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CASE REPORT

Acute lung reaction due to zinc inhalation

J-L. Malo*, J. Malo*, A. Cartier*, J. Dolovich**

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ABSTRACT: A 27-yr-old man began work at a company that produces concrete pipes in April 1987. The pipes are linked with rings covered with zinc. In September 1987, he was transferred to a new job in the same plant where he had to heat zinc wires and shoot the heated zinc in powder form onto the iron rings. He had no past personal nor family atopic history. Two weeks after he began in his new job, he experienced an episode of chills with muscle aches and dyspnoea at the end of a working day. The fever persisted until the next day, at which time he saw a physician. A chest radiograph revealed diffuse interstitial shadows. He was off work for 10 days. His symptomatology disappeared and his chest radiograph cleared. He went back to work and experienced a similar episode. He remained away from work for one month, after which specific inhalation challenges were performed. On a control day, there were no significant changes in forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), or buccal temperature. Two white blood counts (WBC) showed 8,400 and 8,500 white cells. He was exposed to his usual work environment for one hour on two consecutive days. On both occasions, there were significant falls in FEV_1 (16% and 20%) and FVC (10% and 11%), occurring 4–6 h after exposure. Buccal temperature reached 38.1 and 38.7°C on the two occasions, and WBC were 17,000 and 15,900 at the end of each day. Precipitins were negative and specific IgG antibodies could not be detected. In conclusion, this report shows that zinc can cause an acute lung reaction, which seems different from "metal fume fever" for which functional or radiological changes have not been reported.

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"Metal fume fever" is a self-limiting condition characterized by fever and general symptoms of malaise that occur after welding metal [1–3]. The mechanism of the reaction is unknown. In this report, we describe a subject who presented symptoms typical of this condition who furthermore showed evidence of functional and radiological lung involvement.

Case report

A 27-yr-old man began work at a company that produces concrete pipes in April 1987. The pipes are linked with rings covered with zinc. In September 1987, he was transferred to a new job in the same plant where he had to heat zinc wires and shoot the heated zinc in powder form onto iron rings. He had no past personal nor family atopic history. Two weeks after beginning his new job, he experienced an episode of chills with muscle aches and dyspnoea at the end of a working day. The fever persisted until the next day at which time he saw a physician. A chest radiograph revealed diffuse interstitial shadows (fig. 1). He was off work

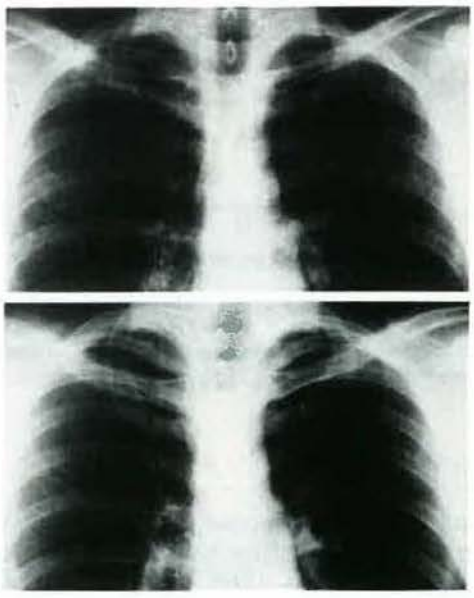

Fig. 1. – Diffuse nodular infiltrates after a period at work (upper panel) with clearing after 10 days off-work (lower panel).

for 10 days. His symptomatology disappeared and his chest radiograph cleared (fig. 1). When he returned to work, he experienced a similar episode on the first day of re-exposure. He remained away from work for one month after which specific inhalation challenges were carried out.

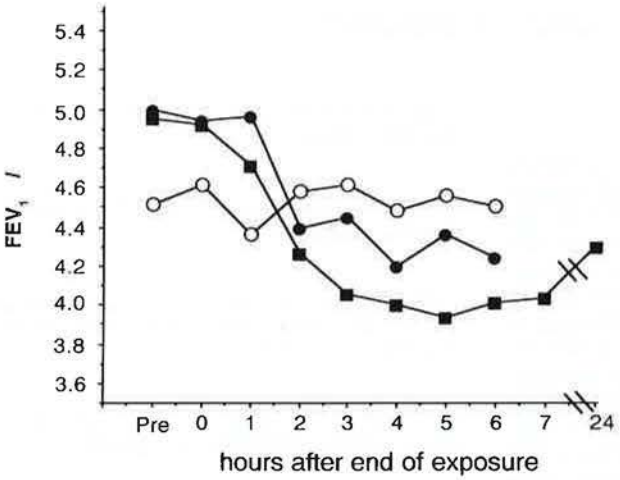

Fig. 2. – Changes in FEV_1 on the control day (no exposure) and two days of exposure at work. Pre. values obtained before exposure; o: control day, no exposure, ●: first day of exposure for one hour at work, ■ : second day of exposure for one hour at work, FEV_1: forced expiratory volume in 1 sec - litres.

