SHORT REPORTS

Non-fatal lung disease due to inhalation of nebulised paraquat

We report what we think is the first recorded non-fatal case of lung disease due to inhalation of nebulised paraquat.

Case report

In July 1977 a 64-year-old woman was in her garden when she noticed a "mist" drifting in from spraying operations in adjacent fields. After about 10 minutes she noticed tightness in her chest and retired indoors. Over the next week she became gradually more breathless. Her garden was totally defoliated by the mist, and a number of mice and voles were subsequently found dead. Inquiry confirmed that the mist was nebulised paraquat (Grammoxone) being sprayed on a root crop. About one week after exposure she consulted her general practitioner, who treated her with a short course of corticosteroids by mouth. She improved only slightly. When she attended hospital as an outpatient on 27 September 1977—that is, nearly two months after the initial exposure—she was seen to be a rather obese woman with severe dyspnoea on slight exertion. There was some wheeze and very scanty basal rales. There were no other significant features on clinical examination. Ventilatory function tests showed no evidence of airways obstruction but severe restriction (FEV_1 0·8 l, FVC 0·9 l, predicted FVC 3·2 l). Peak flow was 180 l/min. Chest radiographs were normal. She had a previous history of allergic rhinitis and chronic sinusitis, but had never before complained of dyspnoea. She had hip arthroplasties performed in 1973 and 1975.

She was thought to be suffering from interstitial lung damage due to the paraquat exposure, and was referred to the employment medical adviser. He thought that her symptoms were almost certainly due to the paraquat exposure, although this had not been previously described. She was kept on enteric-coated prednisolone tablets 2·5 mg twice daily. She failed to attend the clinic again until 28 February 1978. She was then much improved (FEV_1 1·6 l, FVC 2·0 l, peak flow 325 l/min). Chest radiographs remained clear.

Comment

We regret that it was not possible to undertake further investigations such as transfer factor, lung volumes, etc, and that the patient was not seen soon after exposure. Nevertheless, the detailed history gives strong grounds for believing that her symptoms were due to paraquat inhalation. A computer search of the world literature failed to find a similar case, although fatal lung damage to inhaled paraquat is well recognised.[1] We think that this case merits presentation since paraquat spray is widely used in agriculture and the risks may not always be fully appreciated. It is unlikely that this is an isolated case and we hope that it will stimulate a search for others.

[1] Crofton J, Douglas A. *Respiratory diseases*. 2nd ed. Oxford: Blackwell, 1975:691-2.

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Failure of interferon to modify Creutzfeldt-Jakob disease

No treatment is known to modify the course of Creutzfeldt-Jakob disease (CJD). Early reports of a possible beneficial effect of amantadine were not confirmed by clinical experience.[1] Although interferon has been reported to be ineffective in experimental scrapie,[2] we considered a clinical trial of interferon justified in CJD. Despite the obvious similarities in the physical properties of the causative agents of scrapie and CJD[3] and in the clinical and pathological features of these disorders the two agents are not identical.[3] In order to study the possible effect of interferon on the clinical course of CJD we gave 3×10^6 units of human leucocyte interferon as daily subcutaneous injections to two patients with clinically and pathologically confirmed CJD for 4 and 13 months, respectively.

Case reports

(1) A 50-year-old woman, an industrial cleaner, became tired and irritable and complained of impaired memory in the late spring of 1978. In late October she was unable to continue her work after one month's history of progressive dizziness. When admitted to hospital in early November she could not walk unaided and complained of visual disturbance. She had dementia and signs of cerebellar involvement. Electroencephalogram (EEG) showed slow activity with regular bursts of high-voltage delta waves, and slight cortical atrophy was seen in the computerised tomography scan. Routine examinations of cerebrospinal fluid were normal. The disease progressed within two weeks to a bedridden state with disorientation, hallucinations, and temporary rigidity of the arm flexors. When admitted on 18 December to the department of neurology, University of Helsinki, she was anarthric but alert and followed moving objects by gaze. She was able to obey a few simple commands—for example, put the tongue out. All limbs were rigid with diminished tendon reflexes. The plantar reflex was flexor. The head, face, and arms showed occasional myoclonic jerks. EEG showed repetitive triphasic high-voltage discharges, considered to be compatible with CJD. Treatment with human leucocyte interferon was started on 5 January 1979. She deteriorated progressively and interferon was stopped in May. She died in August, after an illness of 15 months. Necropsy confirmed the presence of spongiform encephalopathy.

(2) A 50-year-old woman telephonist first noticed an impairment of recent memory in January 1978. She was a member of a Finnish family with several CJD patients (family S, case V-18).[4] The duration of the disease in the family had varied from 16 to 48 months and the patients had become bedridden after 15 to 20 months. Their EEG had been characterised by slow activity without repetitive discharges.[4] The diagnosis of CJD was confirmed by brain biopsy in June 1978. Treatment with human leucocyte interferon was started in August 1978. At that time she showed a definitive impairment of memory without significant alteration in verbal reasoning. She was orientated to time and place. She had an unsteady gait as well as tremor and signs of an upper motor neurone lesion. While on interferon treatment her illness slowly but steadily progressed and she became an inpatient in February 1979. In early September 1979 she was anarthric and bedridden with spastic tetraparesis and twitching tremors. Interferon treatment was stopped.

Comment

Interferon apparently had no influence on the course of the disease in either case. Although the first patient was already at the final stage when treatment was begun, a further clinical and electroencephalographic deterioration was evident afterwards. In case 2 the course of the illness did not differ significantly from that in the other CJD patients in the family.[4] Human leucocyte interferon is efficiently transported into the circulation from subcutaneous injection sites. Although the concentration of interferon in cerebrospinal fluid is about 1/30 of that in serum,[5] its penetration to the brain tissue is not known. A trial of intrathecal interferon in CJD might be worth while.

[1] Ratcliffe J, Rittman A, Wolf S, Verity MA. Creutzfeldt-Jakob disease with focal onset unsuccessfully treated with amantadine. *Bull LA Neur Soc* 1975;**40**:18-20.
[2] Gresser I, Pattison IH. An attempt to modify scrapie in mice by the administration of interferon. *J Gen Virol* 1968;**3**:295-7.
[3] Gajdusek DC. Unconventional viruses and the origin and disappearance of Kuru. *Science* 1977;**197**:943-60.
[4] Haltia M, Kovanen J, van Crevel H, Bots GTAM, Stefanko S. Familial Creutzfeldt-Jakob disease. *J Neurol Sci* 1979;**42**:381-9.
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