

Steroid Responsive Meningitis and Myelitis in Complicated *Mycoplasma pneumoniae* Infection

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Mycoplasma pneumoniae is a ubiquitous respiratory bacterium that causes community acquired pneumonia and other respiratory disease [1,2]. Neurological sequelae occur in up to 7% of patients with *M pneumoniae* infections and include meningoencephalitis, inflammatory neuropathies and transverse myelitis [3,4]. Different immune mechanisms may account for this heterogeneity. It appears that Mycoplasma Associated Meningitis (MAM) either occurs early, possibly due to bacterial invasion of the CNS or late secondary to a suspected post-infectious immune phenomenon [5]. We present a case of early *M pneumoniae* meningitis and myelitis that failed to show clinical improvement with directed antimicrobial therapy but responded to corticosteroids.

A 24 year old Aboriginal male presented to a rural hospital in New South Wales, Australia with 24 hours of fever (39.3°C), frontal headache, neck pain, vomiting and photophobia. The patient denied recent travel or intravenous drug use and was not immunosuppressed. He had an upper respiratory tract infection four days prior to presentation. Despite treatment with intravenous ceftriaxone (1gm daily) and oral doxycycline (100 mg daily) the high fevers and meningitic symptoms persisted. After five days, intravenous benzylpenicillin, acyclovir and vancomycin were added but without effect. On day nine, the patient became delirious and developed rapidly progressive lower limb weakness and urinary retention. He was transferred to a tertiary centre in Sydney where he was febrile (38.6°C) and disorientated but obeying commands. There was bilateral papilloedema, severe neck stiffness and lower limb flaccid paraparesis (MRC grade 3/5). There were no cranial nerve lesions.

Initial CSF examination (day 5) demonstrated a marked pleocytosis with 620 x10⁶/L white blood cells (WCC) (510x10⁶/L monocytes and 90x10⁶/L polymorphs), an elevated protein (3.57gm/L) and low glucose (2.4 mM/L). No organisms were identified by Gram stain or culture. Pre-antibiotic blood cultures were negative. A repeat CSF (day 10 and one day after steroids) showed 92 x10⁶/L WCC (5x10⁶/L monocytes and 87x10⁶/L polymorphs), a protein of 0.86 gm/L and glucose of 2.6 mmol/L. Investigations for Mycobacteria were negative including PCR and prolonged culture. Mycoplasma DNA was not found in either CSF specimen by PCR but serological analysis using ELISA showed significant elevation of *M pneumoniae* specific IgM on a second sample (optical density 0.64 (>0.48 abnormal)) returning to normal after a further 8 weeks (OD 0.14). Exhaustive CSF and serum investigations for fungi, viruses and bacteria were negative. Anti-neuronal antibodies and autoimmune screens were negative. NMO IgG was negative although non-specific IgG binding to myelin, axons and astrocyte processes was identified. MRI brain on two occasions was normal without evidence of meningeal enhancement. A patchy, long holocord lesion between T5 and T9 affecting the central grey matter with variable gadolinium enhancement was shown by spinal MRI.

Treatment with one gram of intravenous methylprednisone resulted in rapid improvement. The fever, headache and meningism resolved completely within 12 hours, disorientation within 24 hours and paraparesis over a period of 3 weeks with accompanying improvement in urinary function. A total of 5 days of methylprednisone (1gm/day) was given and a further 10 days of doxycycline. Four months later the patient was fully ambulant with no neurological symptoms.

M pneumoniae is a neuro-invasive organism that may be detected in the brain or CSF during meningoencephalitis or associated with post-infectious meningitis in which bacteria are not identified [5-7]. Our patient presented with symptoms, signs and laboratory findings consistent with bacterial or tuberculosis meningitis but despite antimicrobial therapy, including agents effective against *M pneumoniae*, his clinical condition deteriorated and only improved following the administration of intravenous steroids. This feature, in combination with clear evidence of concomitant longitudinally extensive transverse myelitis (a recognized post-infectious/autoimmune phenomenon) suggests that MAM was mediated by a para-infectious, inflammatory response rather than direct bacterial invasion of the CNS [8]. This was supported by serological evidence of *M pneumoniae* infection in the absence of mycoplasma DNA in the CSF. Diffuse IgG binding to multiple neural components may be consistent with known polyclonal B-cell activation by Mycoplasma species [9].

It is feasible that a different organism caused meningitis and serological cross-reactivity accounted for the positive *M pneumoniae* antibodies. However, blood cultures taken prior to the inception of antibiotics were negative and we would expect a quicker response to broad spectrum antibiotics in most cases of bacterial meningitis. Similarly, five days of doxycycline may have eradicated *M pneumoniae* from the CSF prior to PCR testing but would not account for the dramatic effect of steroids.

In cases of serologically proven *M pneumoniae* meningitis with negative CSF *M pneumoniae* PCR and where antimicrobial therapy appears ineffective, we recommend consideration of intravenous steroids as adjunctive treatment.

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