone (100 µg 4 times a day) was started. The following day the patient became febrile (38.4 °C) and the chest radiograph showed bilateral pulmonary infiltrates, most pronounced in the lower lobe of the right lung. Prednisone therapy (40 mg/day) was immediately started and after 7 days oral penicillin was added to treat a suspected supra-infection, but sputum cultures did not grow bacteria. Nine days after the $ZnCl_2$ inhalation the patient had a markedly reduced exercise tolerance with dyspnea upon mild exertion and severe retrosternal pain when coughing. Fifteen days after smoke inhalation the patient became cyanotic and developed subcutaneous emphysema and small bilateral pneumothoraces. The respiratory difficulties did not improve after placing bilateral thoracostomy tubes. Breathing oxygen (15 l/min) by mask increased the PaO_2 from 53 mmHg to 75 mmHg, the $PaCO_2$ remained normal. Systemic steroids were discontinued and indomethacin (300 mg/day) started. The trachea was intubated when the PaO_2 decreased further. Controlled volume ventilation with an FiO_2 of 0.8 resulted in a PaO_2 of 57 mmHg despite increasing the I/E ratio to 2/1 and the level of PEEP to 10 cm H_2O. Profound sedation and muscular paralysis did not improve gas exchange. Pressure-controlled ventilation with a Servo 900 C ventilator (Siemens-Elema) optimized the PaO_2. The thoraco-pulmonary quasi-static compliance was 30 ml/cm H_2O. Seventeen days after $ZnCl_2$ inhalation the patient was transferred because of unremitting hypoxemia.

On admission to Herlev Hospital there were no electrocardiographic or clinical signs of pulmonary hypertension. Gated blood pool scanning of ^{90m}Tc labelled albumin revealed a right ventricular ejection fraction (RVEF) of 35% (normal: 35–53%) and a left ventricular ejection fraction (LVEF) of 53% (normal: 46–68%) [8]. The mean pulmonary artery pressure ($\overline{PAP}$) was 29 mmHg (PAP-syst/diast ~40/23), the pulmonary capillary wedge pressure (PCWP) was 16 mmHg, and the cardiac output (CO) 4.8 l/min. Balloon occlusion pulmonary angiography (BOPA) showed minor vascular changes with few vasular obstructions, later diffuse vascular obstructions were seen. On day 19 the PaO_2 and lung compliance deteriorated further. The time course of arterial blood gas tensions and selected hemodynamic variables of patient 1 are shown in Fig. 1. Because plasma concentrations of free zinc had shown an upward trend since admission to the intensive care unit, therapy with intravenous acetylcysteine (140 mg/kg/day) was initiated and continued for 3 days. Acetylcysteine (100 mg q.i.d.) was also nebulized and delivered intratracheally for the remainder of the clinical course (13 days). Continuous intravenous infusion of L-3,4-dehydroproline (35 mg/kg/day) was also given for 13 days in an attempt to inhibit pulmonary fibrosis.

Because there was no initial evidence of supra-infection and all cultures were negative, all antibiotics were withdrawn. They were given later to treat either bacteria cultured from the trachea or a septic state (hypotension, leukocytosis, etc.). Owing to persistent difficulties with oxygenation, on day 23 the patient was cooled to 31–32 °C with a slight decrease of CO but without any change in PaO_2. All therapeutic maneuvers provided only temporary improvement and the patient's condition gradually deteriorated. On day 24 the PaO_2 decreased, and increasing the FiO_2 from 0.8 to 1.0 caused only a minor and transient increase of the PaO_2. High frequency ventilation (15 Hz) combined with pressure controlled ventilation increased the PaO_2 slightly, but caused unacceptable hypercapnea, which was reduced by adding a continuous flow of oxygen (5 l/min) at the carina. Twenty-eight days after the $ZnCl_2$ inhalation, kidney function declined, plasma potassium increased and a mixed respiratory and metabolic acidosis ensued. The patient died 32 days after $ZnCl_2$ inhalation.

