in Orientals), or myasthenia gravis.⁴ Initially, bulbar muscle dysfunction was believed to complicate a pre-existent, proximal skeletal muscle weakness. Kammer and Hamilton² and other workers showed that hyperthyroid patients without evidence of chronic myopathy sometimes presented with dysphagia. Such patients complained of dysphagia, nasal regurgitation of solids and liquids, voice changes, diplopia, facial paresis, and diminished tongue strength. Nasogastric feeding and intensive physiotherapy to protect the airway was sometimes required. This patient presented acutely and had all of these symptoms but, unlike similar patients, required artificial respiration for six weeks because of complete paralysis of laryngeal and intercostal muscles and inability to swallow.

This case shows that hyperthyroid patients without chronic myopathy may present with a disabling non-myasthenic bulbar myopathy that may require intubation or tracheostomy because of the acute bulbar paralysis.

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Pronounced cerebellar features in legionnaires' disease

Confusion and disorientation are common in legionnaires' disease, and isolated localising neurological signs occur occasionally. We report a case of *Legionella pneumophila* pneumonia associated with pronounced cerebellar disease.

Case report

A 47-year-old man presented with a 48-hour history of malaise, generalised weakness, and shivering attacks, associated with production of mucopurulent sputum. In the 24 hours before admission he had had several episodes of vomiting and diarrhoea and his gait had become so unsteady that he was barely able to stand. On waking on the day of admission he had pronounced slurring of his speech.

Since undergoing a splenectomy for hereditary spherocytosis 29 years previously he had had several episodes of pulmonary embolism and deep vein thrombosis and remained permanently on anticoagulants, but his therapeutic compliance was poor. He smoked 40 cigarettes and drank at least eight pints of beer each day, but had continued regular work.

On admission he appeared distressed (respiratory rate 30/min) and feverish (38.6°C) with a pulse rate of 100/min. There were crepitations and rhonchi throughout both lung fields. Despite pronounced dysarthria he was orientated in time, place, and person, and he co-operated during history and examination. Examination of cranial nerves showed no abnormality but nystagmus was present. Co-ordination was impaired on finger-nose testing, and he had associated dysmetria and dysidiadochokinesia. Co-ordination on heel-toe testing was similarly impaired, with slight weakness in the left leg. Plantar responses were flexor. There was shadowing in the lingula area on a chest x-ray film. Investigations showed a haemoglobin concentration of 16.7 g/dl, white cell count of $24 \times 10^9/l$ (mainly neutrophils), urea concentration of 9.6 mmol/l (57.6 mg/100 ml), and serum sodium concentration of 127 mmol(mEq)/l. Despite improving initially with intravenous ampicillin he had a sudden cardiorespiratory arrest on the fifth day of his illness. Legionnaires' disease was considered at this stage, and intravenous erythromycin was given for two weeks followed by amikacin, metronidazole, and cloxacillin because of persistent fever and raised white cell count. Ventilation had to be continued for six weeks, during which time he developed the clinical and neurological signs of adult respiratory distress syndrome (figure). Hepatic and renal function deteriorated during this period but returned to normal with conservative treatment. Dieterle silver stain consistently failed

to show *L. pneumophila* in his sputum, transtracheal aspirate, and trans-bronchial biopsy specimens. Examination of the cerebrospinal fluid showed no abnormality on two separate occasions. There was a rise in *Legionella* fluorescent antibody titre from 1/16 at five days to 1/512 at four weeks.

Limitation of upward gaze was the only residual neurological sign after recovery and this improved slowly. Two months after artificial ventilation was discontinued he still had restricted lung function and carbon monoxide transfer was 40% of that expected.

Chest x-ray film showing bilateral pulmonary disease leading to the development of adult respiratory distress syndrome in a patient with legionnaires' disease.

Comment

Many of the clinical features of legionnaires' disease were present in this case.¹ Although isolated ataxia has been described there has been only one well-documented account of profound cerebellar disease.² In all but one case in which neurological signs have been reported the cerebrospinal fluid was normal.²⁻³ Alcohol was considered as a cause of this man's cerebellar signs, but he regularly took Brewer's yeast tablets (0.2 mg thiamine daily), there was no evidence of an encephalopathy or delirium tremens, and the onset of acute cerebellar signs began 48 hours after his initial symptoms and were thus directly related to the onset of legionnaires' disease.

There were other interesting features in this case. Firstly, legionnaires' disease occurs more often in compromised hosts or in patients with underlying pulmonary disease, and splenectomy in this man may have further increased his susceptibility.⁴⁻⁵ Secondly, recovery occurred only after six weeks' artificial ventilation, and this is unusual though a permanent diffusion defect remains. Legionnaires' disease should be considered in all cases of pneumonia associated with cerebellar signs.

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