Occupational Medicine 2017;**67:**308–310
Advance Access publication 8 April 2017 doi:10.1093/occmed/kqx039

Biopsy-proven hypersensitivity pneumonitis caused by a fluorocarbon waterproofing spray

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Background We present the case of a 35-year-old male who developed a chronic hypersensitivity pneumonitis (HP) following inhalational exposure to a fluorocarbon waterproofing aerosol spray, caused by his work for an upholstery and soft furnishings retailer. This is the first case report from inhalational fluorocarbon exposure with histological evidence of chronic HP. This is then discussed in the context of previous reports of interstitial lung disease and lung injury, caused by similar occupational and non-occupational exposures.

Key words Hypersensitivity pneumonitis, occupational chemicals, occupational respiratory disease, toxic inhalation.

Introduction

Fluorocarbon resins are compounds comprising fully or partially fluorinated hydrophobic alkyl chains. They are heat resistant up to 260°C and are used as thermo-protective coatings in metal and plastic manufacturing, and waterproofing agents for leather, suede, textiles and paper as mists, aerosol sprays or waxes. Although some may contain impregnating agents such as polysiloxanes, melamine and beeswax, many household and commercial sprays use fluorocarbon-based resins in combination with volatile solvents (e.g. isopropanolol, xylene) and propellants (e.g. butane, compressed air). Inhalational injury caused by exposure to inhaled fluorocarbons is best described as polymer fume fever [1], an acute febrile illness with chest tightness, headache and breathlessness beginning 4–8 h after exposure to degradation products of heated polytetrafluoroethylene, and potentiated by cigarette smoking; reactive-airway dysfunction syndrome and acute respiratory distress syndrome have also been reported.

Case report

A 35-year-old previously fit Caucasian male was referred to the occupational lung disease clinic following video-assisted thoracoscopic surgery and lung biopsy. He presented with a 2-year history of dry cough, with progressive breathlessness on exertion, and 9.5 kg weight loss following 1 year after the onset of cough. There was no wheeze, fever, night sweats or sputum production. He took inhaled salbutamol 200 mcg 2–4 hourly for 2 years with no effect, and no other medications. There was no personal or family history of atopy or asthma. He had a 15-pack year smoking history and quit with the onset of cough. There was no illicit drug use. He had no exposure to birds, tropical fish, compost, moulds or humidifiers. At the onset of symptoms, he had been employed by a furniture retailer for 4 years as a warehouseman, receiving and processing soft furnishings for dispatch to showrooms. He removed plastic wrapping from new furniture, some of which he then sprayed with Guardsman Fabricoat aerosol, a fluorocarbon waterproofing agent for fabric, suede and nubuck upholstery. He sprayed almost daily for 20% of the work shift, in an enclosed indoor space, with no respiratory protection. There was no heat sealing or occupational exposure to isocyanates, anhydrides or metal working fluids. Clinical examination was normal and chest radiograph showed soft infiltrates in both upper zones (Figure 1A). On laboratory investigation, full blood count including total eosinophil count, C-reactive protein, total immunoglobulins (E, G, A, M), rheumatoid factor, serum angiotensin-converting enzyme, anti-nuclear cytoplasmic antigen, anti-nuclear antigen, avian and fungal specific IgG (chicken, budgerigar, pigeon, *Aspergillus* spp., *Micropolyspora faeni*) were normal.

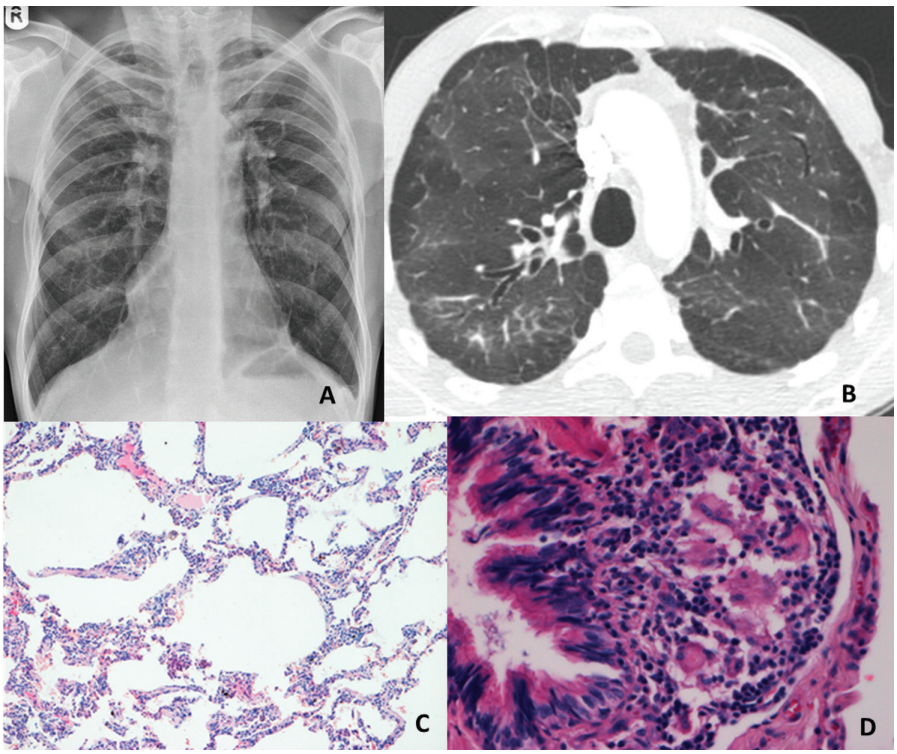

Figure 1. (A) Chest radiograph showing soft infiltrates and reticulation in upper zones bilaterally; (B) high-resolution computed tomography image of thorax in transverse section, at the level of the aortic arch, showing bilateral upper lobe ground-glass opacity with mosaic attenuation, interlobular septal thickening and bronchial dilatation; (C) lung histology at ×10 magnification, showing widening of the interstitium with a moderate lymphocytic infiltrate and mild patchy fibrosis; (D) peri-bronchiolar micro granuloma at ×40 magnification.