Patient 2

The course of respiratory disease in this 20-year-old previously healthy male was identical to that of patient 1 including an unremarkable chest radiograph, until his transfer to Herlev Hospital. On day 18 (the day

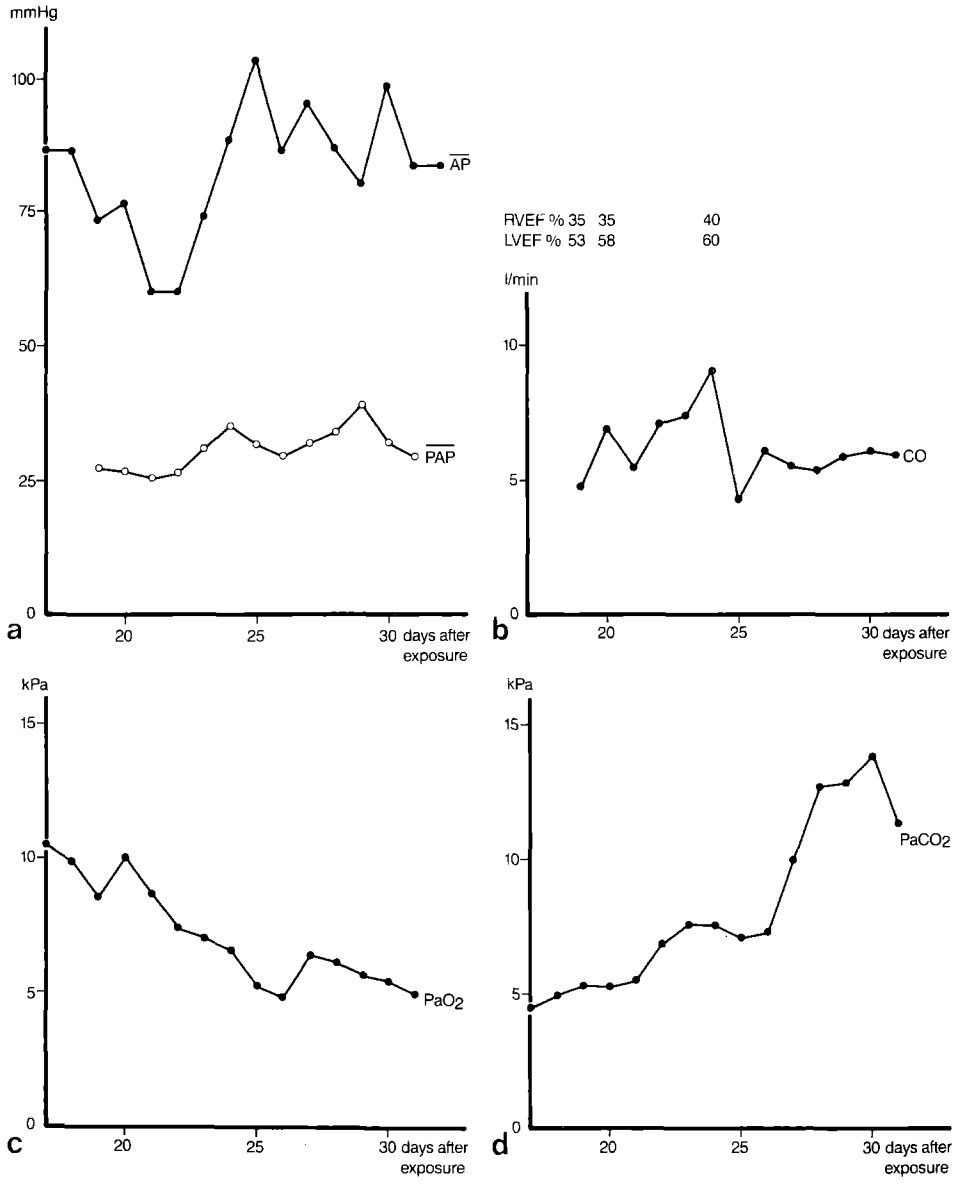

Fig. 1. Hemodynamic profile of patient 1. **a** mean systemic arterial pressure ($\overline{AP}$) and mean pulmonary artery pressure ($\overline{PAP}$), **b** cardiac output (CO) and right and left ventricular ejection fraction (RVEF + LVEF), **c** PaO_2, **d** $PaCO_2$

after transfer) the patient became hemodynamically unstable. Gated blood pool scanning demonstrated an RVEF of 22% and LVEF of 50%, while the $\overline{PAP}$ was 25 mmHg (PAP-syst/diast ~36/20), PCWP 9 mmHg. The quasi-static thoraco-pulmonary compliance was 22 ml/cm H_2O. BOPA showed diffuse microvascular occlusions. The cardiac output decreased from 8.7 l/min to 5.4 l/min and the patient became hypotensive. This episode appeared to result from a septic state with severe diffuse fluid loss, but no bacteria were cultured from the blood. A gradual reduction of renal function followed, accompanied by progressive metabolic acidosis and anuria on day 25. The patient died of progressive hypoxemia and a low cardiac output 25 days after breathing hexite.

