CASE STUDY

Occupational asthma caused by aluminium welding

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ABSTRACT: Work-related asthma has been documented in workers employed in the primary aluminium industry and in the production of aluminium salts. The role of aluminium in the development of occupational asthma has, however, never been convincingly substantiated.

We investigated a subject who experienced asthmatic reactions related to manual metal arc welding on aluminium.

Challenge exposure to aluminium welding with flux-coated electrodes, as well as with electrodes without flux, elicited marked asthmatic reactions. Manual metal arc welding on mild steel did not cause significant bronchial response. The results of inhalation challenges combined with exposure assessments provided evidence that aluminium can cause asthmatic reactions in the absence of fluorides.

Awareness of this possibility may be relevant to the investigation of asthma in workers exposed to aluminium.

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Occupational asthma has been documented in workers exposed to various metals, including platinum, chromium, cobalt, nickel and zinc [1, 2]. Work-related asthma has been described in aluminium smelters [3, 4] and in workers exposed to aluminium salts [5, 6], although the exact aetiological agent has not yet been identified.

We investigated a subject experiencing asthma on exposure to aluminium welding, whose inhalation challenges and environmental assessments provided evidence that aluminium was the most likely causal agent.

Case report

A 32 yr old man had been employed as a maintenance worker in a leather plant. He had to perform electric arc welding on mild steel, using manual metal arc and inert gas metal arc techniques. Approximately once a month, he welded aluminium pieces using a manual metal arc process with a flux-coated electrode. After 4 yrs of intermittent exposure to these various welding processes, he developed chest tightness and wheezing that occurred specifically on days he welded on aluminium. Asthmatic symptoms started 1–4 h after the end of exposure to aluminium welding and persisted for several hours. The subject never experienced myalgia, chills or fever. He was treated with inhaled budesonide (400 µg daily) and salbutamol when necessary.

The subject was referred for investigation 18 months after the onset of work-related asthmatic symptoms. At that time, he was experiencing asthma on exercise. He had smoked five cigarettes·day⁻¹ for 12 yrs and reported a history of allergic rhinitis since childhood. Skin-prick tests with a battery of common inhalant allergens elicited a positive reaction to mixtures of tree, grass and weed pollens. Skin-testing with Al(SO₄)₃, Al(NO₃)₃, Cr₂(SO₄)₃ and NiSO₄ yielded negative results using concentrations of 0.01, 0.1, 1 and 10 mg·mL⁻¹ in saline.

Inhalation challenges

Inhalation challenges were performed in the hospital workshop, according to recent guidelines [7]. Baseline spirometric measurements showed a forced expiratory volume in one second (FEV₁) of 4.38 L (100% predicted value) and a FEV₁/forced vital capacity (FVC) ratio of 73% (89% pred). On a control day without exposure to occupational agents, spontaneous fluctuations of FEV₁ were <10% (fig. 1a). At the end of the control day, the provocative concentration of histamine causing a 20% fall in FEV₁ (PC₂₀) was 0.07 mg·mL⁻¹ [8]. The next day, manual metal arc welding on aluminium for 1 h with the flux-coated electrodes used at work resulted in a transient fall in FEV₁, with a maximum of 17% that was recorded 7 h after the end of exposure. On the following day, exposure to aluminium welding for 2 h provoked an asthmatic reaction with a progressive fall in FEV₁, reaching 52% at 2 h postexposure. The subject was given inhaled salbutamol (200 µg) at that time and again 4 h later. On the next morning, FEV₁ was still 20% lower than pre-exposure value, so that histamine PC₂₀ was not reassessed.

The subject gave informed consent for additional inhalation challenges. One month after the first set of tests, he was exposed to manual metal arc welding on aluminium using electrodes from which the crumbly flux had been removed (fig. 1b). Exposure for 2 h resulted in a 55% fall in FEV₁ at 90 min postexposure. FEV₁ improved significantly after administration of inhaled salbutamol, but declined again 5 and 8 h after the end of challenge exposure. On the next morning, the level of nonspecific bronchial hyperresponsiveness was significantly increased (histamine PC₂₀ <0.01 mg·mL⁻¹) as compared with baseline value [9]. One month later, the subject welded mild steel using a manual metal arc process with rutile electrodes for 2 h without developing significant bronchial response. The day before this challenge, the subject's

Fig. 1. — a) Changes in forced expiratory volume in one second (FEV₁) recorded after welding aluminium with flux-coated electrodes for 1 h (—●—) and 2 h (—▲—); (---○---): control. b) Changes in FEV₁ after exposure to aluminium welding without flux for 2 h (—■—) and to mild steel welding with rutile electrodes for 2 h (---□---), Pre: pre-exposure; BDT: bronchodilator therapy.

histamine PC₂₀ value (0.05 mg·mL⁻¹) was similar to that recorded at the end of the control day. Control challenges were carried out in two previously unexposed asthmatic subjects (histamine PC₂₀ values of 0.02 and 0.06 mg·mL⁻¹) who demonstrated no significant changes in FEV₁ after exposure to aluminium welding for 2 h.

Exposure assessment

The concentration of chemicals contained in the electrodes was assessed using flame atomic absorption spectrometry for metals and specific electrodes for fluorides (Orion Model 96-09 combination fluoride electrode; Orion Research, Boston, MA, USA) and chlorides (Orion Model 96-17B combination chloride electrode; Orion Research). The core of the electrodes was made almost exclusively from aluminium (99.9%). The flux contained aluminium (8%), chlorides (34%), fluorides (21%), potassium (11%) and sodium (9%), as well as small amounts of chromium (1%), nickel (4%) and lithium (3%). X-ray diffraction analysis showed that cryolites (Na₃AlF₆, K₃AlF₆ and/or NaK₂AlF₆) were the principal constituents of the flux.

