In April 1976 he developed an acute exacerbation of his bronchitis accompanied by cor pulmonale. He failed to respond to treatment and died four days after admission to hospital. Necropsy examination was not performed.

Discussion

In view of the disappearance of this man's bovine cough at the same time as the improvement in his other Parkinsonian features during treatment with levodopa and benserazide, it is difficult to come to any other conclusion than that the bovine cough was the presenting feature of his Parkinsonism. So far as possible other causes of the abnormal cough were eliminated. In any case, it is extremely unlikely that a bovine cough due to another cause, and already present for two years, would respond so specifically to antiparkinsonian therapy.

The presence of the bovine cough with normal findings on laryngoscopy are difficult to explain other than on the basis that the laryngeal dyskinesia was insufficient to impair the production of normal cord movement during laryngoscopy, but that it was sufficient to impair the rapid movement and co-ordination of the vocal cords during coughing.

I would like to thank Dr M G Carter, Adverse Reactions Physician, Roche Products Ltd, for help in searching literature for possible previous reports of this phenomenon.

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Leptospirosis presenting with profuse haemoptysis

About fifty cases of leptospirosis are diagnosed in the United Kingdom each year with a mortality of 5%.¹ Haemoptysis and pneumonitis are uncommon manifestations of the disease and both occurred in the patient described below.

Case history

A farmer's son of 16 was admitted with an 18-hour history of frank haemoptysis. During the previous five days he had been unwell with fever, night sweats, general malaise, and muscle aches. These initial symptoms improved for twelve hours before the onset of cough and haemoptysis. Examination on admission showed signs of right lower lobe consolidation, cyanosis, blood-stained sputum, tachypnoea, and fever (39°C); he had eczematous areas on both hands. Investigations disclosed haemoglobin 9.5 g/dl; white blood count $9.7 \times 10^9/l$; platelet count $70 \times 10^9/l$; serum urea concentration 8.3 mmol/l (50 mg/100 ml); and bilirubin concentration 27 mmol/l (1.73 mg/100 ml).

On the assumption that he had an overwhelming bacterial infection treatment was started with intravenous benzyl-penicillin 2 mega-units four-hourly. Eighteen hours later cloxacillin, 0.5 g intravenously six-hourly, and dexamethasone, 4 mg intravenously six-hourly, were added because of increasing respiratory distress and toxæmia. The day after admission haemoptysis increased. Clinical signs of extensive right lung consolidation with spread to the left lower lobe were found. The chest radiograph (see figure) showed extensive opacities in the right lung field and opacities developing in the left lung field. The bleeding became so profuse that endotracheal intubation with repeated bronchial aspiration was needed to prevent asphyxiation by his own blood; a blood transfusion was given. Epsilon amino-caproic acid, 3 g intravenously four-hourly, with platelet transfusions were given to control haemorrhage; haematuria was now present. Clotting tests showed: fibrinogen 6.2 g/l, falling to 1.4 g/l on the third day; prothrombin time 16 seconds (control 12 seconds); partial thromboplastin time 32 seconds; platelets $68 \times 10^9/l$; fibrin degradation products 10–40 µg/ml.

He was ventilated with a Cape ventilator using an inspired oxygen concentration of 100%, a tidal volume of 1000 ml, and a positive end expiratory pressure of 20 cm H₂O. Despite this the pO₂ was only just maintained above 6.4 Kpa (50 mm Hg). To maintain the blood volume, 3.5 litres of whole blood and four packs of platelets containing about 20 ml each were transfused in the first four days. On the third day splenomegaly was noted. Mechanical ventilation was continued for five days and on the sixth day the patient was weaned from the ventilator. Recovery was then rapid and he remains in good

Chest radiograph on seventh day of illness.

health eighteen months later. Complement fixation titres of leptospiral antibody rose from 1/10 on day of admission to 1/160 nine days later. Agglutination tests showed a rise from 1/10 to 1/100 for *Leptospira icterohaemorrhagiae*.

Discussion

This case is presented to emphasise the severity of the haemoptysis, which nearly asphyxiated the patient. In the early stage of the illness there were many features of an infective pneumonitis, but the diagnosis was only made in retrospect by a rising titre of serum antibody to *L. icterohaemorrhagiae*. Leptospiral infections usually present with non-specific features of fever, muscle pains, and headache but should be considered in the differential diagnosis in cases with pneumonitis and haemoptysis.² Pneumonitis has been reported in 11% of cases, haemoptysis in 3%, thrombocytopenia in only 1%.³ Splenomegaly is rare, occurring in less than 1%. The bleeding diathesis in this syndrome is said to be due to leakage through the vascular endothelium. Haemolysins are produced⁴ and intravascular haemolysis occurs; our patient had some features of haemolysis and disseminated intravascular coagulation. The patient described was of the usual age, sex, and occupation³ and probably acquired the infection through eczematous fissures on his hands from rat-contaminated cattle feed. He was treated with blood, platelets, corticosteroids in high dosage, and epsilon amino-caproic acid. Large doses of penicillin were also given initially and are recommended for this condition.⁵ Which of these therapies contributed to his survival is uncertain, but without the skill of the intensive care staff he would have died.

We are indebted to Dr R J C Hart and Dr L H Turner of the Public Health Laboratory Service for making the diagnosis and to Dr J O P Edgcombe for his haematological advice.

¹ Turner, L, *Medicine*, 1975, 3, 125.

² Poh, S C, and Soh, C S, *Thorax*, 1970, 25, 75.

³ Heath, C W, Jr, Alexander, A D, and Gatton, M M, *New England Journal of Medicine*, 1965, 273, 857.

⁴ Beeson, P B, and McDermott, M D, *Textbook of Medicine*, p 438. Philadelphia, W B Saunders Company, 1975.

⁵ Kocen, R S, *British Medical Journal*, 1962, 1, 1181.

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