Despite frequent blood and sputum cultures from both patients, neither bacterial pneumonia nor septicemia were found. Antibiotics were infused at times, but bacteria were never cultured from either the tracheal secretions or blood. At autopsy cultures of blood and lung grew neither bacteria nor fungi.

Patients 3–5

The 3 other soldiers exposed to $ZnCl_2$ smoke, wearing ill fitting gas masks were observed for several days in the local hospital because of coughing and dyspnea. Their lung function (VC and FEV_1) studies were repeated several times after discharge from the hospital. Twelve months after exposure these patients still complained of dyspnea during exercise, FEV_1 varied

Table 2. Tissue zinc concentration (μmol/g dry tissue)

	Patient 1	Patient 2	Control group (X ± 2 SD)
Lung, biopsy I	1.15	1.00	0.77 (0.43 – 1.11) (n = 10)
Lung, biopsy II	1.23	0.98	
Myocardium, left	2.14	1.75	1.89 (1.64 – 2.15) (n = 10)
Myocardium, right	2.09	1.49	
Liver	3.96	3.30	3.09 (1.34 – 4.84) (n = 10)
Kidney	2.82	3.35	2.68 (1.68 – 3.69) (n = 10)
Testis	1.08	1.00	1.13 (0.96 – 1.31) (n = 7)
Striated muscle	3.88	3.42	2.87 (2.56 – 3.18) (n = 10)

from 72% to 86% (normal: 82 – 118%), VC from 89% to 91% (normal: 85 – 115%).

Chemical analyses

Zinc levels were determined with an atomic absorption spectrophotometer (Perkin-Elmer, model 403) in major organs and tissues removed at autopsy, and were compared with those obtained from 10 normal males of a similar age, who died rapidly after trauma (Table 2). Pulmonary tissue zinc levels were within the normal range as measured by x-ray diffractometry and no zinc particles were observed by scanning electron microscopy.

Pathology

Patient 1

The heart was not enlarged. There was possible dilatation of the right ventricle and atrium. The right ventricle measured 3 – 4 mm in thickness. Thick fibrin deposits were observed on both pleural surfaces. Both of the lungs were solid, congested, "rubberlike" and there were scattered subpleural hemorrhages. Histologic sections from the left lung showed extensive intraalveolar and interstitial fibrosis with irregular collagen bundles running in various directions. There was hyperplasia of the type II pneumocytes. Only scattered small foci of lymphocytes and plasma cells were seen. In the alveoli there were areas with considerable hemorrhage into the lung tissue. The other major organs showed acute congestion; the kidneys showed acute tubular necrosis.

Patient 2

The lungs were similar to those described above, but the fibrosis was less pronounced. The heart was not enlarged. The right ventricle was dilated with a prominent trabecular pattern and the wall measured 4 – 5 mm. There was acute congestion of the other major organs.

Pulmonary vascular pathology

The right lung of each patient was submitted for studies of pulmonary vascular pathology.

In each lung the vessels were injected by the technique described by R. Jones et al. [9]. Both the venogram and arteriogram showed widespread evidence of reduction in the number of filled vessels, a variety of irregular filling patterns consistent with vascular injury, and a restructuring of the pulmonary vascular bed typical of acute pulmonary hypertension [10]. In the venograms of lung slices, the central veins appeared of normal size for the first two-thirds of their course, but beyond were narrowed – often abruptly. The side tributaries of some axial veins were markedly thinner than adjacent ones, indicating irregular distribution of venous injury. Pruning or loss of filling of small vessels was present (Fig. 2), especial-

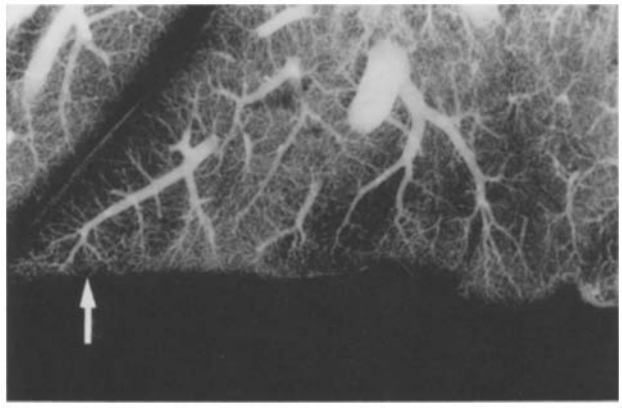

Fig. 2. Venogram (× 1.2) of a 1 cm thick lung slice after inhalation of zinc chloride (right lower lobe, patient 1). The veins are injected with barium-gelatin sulfate and appear white. Regions of reduced venous filling (arrow) are apparent, together with a region of normal venous filling on the opposite edge of the fissure

