Broncho-pulmonary ailments caused by dust or fumes from aluminium or compounds thereof

Definition of causal agent

Aluminium is a silvery – white ductile and malleable metal. In nature it is not found as a free metal because of its reactivity. It occurs in the environment as aluminium oxide (alumina), hydroxide, fluoride, chloride, bromide, sulphate, nitrate, and silicate.

Main occupational uses and sources of exposure:
Occupational exposure may occur during: extraction of bauxite, primary aluminium production, metallurgical industry (production and processing of metal alloy), welding, chemical industry (for manufacturing various alumina based chemicals and as a catalyser), preparation and use of synthetic abrasives and production of explosives and fireworks. Aluminium compounds are also used in production of glass, ceramics, rubber, wood preservatives, pharmaceuticals and waterproofing textiles.

Adverse effects

☐ Restrictive pulmonary disease

Exposure to sub-micron size aluminium powder (non-fibrous and fibrous particles) may give rise to fibrosis of the lung that is called Aluminosis. It is a slight fibrosis characterized by slow and benign evolution. The intensity of fibrosis is correlated to duration of exposure and pulmonary levels of aluminium. Non occupational exposed subjects have a pulmonary content to 50 mg/kg wet weight. Aluminium content in the lung of about 1000 mg/kg dry weight is the limit for the initial development of fibrosis.

Shaver's disease is an historic example of rapid and progressive interstitial fibrosis of the lung. It was induced by the inhalation of aluminium fumes together with silicon dioxide and was attributed to the use of bauxite contaminated with percentages of silicon dioxide greater than 30%. Respiratory effects were severe and complications such as pneumothorax, pulmonary emphysema and death were common. Present day control measures may have reduced the risk.

The fibrogenic potency of aluminium dust is undetermined; moreover inhaled powdered alumina was used as a means of preventing silicosis.

Diagnostic criteria:

Symptoms: Shortness of breath and dry cough, but in early stages there may be no symptoms.

Clinical signs: Signs of fibrotic lung disease e.g. crepitations on auscultation but in early stages, there are no clinical signs

Lung function: restrictive or mixed impairment of low degree. Generally lung function decreases as a profusion of small opacities increases.

Chest X-ray: findings range from interstitial infiltrates to slight profusion of small round or irregular opacities.

Exposure criteria:

Minimum intensity of exposure: occupational exposure confirmed and, if possible assessed by:

- History and working conditions showing evidence of exposure to high concentration of aluminium, and if available:
- Workplace air monitoring results. Present information suggests that a level for alumina in air of 10 mg/m^3 taking place over a period of 37 years gives rise to a pulmonary aluminium content in the order of 900 mg/kg.

Minimum duration of exposure: 10 years, but this varies with the intensity of exposure.

Maximum latent period: a maximum cannot be determined as the lesions are a function of the cumulative dose.

☐ Potroom asthma

The term potroom arises from the use of metal pots for electrolysis processing of alumina. Potroom fumes can cause asthma-like symptoms with continuing lung function impairment even after cessation of occupational exposure. The specific causal agent remains unidentified. The pathogenesis is considered to be bronchial hyper-reactivity induced probably by strong respiratory irritants in the potroom environment (hydrogen fluoride, sulphur dioxide, fluorides in particulates). Potroom fumes also contain vanadium that is known to cause asthma. Attacks similar to potroom asthma have also been reported from other industries with exposure to aluminium fluoride compounds (K_3AlF_6, AlF_3) and cryolite (Na_3AlF_6).

Diagnostic criteria:

Symptoms: episodes of chest tightness, breathlessness, non-productive cough and wheeze.

They may occur during working hours but more typically some hours after leaving work (delayed onset).

The symptoms are work-related; they become more frequent with repeated exposure and improve when away from work. An improvement in symptoms may be expected after cessation of exposure. Increased bronchial reactivity once induced, has a tendency to persist. Atopy does not seem to be significant for the onset and the prognosis.

Clinical signs: those of bronchial obstruction e.g. rhonchi on auscultation.

In the differential diagnosis, other causes of asthma should be considered.

Lung function: bronchial obstruction reversible by bronchodilators; bronchial obstruction triggered by non-specific bronchoconstrictors (metacholine test), normal gas transfer, significant serial peak flow rate variability.

Exposure criteria:

Minimum intensity of exposure: a minimum cannot be established since specific causative agents and thresholds are unknown.

Minimum duration of exposure: the disease may present within a few weeks after the first exposure or less commonly after an interval of several years.

Maximum latent period: several hours.