

Guillain-Barré syndrome associated with tuberculosis

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Summary

Two cases of Guillain-Barré syndrome occurring in association with chronic pulmonary tuberculosis are reported. A cell-mediated, delayed hypersensitivity reaction to, or invasion of the nerve roots by tubercle bacilli would seem to be the likely explanation of the neuropathy.

KEY WORDS: Guillain-Barré syndrome, polyneuropathy, tuberculosis.

Introduction

Guillain-Barré syndrome (GBS), acute inflammatory polyneuro-radiculopathy, is known to follow a variety of viral, mycoplasmal, bacterial and chlamydial infections (Arnason, 1975). In a review of the literature, Leneman (1966) found 8 out of 1100 cases of this syndrome to be associated with tuberculosis (of the lung and brain). The present communication records 2 cases of chronic pulmonary tuberculosis presenting with GBS.

Case reports

Case 1

An 18-year-old man was admitted to hospital with a 10-day history of weakness of all 4 limbs of acute onset. He had been unwell for the previous 6 months with loss of appetite and weight. He had cough of 3 months duration with scanty expectoration and low grade fever of 1 month duration. He had no symptoms referable to the bladder or the bowel. On examination, he was grossly emaciated. Bronchial breath sound was heard over the upper zone of the left side of the chest. All 4 limbs were markedly flaccid and weak, the weakness being more marked in the distal muscle groups. The tendon reflexes were diminished in the upper limbs and absent in the lower limbs. The plantar response was flexor. There was impairment of sensation to pain, temperature and touch in a glove and stocking distribution. The

joint position sense was impaired in the toes and fingers.

Laboratory investigations revealed the following abnormal results: erythrocyte-sedimentation rate, 7 mm in the first hour; cerebrospinal fluid (CSF) protein 210 mg/dl, sugar 75 mg/dl, no cells. Chest X-ray showed multiple confluent, ill-defined and ring shadows in both upper zones suggestive of pulmonary tuberculosis. X-rays of the vertebrae were normal. The patient was too weak to cough up a satisfactory specimen of sputum for the examination for acid-fast bacilli.

He was treated with rifampicin, 450 mg daily, isoniazid, 300 mg daily, streptomycin, 750 mg daily, pyridoxine, 6 mg daily and prednisolone, 10 mg hourly. Physiotherapy was given to the limbs. His cough diminished and appetite improved in 2 weeks of this regime. From the 3rd week onwards, the prednisolone was gradually tapered off. By the 4th week, he was able to move his upper limbs slightly. By the 10th week, he was able to walk unsupported. By the 4th month, there was marked clearing of the lung disease radiologically. From then on, rifampicin was omitted and he was given streptomycin, 1 g, and isoniazid, 700 mg twice a week. At review 10 months later, patient had completely recovered.

Case 2

A 56-year-old woman was admitted to hospital with a 3-week history of weakness of all 4 limbs. The weakness had started in the lower limbs and spread to the upper limbs in 2 days. She had cough of 6 months duration with scanty expectoration, low grade fever and loss of appetite of 2 months duration.

On examination, she was emaciated. In the chest there were crepitations in the upper and midzones bilaterally. She had marked flaccid weakness of all 4 limbs—the weakness being more marked in the distal muscle groups. All tendon reflexes were markedly diminished and the plantar response was flexor. Sensation to pin prick and light touch and joint

position sense were impaired in the distal parts of the limbs.

The abnormal findings on laboratory investigations were CSF: protein 800 mg/dl, sugar 70 mg/dl (no cells). Chest X-ray showed multiple confluent ill-defined and ring shadows in both upper and both midzones suggestive of pulmonary tuberculosis. X-rays of the vertebrae were normal. The patient was too weak to produce a satisfactory specimen of sputum. The same antituberculosis chemotherapy as for case 1 was instituted, but the patient took her own discharge on the 3rd day and died at home.

Discussion

The clinical and radiological features in the 2 cases were sufficiently characteristic to enable a diagnosis of pulmonary tuberculosis to be made despite the lack of bacteriological confirmation. The acute onset of flaccid paralysis of all 4 limbs, sensory impairment in the glove and stocking distribution and the markedly elevated CSF proteins in the absence of a cellular reaction conform to the description of GBS.

The incidence of Guillain-Barré syndrome in Western countries is 1–2 cases per 100000 per year (Schoenberg, 1978). No figures are available for Sri Lanka. The prevalence of tuberculosis in Sri Lanka in 1971 was 2.3/1000 adult population, and the

incidence 44.8/100000 in 1971 and 42.1 in 1980. From the above data, it would appear that the probability of both diseases occurring in the same individual by chance is very small.

The pathogenesis of GBS in infective disease is by no means clear. The majority view is that GBS represents a cell-mediated, delayed hypersensitivity reaction (Thomas, 1978) which is a recognized feature of tuberculosis. However, the possibility of tuberculous polyradiculitis being the cause cannot be ruled out in view of the report by Peiris, Wickramasinghe and Chandrasekera (1974) of a case of GBS demonstrated at necropsy to result from direct invasion of the nerve roots by tubercle bacilli.

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