RADICULOMYELOPATHY DUE TO TYPHOID FEVER

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SYNOPSIS

Myelopathy is a rare neurological manifestation of typhoid. An eighteen year old Malay male presented with clinical features of Typhoid fever and radiculomyelopathy. Electromyography and nerve conduction studies were suggestive of motor radiculopathy. The patient made a complete recovery with chloramphenicol therapy. The details of the case and review of the literature are presented.

INTRODUCTION

Neuropsychiatric manifestations have been reported to occur in approximately 5% of patients with Typhoid fever (1). These include toxic-confusional states, encephalopathy, meningitis, convulsion, hemiplegia, aphasia, optic neuritis, deafness, peripheral neuropathy, Parkinsonian syndrome and transverse myelitis (2,3,4,5). Involvement of the spinal cord is a rare complication of Typhoid and the case reports so far describe an acute incomplete transverse myelitis (6,7,8,9). We report a case of radiculomyelopathy due to typhoid fever.

CASE REPORT

W.I. an 18 year old Malay male was admitted to the University Hopsital, USM with a history of prolonged fever of a month's duration associated with a non-productive cough. Two weeks prior to admission he had noticed progressive weakness of his lower limbs and had been bed ridden for a week. There was a history of typhoid fever in the neighbourhood. Examination at admission revealed that he was febrile, pale and toxic. He had coating of the tongue and hepatomegaly. The nervous system examination revealed normal higher mental functions and cranial nerves. Wasting and weakness of both quadriceps muscles were noted. Power was grade 3/5 in hip muscles, 2/5 in quadriceps and 5/5 distally. There were bilateral Babinski responses, absent knee jerks and exaggerated ankle jerks with ankle clonus. There were no other neurological deficits. Other organ systems were normal.

Investigations showed total leucocytic count 9800 cumm with polymorphs 51%, lymphocytes 48% and monocytes 1%. The Haemoglobin was 7.6 gm%, platelets count was 216000 cumm and ESR was 10 mm/1st hour. Blood culture grew no organism. The Widal test was positive with a four fold rise in titre. The values of TO 1/320 and TH 1/640 dilutions were obtained on two occasions when done at one week intervals. LE cells and antinuclear antibody were negative. A lumbar puncture and myelogram were not consented to by the patient and hence not done. Electromyography (EMG) was normal in the extensor digitorum brevis and abductor pollicis brevis, but showed evidence of chronic partial denervation in both quadriceps femoris. Distal motor nerve conduction velocity was 53 M/sec respectively in the left common peroneal and median nerves. The proximal motor conduction velocity calculated from F wave measurements was 42.5 M/sec in the left common peroneal nerve and 56 M/sec in the left median nerve. The above results indicate a reduced proximal conduction velocity in the left common peroneal nerve. Sural sensory nerve conduction velocity was 50 M/sec.

The patient was diagnosed as a case of Typhoid fever with radiculomyelopathy. He was treated with chloramphenicol 2 gm daily for 21 days. Within a week after starting treatment he became afebrile and by the second week he started to improve in motor power in his lower limbs. The patient was discharged on completion of chloramphenicol therapy. On review a month later, the patient had improved almost completely except for slight 4/5 weakness in his quadriceps femoris.