Fumes generated during aluminium welding with and without flux and during welding on mild steel were assessed using personal samplers (Casella AFC 123 personal

Table 1. — Environmental measurements

Agents*	Aluminium welding		
	Flux-coated electrodes	Electrodes without flux	Mild steel welding with flux-coated electrodes
Inhalable dust mg·m ⁻³	12.6	2.9	25.2
Respirable dust mg·m ⁻³	6.2	1.4	26.8
Aluminium µg·m ⁻³	813	165	ND
Chromium µg·m ⁻³	4.3	4.4	6.4
Nickel µg·m ⁻³	0.2	0.3	0.8
Fluoride µg·m ⁻³	0.2	ND	ND
Ozone, ppb (range)	62(31–76)	99(61–139)	130(46–280)

ND: not detected; *: mean concentration for 30 min air sampling; ppb: parts per billion.

air sampler; Casella London, London, UK) with an airflow of 1.8 L·min⁻¹. Particles were collected on 0.8 µm cellulose acetate filters and analysed using flame atomic absorption spectrometry and specific electrodes (see above). Concentrations of ozone were measured with a TLD1 tape monitor (MDA Scientific, Glenview, IL, USA). The results revealed that aluminium welding both using flux-coated electrodes and electrodes without flux resulted in concentrations of aluminium, chromium and nickel below their respective threshold limit values (table 1). After removal of the flux, aluminium welding no longer released fluoride particles. Welding on mild steel with rutile electrodes generated similar amounts of chromium and nickel, but no aluminium. Significant concentrations of ozone were recorded both during mild steel and aluminium welding.

Discussion

The results of inhalation challenges in the subject studied indicate that asthmatic reactions caused by aluminium welding did not result from a nonspecific irritant effect. Welding on mild steel did not induce a bronchial response, although this process generated higher levels of respirable dust. An increase in nonspecific bronchial hyperresponsiveness was documented after challenge exposure to aluminium welding without flux. Such changes in hyperresponsiveness have been described after asthmatic reactions induced by agents acting through a sensitizing mechanism [10]. In addition, two control asthmatic subjects did not show bronchial response to aluminium welding.

Occupational asthma has been documented in a few workers exposed to manual metal arc welding on stainless steel [11] and to inert gas metal arc welding on mild steel [12]. The causal agents remain largely unknown, since electric arc welding involves inhalation exposure to a variety of agents, depending on the materials and processes that are used. Welding fumes contain complex metal oxide particulates originating from the evaporation of melted metals and consumable electrodes [13]. Moreover, ultraviolet radiation in the electric arc leads to the production of irritant gases, including oxides of nitrogen and ozone. In the subject studied, challenge exposure to aluminium welding, using either flux-coated electrodes or electrodes without flux, provoked asthmatic reactions. The two procedures generated aluminium, chromium and nickel, but fluorides were not detected when the flux had been removed from electrodes. These findings strongly suggest that the

flux constituents, including fluorides, were not responsible for the induction of asthmatic reactions.

It has been postulated, although not proved, that chromium and/or nickel were the offending agents in subjects with occupational asthma due to manual metal arc welding on stainless steel [11]. It is unlikely that these metals were involved in the development of asthmatic reactions in the present subject. Thus, similar levels of airborne chromium and nickel were detected during aluminium and mild steel welding, although the latter process did not provoke a bronchial response. The role of ozone could also be reasonably excluded, since welding on mild steel generated high concentrations of ozone without eliciting asthmatic reaction.

To the best of our knowledge, aluminium has never been documented as causing occupational asthma in welders. Specific inhalation challenges have shown that amino-ethyl ethanolamine contained in fluxes is the agent causing occupational asthma in aluminium solderers [14]. Asthma, known as "potroom asthma", has been recognized to develop in a substantial proportion of workers in the primary aluminium industry [3, 4]. The work-relatedness of asthma has been documented in a few potroom workers using serial assessments of FEV₁, peak expiratory flow rates and nonspecific bronchial responsiveness [15, 16]. Epidemiological surveys of aluminium smelters have found a dose-response relationship between the level of exposure to fluorides and work-related asthmatic symptoms [17], although specific inhalation challenges have not yet been carried out to identify precisely the agent inducing potroom asthma. Clinical asthma or bronchial hyperresponsiveness has been reported in workers exposed to potassium aluminium tetrafluoride used as a flux for soldering aluminium [6]. The authors mentioned that challenge with the flux caused a late reaction and an increase in nonspecific bronchial hyperresponsiveness. A recent report documented asthmatic reactions after inhalation challenge with aluminium powder and AlCl₃ solution in a safe manufacturing worker [18]. Work-related asthma has also been described in factories producing aluminium salts [5]. Inhalation challenges have been performed in three workers with aluminium fluoride dust (AlF₃) and aluminium sulphate dust (Al₂(SO₄)₃), without inducing asthmatic reactions. In this report, inhalation challenge studies combined with environmental assessments provided evidence that aluminium can cause asthmatic reactions even in the absence of detectable fluorides.

It is still unknown what mechanisms are responsible for the induction of asthma in the present subject. Studies have shown that an immunoglobulin E (IgE)-mediated mechanism could be involved in the pathogenesis of metal-induced asthma, although skin reactivity to metal salts has not been consistently demonstrated in subjects with occupational asthma caused by these agents [1, 2]. The subject studied did not show immediate skin reactivity to metal salts. However, the relevance of these findings remains uncertain, since the specific aluminium salts generated during the welding process were not identified.

We conclude that aluminium should be considered a possible cause of occupational asthma. This finding may be relevant to the investigation of health hazards in workers exposed to aluminium and derived compounds.

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