Pulmonary function testing revealed forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) = 89%, FEV_1 = 3.76 L (88% predicted), FVC = 4.15 L (82% predicted), diffusing capacity of the lung for carbon monoxide (DLCO) = 7.7 mmol/min/kPa (66% predicted) and gas transfer co-efficient (KCO) = 1.22 mmol/min/kPa/L (76% predicted). High-resolution computed tomography demonstrated bilateral upper lobe ground-glass opacities with diffuse mosaic attenuation (Figure 1B). He underwent a fibre-optic bronchoscopy with no pathological evidence of mycobacterium tuberculosis or other infection. However, no cell count was performed. Histology of upper pulmonary lobes showed interstitial widening and lymphocytic infiltrate, peri-bronchiolar granulomata and fibrosis (Figure 1C and D), consistent with hypersensitivity pneumonitis (HP). After exclusion of other causes and multi-disciplinary consensus, as has been recently recommended [2], the diagnosis was HP caused by inhalation of fluorocarbon. A repeat computed tomography scan undertaken following 6 months sickness absence, relocation away from exposure, and without steroid treatment, showed resolution of ground-glass opacities but persistent upper lobe fibrosis. He was subsequently dismissed from work and eventually made a successful common law compensation claim. At 5 years after diagnosis, his lung function remained stable: FEV_1 = 3.7 L (90% predicted), FVC = 4.92 L (98% predicted), DLCO = 7.9 mmol/min/kPa (69% predicted).

Discussion

Cases of acute pneumonitis occurring after inhalation of waterproofing spray, usually leather, nubuck or upholstery protector, have been described [3,4]. The classic presentation is of a worker or consumer attending an emergency department with breathlessness, dry cough and fever that occurs immediately or within several hours of inhalation, and frequently after a single exposure in an enclosed space; the individual is hypoxaemic, with leucocytosis and bilateral radiographic infiltrates. Evidence for treatment with corticosteroids is limited to case reports and many patients experience self-limiting illnesses; some develop fibrotic disease [3]. Where pathological diagnoses have been sought in an acute setting, bronchioloalveolar lavage has shown neutrophilia or eosinophilia and lung histology has shown septal oedema, neutrophilic or eosinophilic alveolar migration and proliferation of type II alveolar cells [3–5]. This is in keeping with animal studies demonstrating direct toxic alveolar injury due to fluorocarbons [6]. Sub-acute or chronic presentations have been described, usually following daily occupational exposure in confined spaces over a number of weeks [3,7]; however, one individual, memorably described as having 'hill walkers' lung', developed a sub-acute toxic alveolitis with no granulomata formation following a single episode of spraying walking boots [8]. Where individuals have developed established

fibrotic lung disease, histology has shown desquamative interstitial pneumonia [7] or non-specific fibrosis [3]; no authors have described HP on biopsy.

Several outbreaks of respiratory disease due to fluorocarbon waterproofing sprays have been reported to surveillance schemes in the UK, USA and Europe [4]. The majority were acute presentations following indoor exposures, and clinical features were suggestive of either toxic alveolitis or hypersensitivity. None of the exposures involved heated fluorocarbons. Most reports have followed worldwide changes in legislation (after 1989) as a result of the Montreal Protocol, requiring the reformulation of aerosols to remove the ozone-depleting solvent trichloroethane. It has been suggested that because traditional fluorocarbon resins have poor solubility in newer chlorofluorocarbon-free solvents, they have been modified to increase their solubility, resulting in a smaller droplet size [9]. Furthermore, the newer solvents, such as heptane, may allow fluorocarbon resins to travel further than the bronchial tree and reach alveoli [4]. The alveolar mechanism remains unknown, though Yamashita and Tanaka [10] have suggested a direct effect of fluorocarbons on type II alveolar cells may cause increased surface tension, reducing the effect of surfactants, leading to airway collapse and gas exchange impairment. This is the first case of histologically proven HP in the context of a regular and latent inhalational exposure to fluorocarbons in an occupational setting.

Key points

- Exposure to fluorocarbon aerosol can be encountered at work and at home in waterproofing sprays for leather, nubuck and other textiles; these have been reported to cause toxic alveolitis and outbreaks of respiratory illness.
- Our patient is the first reported case of occupational hypersensitivity pneumonitis caused by a fluorocarbon waterproofing spray, diagnosed after lung biopsy and exclusion of other causes.
- The best prognosis for a patient diagnosed with hypersensitivity pneumonitis lies with removal from exposure to the causative agent; even then they may experience a chronic or progressive fibrosis with lung function deficit or decline.

Conflicts of interest

S. T. is a speaker for Roche Diagnostics and received travel support from them. The other authors declare no conflicts of interest.

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