Work-related asthma in an aircraft engine mechanic

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Introduction

Air pollutants, both gaseous and particulate, have been shown to be associated with hospital admissions for lung and heart disease and, particularly in children, with variation in symptoms and peak flow (1). While exposure to ambient volatile organic compounds (VOCs) has been linked with respiratory symptoms (2), there is scant evidence for the involvement of VOCs in the aetiology of asthma apart from one report of occupational asthma induced by diesel exhaust in rail road workers (3). Aircraft maintenance workers are exposed to high levels of aviation fuel vapours and jet stream emissions from aircraft engines. We report a case of work-related asthma in an aircraft engine mechanic exposed to aviation fuel

Case report

A 42-year-old male smoker developed symptoms of cough, breathlessness and wheeze in 1982, coinciding with beginning a job as an aircraft engine mechanic. He worked at various airports in the U.K., and was exposed to aircraft fuel fumes during refuelling and to jet stream from aircraft engines. He usually worked on engines under cover but his exposure to fumes was similar to that of his colleagues. At other times he would be exposed to aviation fuel in a smaller laboratory and he described similar changes in symptoms in both environments. His symptoms worsened over the next 4 years when he was diagnosed as having asthma on clinical grounds. He was treated with inhaled Duvent (fenoterol and ipratropium bromide) 2 puffs q.i.d. and oral prednisolone 10 mg o.d. However, he remained symptomatic and required frequent increases in the dose of oral prednisolone for control of his asthma. He had reported to his general practitioner that his symptoms improved when away from work for periods of 2-3 days and particularly on holiday.

He was referred to our department in 1991 and started on inhaled beclomethasone (BDP) 500 µg b.d. Measurements of peak expiratory flow (PEF) demonstrated the characteristic pattern of work-related asthma (Fig. 1) with fall in PEF on five exposure periods and increased in four rest periods. His eosinophil count was normal at 0.04 x 10⁹ l⁻¹, IgE levels were normal at 30 KU l⁻¹ (normal range 0-200) and RAST to cat dander, house dust mite and grass pollen were negative. Spirometry showed an FEV₁ of 3.87 l and FVC of 6.05 l (predicted values 3.87 and 4.82 respectively). The dose of inhaled BDP was increased stepwise to 1000 µg t.i.d. and salmeterol 100 µg b.i.d. was added to control his symptoms while continuing regular treatment with ipratropium bromide 80 µg q.i.d. and salbutamol as required. He was advised to reduce exposure to aircraft fuel and jet stream at work with resulted in some improvement in asthma control. Bronchial provocation testing was not performed due to his peripatetic job. In 1995 he was investigated for symptoms of tiredness and maxillary sinusitis, and was found to have developed acute myeloid leukaemia. He was treated with cytarabine, daunorubicin and etoposide and a remission was successfully induced. He has remained away from work and over this time his asthma has improved substantially, now taking inhaled BDP 500 µg b.d. and infrequent salbutamol on demand. His PEF readings now range between 450 and 500 l min⁻¹.

Discussion

This is the first reported case of occupational asthma in an aircraft engine mechanic. We have based the diagnosis on a typical history and, in most exposure periods, peak flow changes in relation to exposure although some periods of exposure caused no change in peak flow. We accept that without bronchial provocation test the exact causal substance(s) remain in doubt but ethically this is impossible in this man. It is plausible that fuel vapour or combustion products may contribute to the cause of this man's occupational asthma.

Though aviation fuel (kerosene) differs from motor fuel (petrol and diesel), exhaust emissions from the jet engines are essentially similar to petrol cars and contain nitrogen oxides, carbon monoxide, carbon dioxide, VOCs, aldehydes, hydrocarbons and particulate matter. The exact composition of emission from the aircraft engines depends on the manoeuvre performed by the aircraft. During taxiing and landing there is incomplete combustion of fuel leading to an increase in unburned hydrocarbons, while during take off there is complete combustion leading to an increase in nitrogen oxide emissions.

Diesel exhaust has been implicated as a cause of occupational asthma in rail-road workers (3) although which component (gaseous or particulate) may have been the causal factor is unclear. The evidence for VOCs as a cause of symptoms in our patient can be supported by various pieces of epidemiological evidence. Asthma has
been reported in women after exposure to domestic kerosene fumes (4), while VOCs have been associated with decline in lung function in asthmatic subjects at concentrations of 25 mg m\(^{-3}\) (5), levels which are not unusually found in an occupational environment. An increased incidence of respiratory symptoms and asthma has been reported in children living in an area of high ambient VOC levels (2), while we have previously shown that airport workers exposed to aircraft fuel are more likely to report a history of productive cough (5). However, despite this evidence which might support VOC exposure as a potential cause in our subject's occupational asthma, it is impossible to decide which component of burnt or unburnt fuel was the responsible factor.

References