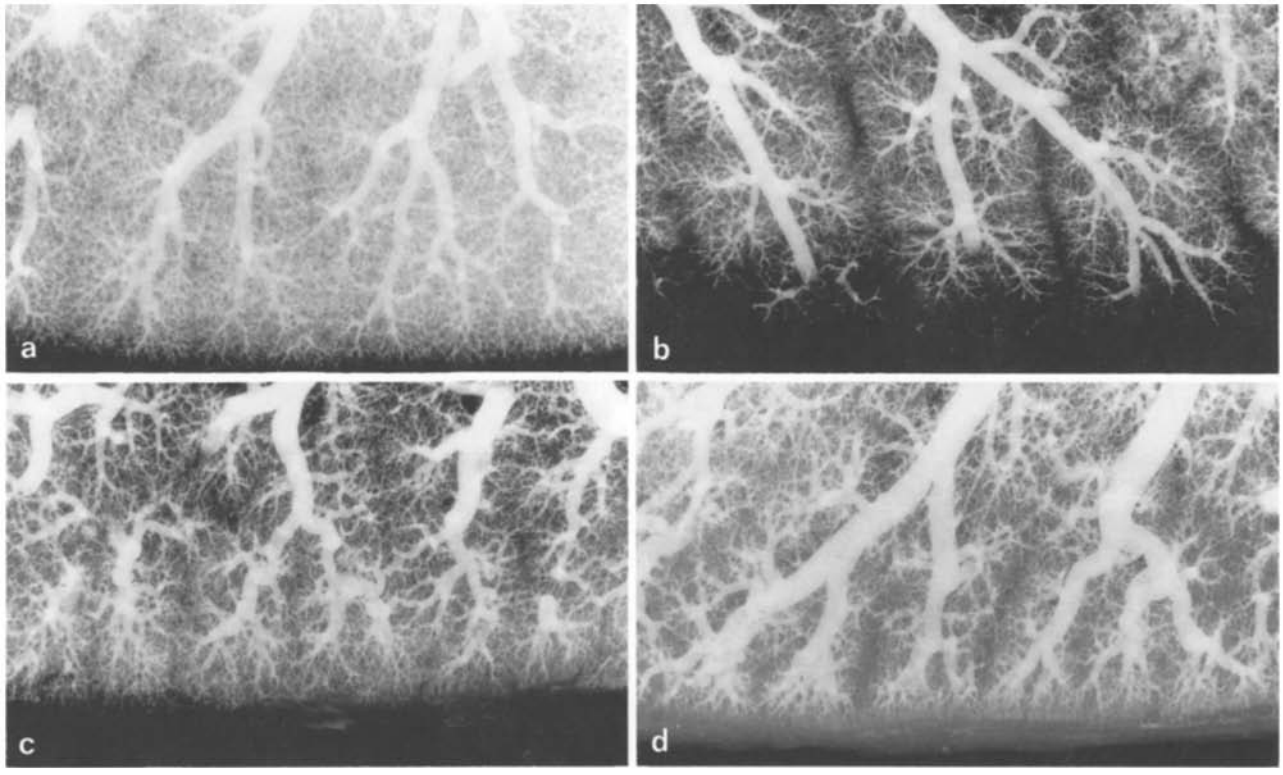

Fig. 3a–d. Arteriogram (×1.2) of a 1 cm thick lung slice. The arteries are injected with barium-gelatin sulfate and appear white: **a** Normal lung (right lower lobe, 39-year-old female) showing even arterial filling; **b** Lung region after zinc chloride inhalation (right upper lobe, patient 2) showing reduced filling of small arteries; **c** Lung region after zinc chloride inhalation (right lower lobe, patient 2) showing short dilated stubby vessels; **d** Lung region after zinc chloride inhalation showing dilated lobular and prelobular vessels in regions of tissue condensation

ly along the anterior margin of the upper lobe and along the diaphragmatic surface. Striking radiographic hypertranslucency suggested emphysematous changes. Other filling abnormalities included pulmonary vein to pulmonary vein arcades and regions of excessive filling with crossfilling to arteries indicating anastomoses.

In the arteriogram slices the main pulmonary artery was of normal size, although the axial and segmental arteries were abnormally narrow with a spindly appearance. Other filling abnormalities included loss of filling of small arteries (Fig. 3a+b), and dilated short stubby vessels in regions where vascular filling was restricted by tissue consolidation. In some regions lobular and prelobular arteries were dilated (Fig. 3c+d).

