ACUTE POLYRADICULOPATHY IN MYCOPLASMA PNEUMONIA

G. DEVATHASAN
H.L. CHAN

SYNOPSIS

A young girl with mycoplasma pneumonia occuring in association with acute polyradiculopathy is described. This report also discusses this neurological complication of Mycoplasma Pneumonia infection and its possible casual relationship.

INTRODUCTION

The cause of acute polyradiuculopathy often termed Guillain-Barre Syndrome) is unknown. Although the clinical picture suggest an infection or reaction to an infection, in the great majority of cases, no associated infection is demonstrable. We document here a patient with acute polyradiculopathy occuring in close association with a mycoplasma pneumonia. Until 1973, only about 15 cases of this association has been reported in the Enlish literature (Lorer and Kalvasky, 1973). No mention of this is made in standard medical (Knight, 1977) and neurological textbooks (Merrit, 1973).

CASE REPORT

A 15 year old Chinese girl presented with fever and weakness of the lower limbs with parasthesia of 3 days duration. There was no history of sore throat or cough. The weakness progressed over 3 days and she was unable to walk steadily. She also had muscle pains all over.

On examination, she was of small build, mentally rather dull. The cranial nerves were intact and upper limbs were normal neurologically except for subjective parasthesia. In the lower limbs, the muscles were tender and the reflexes were absent. Power of proximal and distal muscles was grade 4. Vibration sense and pain to pin-prick were intact. Gait was unsteady. There was no definitive evidence of cerebellar dysfunction. Examination of other systems including the lungs was normal.

The next day, she complained of abdominal pain and had bladder distension requiring catheterisation. She was unable to walk and power in the lower limbs deteriorated to grade 3.

University Department of Medicine (II)
Singapore General Hospital, Singapore 3
G. Devathasan,
MBBS, M. Med, MRCP (UK)
Lecturer
H.L. Chan, MBBS, FRACP,
MRCP (UK), Dip. Derm. (Lond)
Associate Professor

Investigations showed a haemoglobin of 11.5g/dl. WBC count 8,000/cu mm, EST 29 mm fall first hour. Chest Xray revealed consolidation over the right lower zone (Fig. 1). On lumbar puncture, there was no rise in pressure. Cerebro-spinal fluid examination showed cells O, glucose 58 mg/100 ml, Cl 690 mg/100 ml, total proteins 20 mg%, globulins negative. Throat swab was negative for mycoplasma; Cold agglutinins was positive at 1/20. Complement fixation test for mycoplasma titres showed a four fold rise over a 4 week period — from 1/32 to 1/256. Psittacosis and Lymphogranuloma venereum titres were below 1/8. Repeat CSF was normal. Nerve conduction studies showed a slowing of conduction velocities of the nerves studied (median, ulnar, lateral popliteal, saphenous, posterior tibial and sural nerves). The amplitudes of all the sensory evoked potentials were markedly reduced.

She continued to have fever and was treated with ampicillin for 4 days without significant response. On the 7th day, she was started on tetracycline 500mg 6 hourly with dramatic response to the fever. Repeat chest X-ray was normal. Neurologically, she made a slow but complete recovery after 3 months.

DISCUSSION

Ever since mycoplasma pneumoniae infection was described as a distinct entity in 1938 and the agent isolated in 1944, there were many well proven cases with neurological complications. These include meningoencephalitis, psychosis, cranial nerve palsies, hemiparesis, transverse myelitis and acute polyradiculopathy.

The clinical picture of acute polyradiculopathy in mycoplasma penumoniae infection cannot be differentiated from the classical "Guillain Barre Syndrome" of obscure aetiology. The presenting features include parasthesia, weakness, areflexia, cranial nerves involvement, autonomic nervous system paralysis with hypertension or hypotension and respiratory muscles may be paralysed requiring mechanical ventilation (Lorer and Kalvasky, 1973; Hodges et al, 1972). In fact, based on these observations, all patients with acute neuropathy or polyradiculopathy of dubious cause should have blood screened for cold agglutinins and complement fixation test for mycoplasma pneumoniae.

The CSF changes are variable. It may be normal or show protein cell dissociation. If serosanguinous, it is accompanied usually by meningeoncephalitis but CSF glucose remains normal. A survey of 15 cases of acute polyradiculopathy due to mycoplasma pneumonia by Lorer et Kalvasky (1973) showed that

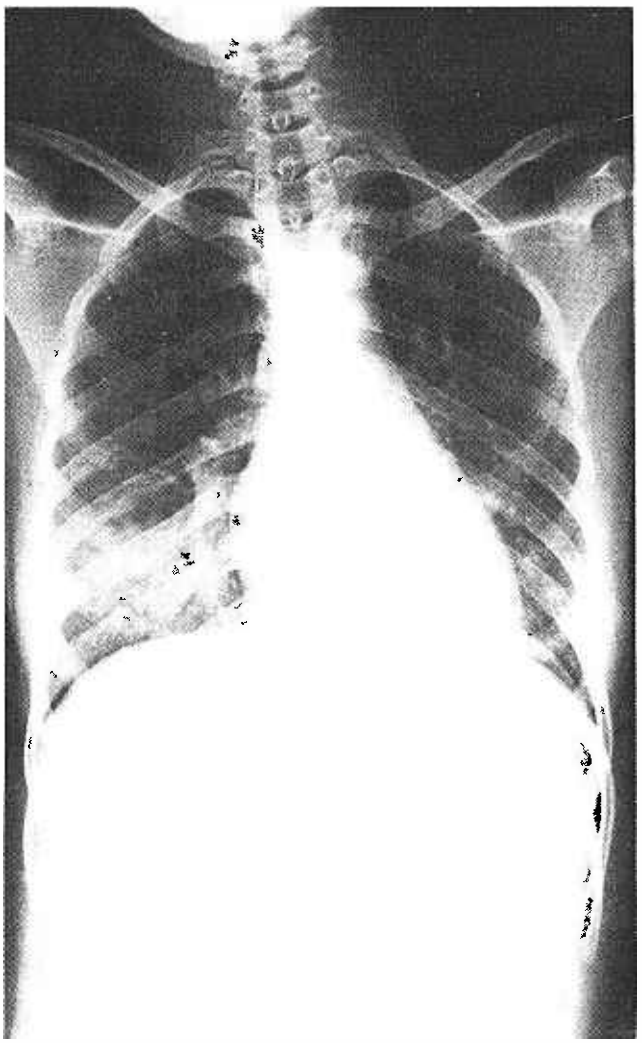

Fig. 1 CXR showing right lower zone consolidation

high CSF protein tend to be associated with severe neurological complications and residual damage, while normal or slightly elevated protein is associated with mild changes and complete recovery. This patient had a normal CSF, mild neurological involvement and complete recovery.

The aetiology is probably related to circulating antibodies rather than the organism invading the CNS since isolation of CSF is negative in most cases. Complement fixation antibodies against brain tissue have been identified by in vitro studies (Biberfeld, 1971) in patients with neurological complications as well as those with uncomplicated Mycoplasma pneumoniae infection. Immunoflourescent studies on biopsy specimens are lacking as the condition is rarely fatal. In this light, there is remarkable similarity with post-streptoccocal infection presenting with arthritis, carditis and chorea. Eradicating the source of infection may promote recovery from extra-pulmonary complications by removing the source for further antigenic stimulation of lymphocytes. It is unlikely that production of neurotoxins by the organism is the aetiology as this

cannot be demonstrated in experiments. Mycoplasma neurolyticum and gallisepticum which produce neurotoxins and invade animals are not pathogenic to man.

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