

Case report

Mycoplasma pneumoniae causing nervous system lesion and SIADH in the absence of pneumonia

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Received in revised form 12 July 2002; accepted 20 August 2002

Abstract

A patient was admitted for fever and acute respiratory failure (ARF), rapidly progressive tetraparesis, delirium, behavioral abnormalities, and diplopia. Leukocytosis and a rise in C-reactive protein were present. A syndrome of inappropriate anti-diuretic hormone secretion (SIADH) was also diagnosed. Lumbar puncture yielded colorless CFS with mononuclear pleocytosis and protein rise. Electrodiagnosis revealed demyelinating polyneuropathy and axonal degeneration. Serum IgG and IgM for *mycoplasma pneumoniae* (MP) was consistent with acute infection, and erythromycin was started with rapid resolution of symptoms. Contrarily to most reports, an associated respiratory disease was not present and SIADH in association with MP has been reported only once, in a patient without direct central nervous system (CNS) involvement. Differential diagnosis and possible pathogenic mechanisms are discussed.

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Keywords: *Mycoplasma pneumoniae*; Tetraparesis; Acute respiratory failure; Peripheral neuropathy; SIADH; Guillain–Barré Syndrome; Acute disseminated encephalomyelitis; Intensive care unit

1. Introduction

In most series, neurological complications have been diagnosed in 2–5% of patients with pneumonia due to *mycoplasma pneumoniae* (MP) [1–3], typically presenting within a few weeks of respiratory symptoms [3]. Long-term sequelae can occur in up to 20–30% of the cases [1]. Encephalitis was the most frequent manifestation in one series [4], and is the most reported in single case reports [5], but meningitis, brainstem syndromes, myelitis, cranial, and peripheral neuropathies, Guillain–Barré Syndrome (GBS), and acute disseminated encephalomyelitis (ADEM) are also possible [1]. Immunological and direct invasion are possible mechanisms of disease [1,3,5]. Only a few reports described neurological manifestations of MP in the absence of pneumonia [6,7], most in children. SIADH was once associated with MP pneumonia [8]. We report a patient with

neurological involvement and SIADH attributed to MP, in the absence of respiratory disease.

2. Case report

A previously healthy 30-year-old male was admitted after a 7-day history of fever, delirium, behavioral abnormalities, facial paresis, diplopia, weakness of the right upper limb, paraesthesia, and radicular pain of the left lower and the right upper limbs and urinary retention. The patient denied cough, expectoration, hemoptysis, dyspnea, nasal discharge, chest pain, anorexia, and weight loss. Haemogram, leukogram and C-reactive protein were normal, and serum sodium 132 meq/l. Lumbar puncture yielded colorless CFS under normal opening pressure, 30 cells (no predominance), protein 154 mg/100 ml and glucose 57 mg/100 ml (serum glucose 80 mg/100 ml); posteroanterior chest X-ray in the upright position was normal; brain CT scan and MRI were normal. The following day progression of tetraparesis and acute respiratory failure (ARF) led to mechanical ventilation. Vital signs were stable and axillar temperature 38 °C.

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Table 1

Evolution of serum *M. pneumoniae* serology, before and after erythromycin

Immunoglobulin	First serology	Second serology	Erythromycin	Third serology
IgM	0.479 (positive titres if >0.477)	0.518 (positive titres if >0.383)	–	0.466 (positive titres if >0.4)
IgG	0.384 (positive titres if >0.248)	0.571 (positive titres if >0.222)	–	1.1 (positive titres if >0.223)

Chest examination showed normal respiratory excursions, a resonant percussion, presence of normal breath sounds, absence of crackles or wheezing, and normal cardiac sounds. He was sedated but could localize pain only with his left arm; meningismus was absent. Pupillary and ocular fundi examinations were normal. A bilateral peripheral facial paresis was observed, and the examination of other cranial nerves was unremarkable. He had a flaccid tetraparesis mainly of lower limbs, both tendon and abdominal reflexes were weak and he had bilateral flexor plantar responses. Within 4 days he progressed to stupor, had spontaneous decerebrate posturing of left upper limb, right third, and bilateral sixth nerve paresis, paraplegia, absent tendon reflexes in lower limbs, and no plantar responses. White cell count was $17.700 \times 10^9/l$ (neutrophil count 84%); C-reactive protein was 19, both increasing in the following days; and serum sodium was 123 meq./l, decreasing further to 112 meq./l. Daily chest X-ray consistently showed normal findings. A second lumbar puncture yielded colorless CFS, 104 cells (predominance of lymphocytes), protein 116 mg/100 ml and glucose 65 mg/100 ml. Investigation of hyponatraemia disclosed an increased urinary sodium concentration (533 mmol/24 h) and low osmolality (233 mOsm/l), making the diagnosis of SIADH, which was successfully treated with fluid restriction, hypertonic solute administration, and furosemide.