Biopsy tissue

Sections (1 μm and 700A thick) were prepared from tissue from the left lung that was embedded in epoxy resin, stained with toluidine blue (1% in 1% borax) or uranyl acetate and lead citrate, and examined by light and electron microscopy. Although patent small vessels were evident in both lungs (Fig. 4a, b) their number was obviously reduced and marked endothelial cell injury was apparent (Fig. 4c, d).

Discussion

Unlike previous case reports of the inhalation of zinc chloride fumes the 2 patient histories in this paper describe a slow onset and extended duration of acute respiratory failure (32 and 25 days, respectively) [6, 11].

Volume controlled mechanical ventilation with positive endexpiratory pressure (CMV with PEEP) did not provide satisfactory oxygenation; varying levels of PEEP (0–10 cm H_2O) and I/E ratios (from 1/5–2/1) made no difference. As pulmonary compliance was low and fibrosis was extensive, CMV with PEEP created high peak airway pressures (65–80 cm H_2O) which were delivered to the least consolidated areas, possibly further impeding blood flow and increasing the risk of barotrauma. Gas exchange improved using pressure controlled ventilation with inversed I/E ratio (2:1, 3:1). This ventilatory strategy delivered the same tidal volume but with a much lower peak airway pressure (40–60 cm H_2O). The long in-