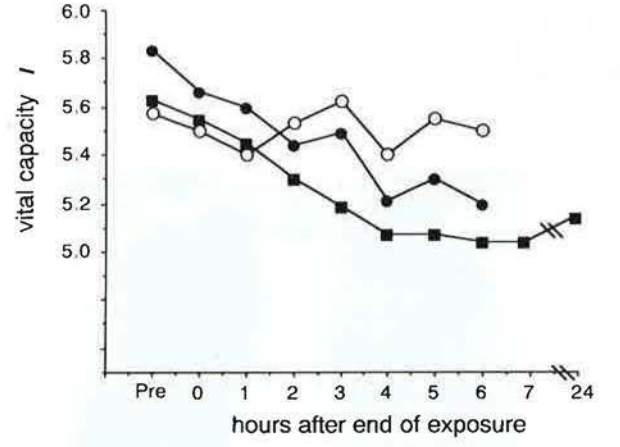

Fig. 3. – Changes in vital capacity on the control day (no exposure) and two days of exposure at work. Pre. values obtained before exposure; o: control day: no exposure, ●: first day of exposure for one hour at work, ■: second day of exposure for one hour at work.

Specific inhalation tests

These tests were performed in the following way: on the control day, spirometry including forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) was performed in accordance with proposed standards [4] in the hospital laboratory. Baseline FEV_1, FVC and the FEV_1/FVC ratio were normal (105% predicted, 113% predicted and 98% predicted, respectively) [5]. Spirometry and buccal temperature was assessed hourly for 7 h; there were no significant changes throughout the day (figs 2–4). White blood counts showed 8,400 and 8,500 polymorphonuclears (60% and 58% neutrophils respectively) at the beginning and at the end of the control day. The concentration of methacholine causing a 20% change in FEV_1 was assessed using a standardized procedure; no significant bronchial hyperresponsiveness (PC_{20} >128 $mg \cdot ml^{-1}$) was demonstrated [6]. On the next two consecutive days, he was exposed to his usual work environment for one hour. On both occasions, there were significant falls in FEV_1 (maximum of 16% and 20%) and FVC (maximum of 10% and 11%), occurring 4–6 h after exposure (figs. 2–3). Buccal temperatue reached 38.1°C and 38.7°C on the two occasions (fig. 4), and white blood counts were 17,000 and 15,900 white cells respectively (84% neutrophils on each occasion). The subject experienced malaise and general muscle ache on both occasions. His PC_{20}, assessed at the end of the second day of active challenges, was still within the normal range (PC_{20}=74 $mg \cdot ml^{-1}$).

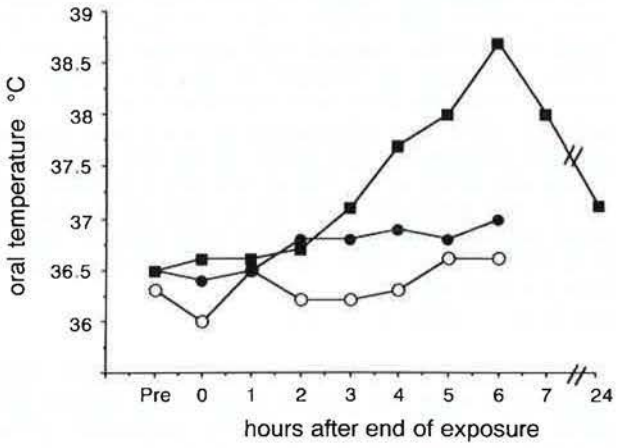

Fig. 4. – Changes in oral temperature on the control day (no exposure) and two days of exposure at work. Pre. values obtained before exposure; o: control day: no exposure, ●: first day of exposure for one hour at work, ■: second day of exposure for one hour at work.

Immunological tests

Standard double diffusion in agar for precipitins was performed using the subject's serum and zinc sulphate at 6 different concentrations (10^{1}–10^{-4} $mg \cdot ml^{-1}$). These tests were negative.

A micro-Elisa technique with zinc sulphate in phosphate buffered saline (PBS) and zinc sulphate in human serum albumin (HSA) at 3 different concentrations (10^{0}–10^{-2} $mg \cdot ml^{-1}$) was used to test for specific IgG antibodies. As shown in table 1, the results were not significantly different from those for two control, non-exposed individuals.

