

## **A CASE OF TUBERCULOID LEPROSY WITH EITHER WIDESPREAD NERVE INVOLVEMENT OR A SECOND NEUROLOGICAL DISEASE— A CASE REPORT**

Sir,

We wish to present the case of a 50-year-old Bhutanese farmer, who suffered numbness and tingling of the whole of the right arm and left leg, commencing in April 1992. He was admitted 5 days after onset to a general hospital in East Bhutan where he stayed for 3 months, with an interval of 2 weeks at home. No records are available from that time, but according to information we received from the hospital, he underwent anti-leprosy treatment, although no cardinal signs of leprosy were found. During hospital admission he developed weakness in the hands and feet and was unable to walk 'after an injection' (probably Vitamin B-complex). He was referred to our hospital in early August 1992.

There was no family history of leprosy or neurological disease and no history of poison intake or handling. On examination all muscles in the arms and legs were hypotonic and muscle strength was decreased. Because of weakness of the quadriceps muscle on both sides he was unable to walk. The tendon reflexes were absent and there was sensation loss in the arms and legs. There was tenderness over the lower part of the spine. Cranial nerves were intact. Neither enlarged nerves nor skin patches could be found, therefore on clinical grounds he was judged not to have leprosy and so referred the same day to the national referral hospital in Thimphu.

He was referred back to us with swelling of the face, hands and feet 3 weeks later. On examination we found 2 swollen and red, anaesthetic patches on the back and right shoulder, and several enlarged nerves (right ulnar, both lateral popliteal, left radiocutaneous, left greater auricular). Tendon reflexes were still absent, but the abdominal skin reflexes remained. Position sense seemed impaired, but that could be due to language problems (as he could take a match out of a box and strike it without much difficulty). Detailed VMT showed bilateral weakness of the ulnar, median, lateral popliteal and posterior tibial nerve innervated muscles as well as right side radial nerve paresis. Weakness was also noted in the quadriceps femoris muscles bilaterally. Facial nerve was intact. Sensation loss was found in both forearms and both legs from the groin downwards as well as the lower part of the buttocks. Pressure sores were present over both greater trochanters. Skin smears for AFB were negative. VDRL was weakly positive, but the Rapid Plasma Reagin card test was negative.

The patient was diagnosed as having type I leprosy reaction and he was put on Prednisolone 60 mg OD as well as MB MDT (WHO) on 2 September 1992—2 new patches on the back and left upper arm appeared 2 days later, but subjectively he improved soon after the start of treatment.

Skin and nerve biopsies were performed. The skin biopsy was badly damaged during transport to the UK and showed 'inflammation, not inconsistent with leprosy'. The nerve biopsy of left radiocutaneous nerve showed 'a florid neuritis with some granulomas but no AFB. This must be leprosy, tuberculoid.' A neurologist we consulted believed that it was most likely a case of Guillain-Barré polyradiculitis or polyneuropathy. The CSF taken on 19 September 1992 showed normal protein, sugar and cell contents, but this does not exclude Guillain-Barré syndrome. Electric diagnostic studies of peripheral nerves are not possible in Bhutan.

During the following months muscle function improved slightly, but on discharge he was still unable to walk due to weakness of the thigh muscles. His sensation did not improve.

In summary, this appears to be a case of histopathologically confirmed tuberculoid leprosy with either widespread nerve involvement including the femoral nerves or, more likely, with a second neurological disease (Guillain-Barré syndrome?—which however did not improve significantly). We were unable to find a similar case that had been reported.

The possible cause could be leprosy involvement of the central nervous system, but 'ordinarily *M. leprae* do not invade the central nervous system in humans. There are only 3 instances in the literature in which leprosy bacilli have been detected in the central nervous system. *M. leprae* have been found in the interstitial tissue in dorsal root ganglia, Gasserian ganglia and also in anterior horn cells, but never in the cerebrum. There was no evidence of destruction of neuronal cells to cause any sensory or motor nerve changes.' (Dr C. K. Job, personal communication.)

It is possible that the presenting disease was not leprosy, but that the tuberculoid leprosy became manifest only after the commencement of anti-leprosy treatment. This is observed in other patients as well. (Dr M. F. R. Waters, personal communication.)

Gidakom Hospital  
The Leprosy Mission  
P.O. Khasadrapchu  
Butan

F. J. MOET &  
STEEN M. ANDERSEN

*Acknowledgement:* Dr Sebastian Lucas kindly prepared and examined the biopsies.