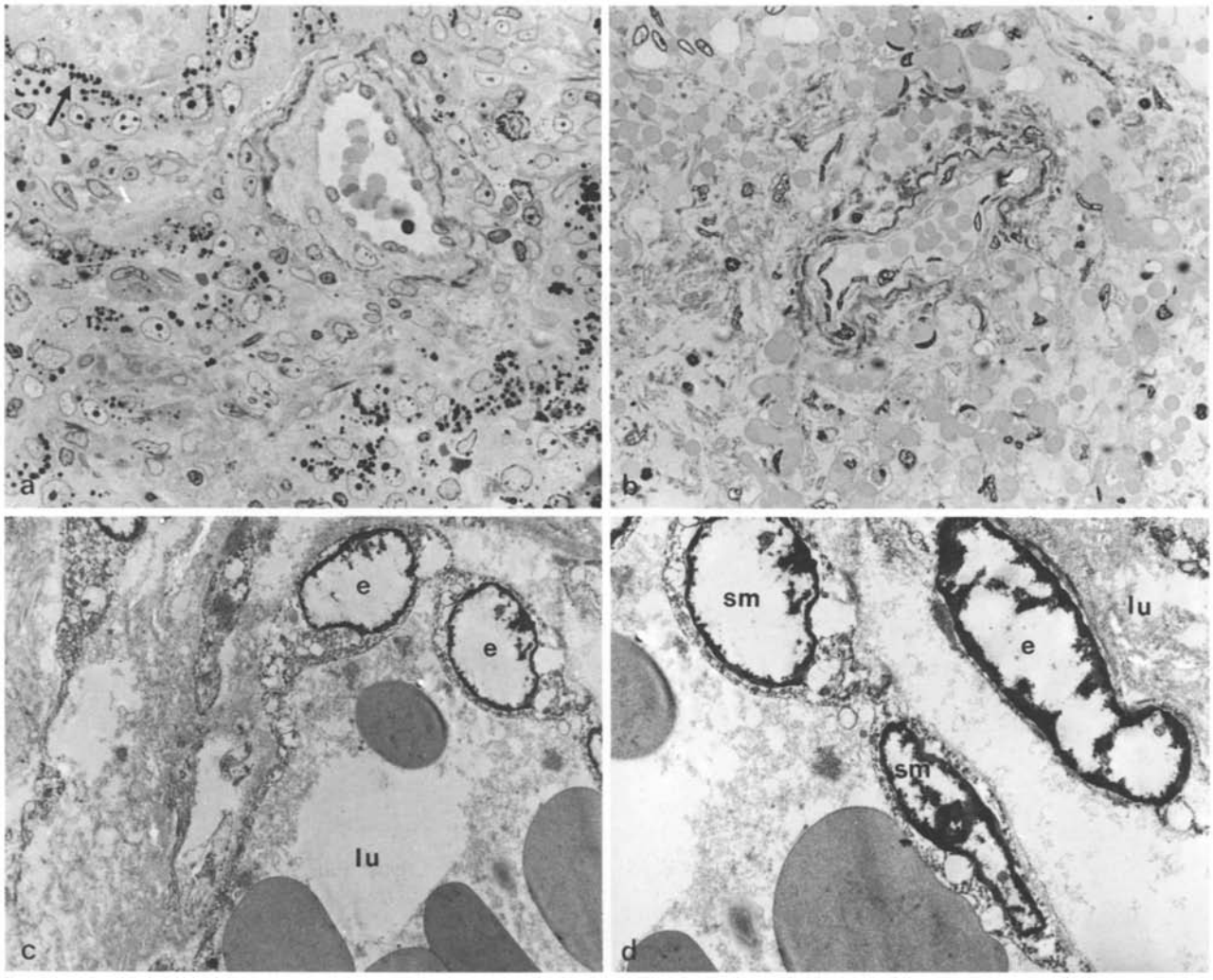

Fig. 4a–d. Photomicrographs of biopsy lung tissue showing small, but patent vessels in regions of lung consolidation: **a** Region from left lung (×394; patient 1). Alveoli are lined by cuboidal cells containing well-stained droplets (→) (these cells were noted to be type 2 epithelial cells with electron microscopy); **b** Region from left lung (×394; patient 2); **c** Region from left lung (×3600; patient 2) showing necrotic endothelial (*e*) cells and vessel wall, lumen (*lu*); **d** Region from left lung (×5200; patient 2) showing necrotic endothelial (*e*) and smooth muscle (*sm*) cells, lumen (*lu*)

spiration and the short expiration produced an auto-PEEP effect, which was substantiated by stopping the ventilator during expiration. At no flow conditions the effective PEEP was around 15 cm of H_2O even though, the set-PEEP was 4 to 7. Furthermore, it is conceivable that closed airways are squezed open with a decelerating flow and are prevented from closing during the short expiratory phase.

The early phase of lung injury from $ZnCl_2$ inhalation is marked by increased permeability to plasma proteins and fluids through the alveolocapillary membrane, causing interstitial and alveolar edema [16–18]. Later, fibroblasts proliferate and collagen increases in the interstitium and alveoli [19]. L-3,4-dehydroproline (DHP), a proline analog, has recently been infused to treat ARDS [20]. In animal studies DHP is known to inhibit the lung's biosynthesis of collagen after injury, thereby retarding the deposition of collagen and possibly inhibiting the proliferation of fibroblasts [21]. Ideally, antifibrotic drug therapy should be started as soon as possible after injury since an increased quantity of collagen fibrils has been observed as early as 3–4 days after an inhalation injury.

An integral part of the pathogenesis of severe ARDS is vasoocclusion of the pulmonary arterial bed [16, 24, 25] and such obstructions may contribute to the development of acute pulmonary hypertension. Using balloon occlusion pulmonary angiography occlusive arterial changes were observed in both of the patients with fatal lung injury. Both patients' lungs

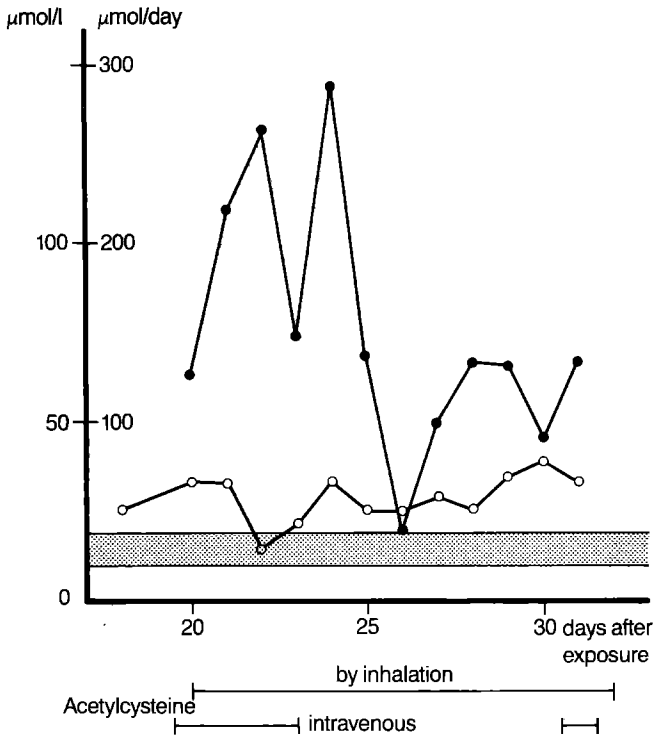

Fig. 5. Zinc concentrations in plasma and urine of patient 1. ● urine excretion; ○ plasma concentration

had pulmonary arterial occlusions and loss of small vessels when studied at autopsy. Milliken et al. [6] described right ventricular hypertrophy in a patient exposed to $ZnCl_2$ fumes. A close relationship has been reported between the degree of lung injury and the degree of pulmonary hypertension in ARDS [22, 23].