Table 1. – MicroELISA for specific IgG antibodies to zinc

	subject	control subject no. 1	control subject no. 2
Zinc sulphate in PBS			
1 mg·ml^{-1}	0.371	0.485	0.438
0.1 mg·ml^{-1}	0.371	0.491	0.468
0.01 mg·ml^{-1}	0.414	0.471	0.472
Zinc sulphate in HSA			
1 mg·ml^{-1}	0.302	0.293	0.397
0.1 mg·ml^{-1}	0.311	0.351	0.406
0.01 mg·ml^{-1}	0.379	0.334	0.336

PBS: phosphate buffered saline; HSA: human serum albumin.

Discussion

This report shows that acute exposure to zinc can cause a reaction characterized by fever, increase in white blood cells, falls in FEV_1 and FVC and radiological interstitial infiltrates. However, neither precipitins nor specific IgG antibodies were detected. To the best of our knowledge, only one other similar case has been documented in the literature. VOGELMEIER *et al.* [7] described a welder who experienced the same clinical and functional abnormalities after welding zinc-coated materials for the first time. No immunological studies were carried out. However, the bronchoalveolar lavage showed increased neutrophils but no lymphocytosis. As the presence of both increased neutrophils and lymphocytes are expected in hypersensitivity pneumonitis, the authors proposed that this condition was not compatible with hypersensitivity pneumonitis even though the symptomatology was similar. It is known, however, that acute hypersensitivity pneumonitis can result in a predominant neutrophilia in bronchoalveolar lavage [8]. We did not carry out bronchoalveolar lavage in our subject to characterize the cellular influx. However, the clinical, physiological and radiological features are those commonly encountered in hypersensitivity pneumonitis or an acute toxic reaction.

Hard metals have recently been incriminated as a cause of interstitial lung diseases and occupational asthma. Cobalt has been shown to cause asthma in some subjects through an IgE-dependent mechanism [9], and the appearance of interstitial fibrosis with giant cells in others [10]. We have recently shown that soldering galvanized metal may cause occupational asthma, possibly due to the release of zinc into the environment [11]. One of the two individuals we described also had fever and leukocytosis.

Our case report, combined with that of VOLGELMEIER *et al.* [5], shows that metal fume fever may cause pulmonary involvement. This is contrary to that which is generally described in textbooks on occupational lung diseases [1–3]. No functional or radiological abnormalities have been described, probably because they were not systematically sought after. Elevated levels of zinc in the serum [12] and zinc and copper in the urine [13] have been documented. FARRELL recently presented an interesting case of angioedema and urticaria after zinc fume exposure [14]. That subject also reported metal fume fever. Prospective assessment of individuals who solder with hard metals should be performed in order to establish the frequency of the condition. Further studies using bronchoalveolar lavage and/or lung biopsies are also needed to investigate the mechanism of the reaction.

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Une réaction aigue pulmonaire chez l'inhalation de zinc. J-L. Malo, J. Malo, A. Cartier, J. Dolovich.

RÉSUMÉ: Un ouvrier de 27 ans commença à travailler en avril 1987 pour une compagnie fabriquant des tuyaux en ciment. Ces tuyaux sont reliés avec des anneaux faits de zinc. En septembre 1987, il fut muté à un nouveau poste de travail où il avait à chauffer des fils de zinc et à propulser le zinc chaud et sous forme de poudre sur les anneaux métalliques. Il n'avait aucune historie antérieure, familiale ou personnelle, d'atopie. Deux semaines après avoir entrepris ce type de travail, il souffrit d'un épisode de fièvre avec douleurs musculaires et dyspnée à la fin d'un jour au travail. La fièvre persista le lendemain, ce qui l'emmena à consulter. A ce moment, une radiographie pulmonaire montra des infiltrats diffus de type interstitiel. Il s'absenta de son travail durant 10 jours. Ses symptômes disparurent et la radiographie pulmonaire devient normale. Il retourna au travail et souffrit d'un épisode semblable. Il fut mis en congé de travail durant un mois et des tests de provocation bronchique spécifique furent effectués. Lors d'une journée témoin de non exposition, il n'y eut aucun changement significatif du VEMS, de la capacité vitale forcée (CVF) et de la température buccale. Deux numérations globulaires montrérent 8400 et 8500 polymorphonucléaires. Il travailla durant une heure lors des deux journées suivantes consécutives. Lors de ces deux journées, le VEMS chuta de 16% et 20% et la CVF de 10% et 11%. Ces changements furent maximaux 4 à 6 heures après la fin de 38.7°C l'exposition. La température buccale atteignit 38.1°C et 38.7°C et la formule sanguine montra 17, 000 et 15, 900 polymorphonucléaires respectivement à la fin de chaque journée. On n'a pu déceler de précipitines dans le sang circulant ni d'augmentation des IgG spécifiques contre le sulfate de zinc. Nous concluons que le zinc peut causer une réaction aiguë pulmonaire. Cette réaction semble différente de celles décrite sous le vocable "metal fume fever" puisque des changements fonctionnels ou radiologiques n'ont pas été décrits dans cette condition.

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