

On examination he had no fever but had notable photophobia and nuchal rigidity. There were no other abnormal physical findings. Investigations showed that he had a normal full blood count and an erythrocyte sedimentation rate of 3 mm in one hour. His cerebrospinal fluid was under normal pressure with a leucocyte count of $1045 \times 10^6/l$ (all mononuclear cells). Cultures, including that for mycobacterium tuberculosis, were negative. Serological testing for cytomegalovirus, herpes simplex, adenovirus, varicella zoster, mumps, Epstein Barr virus, and arbovirus antigen (including tick borne encephalitis virus) gave negative results. Clinically the boy slowly improved without specific treatment but he had continuing lethargy, fatigue, and sporadic arthralgia but no overt arthritis.

A month after his original presentation he was found disorientated and dysphasic, with nuchal rigidity, but no fever. He also had papilloedema, right sided hemiparesis, and a right facial palsy. A computed tomogram was normal and an electroencephalogram was consistent with meningoencephalitis. His cerebrospinal fluid was again examined. The white cell count was $374 \times 10^6/l$, protein was raised at 2100 mg/l, and results of bacteriology were negative. Over 48 hours his neurological signs resolved. Serological investigations confirmed antibodies to *B burgdorferi* at a titre of 1/1024, confirming recent infection by this organism. Subsequently he steadily improved, although he still complained intermittently of fatigue, lethargy, headache, arthralgia, and a non-productive cough.

Comment

When questioned veterinary and forestry professionals in the New Forest and adjacent areas reported no recent imports of deer or changes in the density of tick infestation. We know that many children and adults are bitten by ticks locally but we have never been given or spontaneously sought a history of tick bite or erythema chronica migrans. We cannot say whether our patient is an isolated curiosity, whether this particular illness has always been with us but unrecognised, or whether we may find ourselves with a new disease on our doorstep. We await the summer with interest.

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Asthma caused by pulverised fuel ash

Pulverised fuel ash is the residue that remains when pulverised coal is burnt in power stations. Low grade coal, ground to a particle size of 0.1-100 μm , is blown into boilers in jets of airborne coal particles, and the residual ash is collected in electrostatic precipitators. Pulverised fuel ash is an important byproduct of the power industry: it is used for land filling and, because it sets hard in the presence of lime and water, is widely used as a building material in breeze blocks. It has not previously been reported as causing asthma. We report a case of asthma in a worker at a power station that was caused by inhalation of pulverised fuel ash.

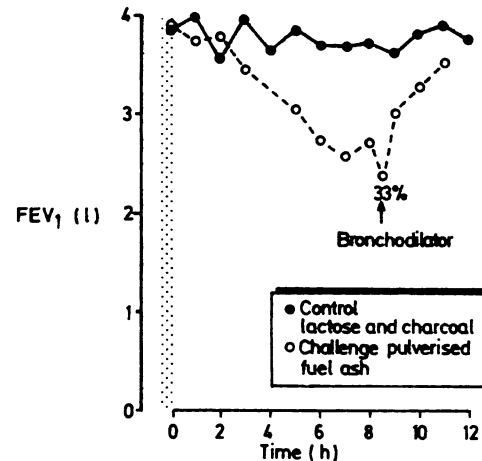
Case report

Nine months after starting work as a plant attendant at a power station a 27 year old man developed episodes of shortness of breath with wheezing, which improved at weekends and during holidays. His work included cleaning the filters of the bags. On two occasions he was admitted to hospital with acute severe asthma. He had suffered from hay fever since childhood, and skin prick tests with extracts of grass pollen and house dust elicited immediate reactions.

He recorded his peak expiratory flow rate every two hours for 28 days.¹ This deteriorated during work and improved when he was away from work. He underwent single blind occupational type inhalation tests.² Tipping of 250 g of lactose and 2.5 g of charcoal (producing a dust that looked like pulverised fuel ash) for 30 minutes elicited no change in his forced expiratory volume in one second, but tipping of 250 g of lactose with 2.5 g of pulverised fuel ash provoked a

late asthmatic reaction (figure). This late reaction was reproduced by a second inhalation test with the ash. The concentration of histamine required to cause a 20% fall in forced expiratory volume decreased from 12 mg/ml one day before the inhalation test with pulverised fuel ash to 2.5 mg/ml 24 hours after the test. Serum neutrophil chemotactic activity increased by 120% during the late asthmatic reaction.³

After these results he was relocated at work to prevent him being exposed to pulverised fuel ash any longer. He remained free of symptoms and required no treatment. Serial measurements of peak expiratory flow rate after he was relocated showed no evidence of asthma.



Inhalation test with challenge period shaded.

Comment

This patient's symptoms suggested that he had developed asthma related to his work, and this was confirmed by the results of serial measurements of his peak expiratory flow rate. Inhalation testing with pulverised fuel ash, to which he was exposed at work, provoked a reproducible asthmatic reaction. This late response was associated with a rise in serum neutrophil chemotactic activity (measured during the second inhalation test). The interval of nine months before the onset of his symptoms, the late asthmatic reaction provoked by the inhalation test with pulverised fuel ash, the associated increase in airway reactivity,⁴ and the resolution of asthma after relocation at work suggest that pulverised fuel ash was the primary cause of this man's asthma.

Pulverised fuel ash is a complex material: 60-80% of its weight is made up of water insoluble aluminosilicates, 15-30% of crystals (the main species being mullite, magnetite, carbon, and quartz), and the remaining 2-4% of water soluble sulphates of calcium, sodium, and potassium with trace quantities of arsenic, boron, copper, molybdenum, and selenium.⁵ We did not identify which of these materials, either alone or in combination, caused our patient's asthma.

Some 12 million tons of pulverised fuel ash are produced each year in the United Kingdom and 50 million tonnes each year in the United States. The greatest risk of developing asthma due to inhaled pulverised fuel ash is run by those working in the power and construction industries.

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