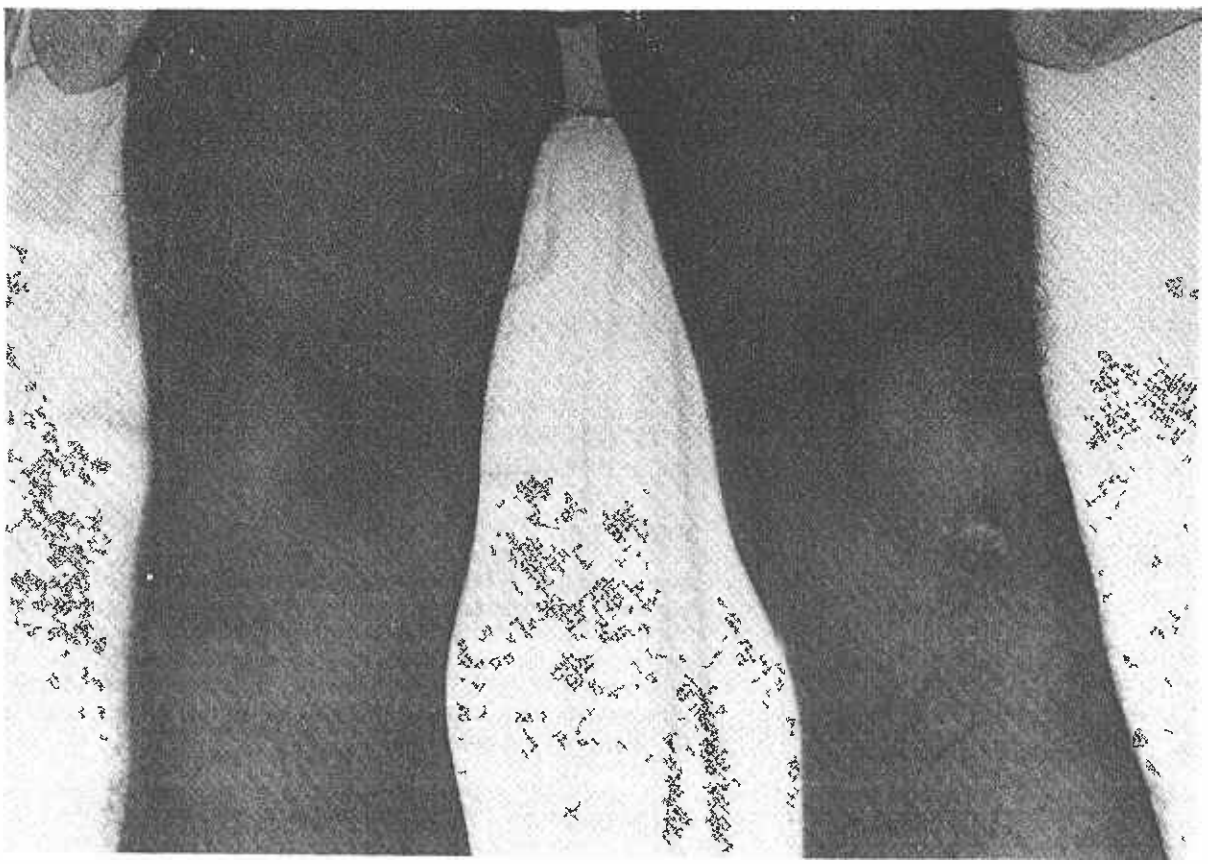

Fig. 1. Wasting of both quadriceps femoris muscles in typhoid radiculomyelopathy.

DISCUSSION

Typhoid fever is widely prevalent in Malaysia and endemic in Kelantan. From February — September 1986, 376 Typhoid cases have been admitted to the University Hospital, USM. Of these, only the present case had spinal cord involvement. This concurs with the evidence so far that myelopathy is an exceedingly rare manifestation of Typhoid. The cases reported so far have presented during the acute or convalescent phase of typhoid with sudden onset of spastic paraplegia (6,7,8,9). The relative absence of sensory signs and bladder dysfunction have led the above authors to postulate an incomplete transverse myelitis with good recovery. The present case was similar to the above cases in that only the motor system was involved, but different in that the patient had marked wasting of the quadriceps and absent knee jerks suggestive of lower motor neuron involvement. This was confirmed by EMG which showed evidence of chronic partial denervation. The normal distal motor nerve conduction and reduced proximal nerve conduction velocity suggested that the motor roots were involved. The positive Babinski signs and bilateral ankle clonus indicate an involvement of the pyramidal tracts in the spinal cord as well. Though a lumbar puncture and myelogram could not be done to exclude other causes of myelopathy, the consistently positive Widal test in high dilutions and the complete recovery with chloramphenicol indicate that typhoid was the aetiological factor.

The exact nature of spinal cord involvement in typhoid is unknown. Arteritis producing spinal cord infarction has been considered unlikely in view of the complete recovery (9). The endotoxin of Salmonella typhi has been thought of as a possible factor in producing the neurological manifestations (5). An alternative pathogenesis by Vassa et al suggested that the myelopathy could be a non specific allergic demyelinating form of disseminated encephalomyelitis which can occur as a reaction to a number of bacterial and viral illnesses (9). An explanation not considered by others so far is the possibility of a Typhoid granuloma in the spinal cord. A fine resolution spinal computerised tomographic scan should help to clarify the nature of the spinal cord involvement. In conclusion, radiculomyelopathy is a rare manifestation of Typhoid fever. A greater awareness and early diagnosis are indicated in this disorder that has an excellent prognosis with adequate treatment.

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