CASE REPORT

Interstitial lung disease due to fumes from heat-cutting polymer rope

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Abstract
Interstitial lung disease (ILD) due to inhalation of fume/smoke from heating or burning of synthetic polymers has not been reported previously. A fish farm worker developed ILD after cutting rope (polypropylene and nylon) for about 2 hours per day over an extended period using an electrically heated 'knife'. This process produced fume/smoke that entered the workers breathing zone. No other likely cause was identified. This case suggests that exposure to airborne contaminants generated by the heating or burning of synthetic polymers has the potential to cause serious lung disease.

Key words
Inhalational toxicology; interstitial lung disease; man-made synthetic fibres; occupational exposure; occupational respiratory disease.

Introduction
The term interstitial lung disease (ILD) covers a range of conditions affecting the lung interstitium often associated with fibrosis. Recognized causes include inhaled inorganic and organic substances, drugs, connective tissue disease and infection, in addition to idiopathic cases. Extrinsic allergic alveolitis (EAA) or hypersensitivity pneumonitis is a pulmonary inflammatory reaction that may occur in response to inhaled organic dusts and low-molecular weight chemicals including di-isocyanates. We describe a case of ILD, diagnosed as EAA, most likely caused by repeated exposure to smoke/fume from cutting polyethylene and nylon ropes.

Case report
A 59-year-old fish farm worker developed a severe respiratory illness in November 2009. Over several days, he developed cough productive of clear sputum and headaches. Antibiotic medication was prescribed, but his condition deteriorated. He was transferred to the Royal Hobart Hospital emergency department with low oxygen saturation levels. A diagnosis of acute exacerbation of chronic obstructive pulmonary disease was made. He was treated with antibiotics and prednisolone.

He was referred to a respiratory clinic in December 2009. The worker was an ex-smoker (16 years) with no history of respiratory disease, eczema, hay fever or other allergic conditions. Lung function tests showed airflow limitation, impairment of diffusing capacity and a restrictive abnormality. High-resolution computerized tomography showed increased ground glass and fine reticular opacities within the bases of both lower lobes, which persisted on prone views (Figure 1). There was an area of increased opacity within the right middle lobe, associated with crowding of bronchovascular markings, thought to be a benign process due to atelectasis or an old infective process.

A diagnosis of EAA was made in March 2010 on the basis of an open lung biopsy demonstrating widespread interstitial inflammation, largely peri-bronchiolar in location. In the affected areas, the interstitium was expanded by lymphocytes and histiocytic cells, with occasional ill-defined granulomata seen. Precipitins to aspergillus, pigeon, budgerigar and poultry antigens were negative. Viral serology showed no evidence of recent infection with influenza or pertussis. There was a peripheral blood eosinophilia (0.77/nl).
Prior to the onset of his illness, he had worked at a fish farm for about 10 years. He had not been involved in handling fish feed or fish processing in recent years. More than 3 years previously he had handled fish waste to make compost. He had also been employed at a limestone quarry, on a fishing boat, in an apple orchard and as a truck and tractor driver. He had worked in the timber industry, but not with Tasmanian Blackwood timber. There was no use of isocyanate containing products at home or at work. He did not own any birds. His main recreational interest was fitness running.

At the time of onset, he had been cutting polypropylene and nylon rope using an electrically heated ‘knife’ for about 2 hours per day without mechanical ventilation. The work was performed in a small shed open on one side. A dark fume/smoke was generated during the cutting process, especially when cutting wet 30-mm-thick rope. By June 2010, when a site visit was undertaken, mechanical exhaust ventilation had been introduced, but without it the fume from rope cutting was observed to readily enter a worker’s breathing zone.

There had been earlier episodes of respiratory illness with coughing, wheezing and sputum production recorded in August 2008 (serology indicating influenza B infection), February 2009, June 2009 and October 2009.

The worker improved and returned to work in March 2010 driving a crane in the vicinity of the rope-cutting operation but avoiding cutting rope. His symptoms worsened. Three weeks after returning to work, there had been a significant deterioration in lung function. He did not continue at work and has remained off work since.

Discussion

This is the first report of confirmed ILD caused by the inhalation of fume/smoke from the heat-cutting of nylon and polypropylene rope. The absence of any other identified cause, in particular avian/mammalian proteins, microbial spores or di-isocyanates, and the temporal association between exposure and the development of respiratory symptoms, makes this the most likely explanation. There is no information in the literature about...
the frequency with which biopsy-proven EAA occurs in the absence of an obvious cause.

It is surprising that this association has not previously been reported given the widespread use of polymers in industry. It is possible that occupational causation may have been missed due to lack of awareness of occupational exposure or was dismissed due to the absence of literature reports. Alternatively, the specifics of exposure in this case may have created an unusual degree of risk.

Acute effects of polymer fume exposure are well described [1] with temporary systemic symptoms occurring several hours after exposure, often leading to a mis-diagnosis of viral ‘flu’. However, reports of respiratory disease following polymer fume exposure are infrequent, with case reports of deterioration of obstructive pulmonary disease following multiple episodes of polytetrafluoroethylene exposure [2], and a case in which a computed tomography scan revealed patchy interstitial shadows in both lungs and pulmonary function tests a moderate decrease in the diffusing capacity [3]. While the features of the latter case are in keeping with ILD, no diagnostic procedure was undertaken to confirm this, in contrast to our case.

Further support for the association is the finding of ILD due to the inhalation of synthetic fibres of nylon by Pimental [4] and Kern [5]. Kern described biopsy-proven ILD due to occupational exposure to nylon flock.

As the chemistry of burning synthetic polymers is complex, it is possible that the fume/smoke involved contained agents known to be associated with EAA, such as isocyanates or acid anhydrides, or other unidentified agents. No occupational hygiene studies were undertaken, however, as it was considered likely that the fume/smoke would be a complex mixture without relevant exposure standards.

Our case could be considered to be a variant of ‘flock-workers lung’ but differs in that the responsible agent is likely to be a combustion product rather than dust generated from synthetic fibres. While this case could also be idiopathic, it seems unlikely that the workplace exposure was coincidental.

Key points
- Exposure to fume/smoke generated by the heating or burning of synthetic polymers may cause interstitial lung disease. Further research on this subject would be of value.
- Simple measures, such as exhaust ventilation to remove fumes from a workers breathing zone, are logical preventative measures.
- The diagnosis of extrinsic allergic alveolitis is complex and requires a consistent clinical and occupational history, as well as detailed investigations.

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Conflicts of interest
None declared.

References