Many non-fatal $ZnCl_2$ smoke inhalations have been described [1, 4, 11, 26] including a recent military training accident in which 9 Danish soldiers were hospitalized with upper airway irritation. None developed progressive pulmonary disease and all were discharged after a few days [1]. The major difference between the two severely injured patients and the three who survived is almost certainly the quantity of inhaled $ZnCl_2$ particles.

Non-steroid antiinflammatory drugs can elevate the urinary excretion of zinc [12], but zinc excretion is reduced if therapy is extended beyond one week. Acetylcysteine is a heavy metal chelating agent [13] and also a free radical scavenger [14]. Acetylcysteine was infused intravenously [15] and nebulized directly into the lungs. The effectiveness of acetylcysteine in removing zinc can be seen in Fig. 5, which demonstrates an increased urinary zinc excretion after acetylcysteine administration. An increased zinc concentration in plasma but not in various organs has been reported in one patient dying 11 days after the inhalation of hexite smoke [7].

In our patients the plasma zinc concentrations slowly increased during the illness. The infusion of acetylcysteine increased the urine zinc excretion and briefly decreased the plasma concentration (Fig. 5). Only in patient 1 was the post mortem Zn concentration elevated in lung tissue (Table 2). Both patients showed elevated zinc concentrations in striated muscle when compared to a control group. All other organ samples had zinc concentrations within the normal range.

Coughing and dyspnea immediately after zinc chloride inhalation is followed hours later by a rise in core temperature and by diffuse consolidation of the chest radiograph. Densities on the chest radiograph often remain after the patient has recovered from symptoms [11, 26]. In 1961 Johnson et al. [26] published a description of this disease in 3 patients with mild symptoms of respiratory irritation. They noted a rapid reduction of the vital capacity to 65–82% of the predicted value which returned to normal during convalescence. Schmahl et al. [3] reported complete recovery of lung function in 2 of 3 patients studied 4 weeks after inhalation. The third patient developed a spontaneous pneumothorax on day 4 after $ZnCl_2$ exposure and the vital capacity required several months to return to a normal value.

In future accidents involving smoke bombs containing $ZnCl_2$ each patient should have frequent arterial blood gas determinations, radiographs and lung function tests. Patients who develop pulmonary infiltrates after the initial phase of upper airway symptoms should be closely monitored, and plasma and urine zinc levels measured. In patients with major exposure, therapy with intravenous and nebulized acetylcysteine should be started early. Early antifibrotic therapy with L-3,4-DHP may also be valuable. Improved therapy is necessary to prevent the relentless and fatal lung injury that can follow $ZnCl_2$ smoke inhalation.

Acknowledgements. We thank Dr. Bo Hainau, M.D., of the Department of Pathology, Herlev Hospital, for preparing the histologic material and Dr. Morten Nielsen, M.D., of the Patho-Anatomic Institute, University of Copenhagen, for measuring zinc levels in the lung tissue. Dr. Richard A. Salvador of Hoffmann, La Roche, Nutley, New Jersey, USA, supplied us with L-3,4 dehydroproline for intravenous use. This paper was supported by NHLBI SCOR grant HL23591.

References

1. Pedersen C, Hansen CP, Grønfeldt W (1984) Zinc chloride smoke poisoning following employment of smoke ammunition. J Dan Med Assoc (Copenhagen) 146:2397
2. Brain D, Valberg PA (1980) Deposition of aerosol in respiratory tract. In: Murray JF (ed) Lung disease – state of the art. American Lung Association, New York, pp 225–273