First assumption was of an acute infection of central and peripheral nervous system. No growth of bacteria, virus or mycoplasma was detected in sputum, blood, urine, and CSF cultures, including mycobacteria. Serologic tests for *M. pneumoniae* antibody IgG and IgM were positive, with a gradual increase of IgG, consistent with ongoing infection (Table 1). Polymerase chain reaction (PCR) for MP in the CSF was not possible because of logistics. Cold agglutinins were not studied. Serodiagnosis for Lyme disease, syphilis, adenovirus, mumps, Q-fever, coxsackie A and B, measles viruses, epstein-barr, herpes simplex, varicella-zoster, cytomegalovirus, influenza A and B, and HIV 1 and 2 were negative. The diagnosis of GBS, ADEM, vasculitis, porphyria, arsenic, and lead intoxication were also considered, but specific work-up was negative, as well as auto-antibodies to gangliosides and galactocerebrosides. Nerve conduction studies and electromyography revealed absent F-responses, increased distal latencies, slowing of nerve conduction velocities, decreased amplitude of evoked motor responses, absence of sensory nerve potentials, in lower and upper limbs, and presence of fibrillation potentials (+3) at the abductor pollicis brevis, suggestive of demyelinating sensitive-motor polyneuropathy with axonal degeneration. EEG showed only minor abnormalities. A second MRI of

brain and spine revealed piamater and arachnoid enhancement surrounding conus medullaris and cauda equina, indicating a focal inflammatory lesion, probably due to spinal tap.

A diagnosis of probable acute infection of central and peripheral nervous system by MP was made, and erythromycin 1 g qid for 21 days was started, with rapid resolution of neurological symptoms, along with a decrease in leukocytosis and C-reactive protein. At discharge the patient examination disclosed a bilateral VII nerve palsy, a flaccid tetraparesis of grade 4/5 and distal paraesthesia of all limbs. At 3 months follow-up, the patient was able to walk with cane assistance, had a mild bilateral VII nerve palsy and paraesthesia of distal legs.

3. Discussion

The case here described is interesting because it was possible to document a widespread lesion of the central and peripheral nervous system by acute MP infection, as evidenced by serologic profile of MP antibodies and a clear temporal relationship between beginning of erythromycin and resolution of neurological symptoms. In our patient, the tetraparesis and ARF were responsible for admission in intensive care unit, making it a rare but important differential diagnosis for causes of rapidly progressing motor weakness and ARF. Contrarily to most reports, our patient did not have a respiratory infection and so an infection limited to nervous system was present. The absence of respiratory symptoms, the normal findings in chest examination and in daily chest X-ray consistently excluded a respiratory tract infection. To our knowledge, the association of SIADH and MP was only reported once [6], in a patient with pneumonia who developed SIADH and in whom seizures and stupor state were present during hyponatraemia, with no documented nervous system lesion. In our case, SIADH seems to be in direct relation with the meningoencephalitis.

Pathogenesis of neurological disease associated with MP is poorly understood but autoimmune response or direct invasion are commonly cited possibilities [1,3,5]. In this case we cannot draw a firm conclusion about pathogenesis since it was not possible to document the presence of MP in nervous system. Nevertheless, the mechanism of direct invasion would be the most likely based on the serologic profile of acute infection by MP, the simultaneous progression of neurological deficit and increase in infectious analytic parameters, and the presence of clinical and laboratory apparent benefit after erythromycin treatment.

M. pneumoniae may cause severe nervous system disorders and long-term neurological sequelae, making early diagnosis and treatment very important.

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