3. Schmahl K (1974) Klinik der Zinknebelvergiftung. Pneumonologie 105:161
4. Schenker MB, Speizer FE, Taylor JO (1981) Acute upper respiratory symptoms resulting from exposure to zinc chloride aerosol. Environ Res 25:317
5. Evans HE (1945) Casualties following exposure to zinc chloride smoke. Lancet 2:368
6. Milliken JA, Waugh D, Kadish ME (1963) Acute interstitial pulmonary fibrosis caused by a smoke bomb. Can Med Assoc J 88:36
7. Schaidt VG, Mallinckrodt MG, Opitz O (1979) Über die Zinkverteilung in Körperflüssigkeiten und Organen nach tödlicher Zinknebelvergiftung. Beitr Gerichtl Med 37:351
8. Brynjolf I, Kelbæk H, Munck O, Godtfredsen J, Larsen S, Eriksen J and other members of the ALCAMY-Group (1984) Right and left ventricular ejection fraction and left ventricular volume changes at rest and during exercise in normal subjects. Eur Heart J 5:756
9. Jones R, Zapol WM, Reid L (1984) Pulmonary artery remodeling and pulmonary hypertension after exposure to hyperoxia for 7 days. A morphometric and hemodynamic study. Am J Pathol 117:273
10. Jones R, Zapol WM, Tomashefski JF, Kirton OC, Kobayashi K, Reid L (1985) Pulmonary vascular pathology: human and experimental studies. In: Zapol WM, Falke KJ (eds) Acute respiratory failure. Marcel Dekker, New York, pp 23–160
11. Pare CMB, Sandler M (1954) Smoke bomb pneumonitis: description of a case. J R Army Med Corps 100:320
12. Elling H, Kiilerich S, Sabro J, Elling P (1980) Influence of a non-steroid antirheumatic drug on serum and urinary zinc in healthy volunteers. Scand J Rheumatol 9:161
13. Lund ME, Banner W Jr, Clarkson TW, Berlin M (1984) Treatment of acute methylmercury ingestion by hemodialysis with N-acetylcysteine (Mucomyst) infusion and 2,3-dimercaptopropane sulfonate. J Toxicol Clin Toxicol 22:31
14. Bernard GR, Lucht WD, Niedermeyer ME, Snapper JR, Ogletree ML, Brigham KL (1984) Effect of N-acetylcysteine on the pulmonary response to endotoxin in the awake sheep and upon in vitro granulocyte function. J Clin Invest 73:1772
15. AMA drug evaluations (1985) American Medical Association, Fourth Edition, Chicago, Illinois, p 142
16. Pontoppidan H, Rie MA (1982) Pathogenesis and therapy of acute lung injury. In: Prakash O (ed) Applied physiology in clinical respiratory care. Marinus Nijhoff Publishers, Amsterdam
17. Zapol WM, Trelstad RL, Snider TM, Pontoppidan H, Lemaire F (1983) Pathophysiologic pathways of the adult respiratory distress syndrome. In: Tinker DJ, Rapin M (eds) Care of the critically ill patient. Springer, New York, pp 341–358
18. Trelstad RL, Zapol WM, Martin EG (1985) Interstitial alterations following acute injury. In: Zapol WM and Falke KJ (eds) Acute respiratory failure. Marcel Dekker, New York, pp 185–207
19. Zapol WM, Trelstad RL, Coffey JW, Tsai I, Salvador RA (1979) Pulmonary fibrosis in severe acute respiratory failure. Am Rev Respir Dis 119:547
20. Zapol WM, Quinn D, Coffey J, Salvador RA (1984) L-3,4-dehydroproline suppression of fibrosis in ARDS: early clinical results. Am Rev Respir Dis 129:102
21. Salvador RA, Fiedler-Nagy C, Coffey JW (1985) Biochemical basis for drug therapy to prevent pulmonary fibrosis in the adult respiratory distress syndrome. In: Zapol WM, Falke KJ (eds) Acute respiratory failure. Marcel Dekker, New York, pp 477–506
22. Zapol WM, Snider MT (1977) Pulmonary hypertension in severe acute respiratory failure. N Engl J Med 296:476
23. Zapol WM, Snider MT, Rie MA, Quinn DA, Frikker M (1985) Pulmonary circulation during adult respiratory distress syndrome. In: Zapol WM, Falke KJ (eds) Acute respiratory failure. Marcel Dekker, New York, pp 241–273
24. Tomashefski JE, Davies P, Bogtis C, Greene R, Zapol WM, Reid LM (1983) The pulmonary vascular lesions of the adult respiratory distress syndrome. Am J Pathol 112:112
25. Greene R, Boggis CRM, Jantsch HS (1985) Radiography and angiography of the pulmonary circulation in ARDS. In: Zapol WM and Falke KJ (eds) Acute respiratory failure. Marcel Dekker, New York, pp 275–302
26. Johnson FA, Stonehill RB (1961) Chemical pneumonitis from inhalation of zinc chloride. Dis Chest 40:619
27. Macaulay MB, Mont AK (1966) Smoke bomb poisoning: a fatal case following the inhalation of zinc chloride smoke. J R Army Med Corps 113:27
28. Matarese SL, Matthews JI (1986) Zinc chloride (smoke bomb) inhalational lung injury. Chest 89:308

Dr. Else Hjortsø
Department of Anaesthesia
Herlev Hospital
DK-2730 Herlev
Denmark