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Clinical Medicine Insights: Case Reports

A Case of *Mycoplasma Pneumoniae*-Associated Encephalomyelitis in a 16-Year-Old Female Presenting to an Adult Teaching Hospital

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ABSTRACT: We report a rare cause of encephalitis that is not often described in the adult clinical setting. Our case was a 16-year-old female who presented with a clinical picture of viral encephalitis; however, magnetic resonance imaging showed a demyelinating lesion of the left frontal lobe. In this age group, differential diagnoses of acute demyelination encephalomyelitis and multiple sclerosis were entertained. Further investigations demonstrated positive *Mycoplasma pneumoniae* serology. As a result, a diagnosis of *Mycoplasma pneumoniae*-associated encephalitis was made based on a process of exclusion.

KEYWORDS: Mycoplasma pneumonia, encephalitis, demyelination

CITATION: Tran et al. A Case of *Mycoplasma Pneumoniae*-Associated Encephalomyelitis in a 16-Year-Old Female Presenting to an Adult Teaching Hospital. *Clinical Medicine Insights: Case Reports* 2013:6 209–211 doi: 10.4137/CCRep.S13309.

TYPE: Case Report

FUNDING: Author(s) disclose no funding sources.

COMPETING INTERESTS: Author(s) disclose no potential conflicts of interest.

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Introduction

Neurologic complications from *Mycoplasma pneumoniae* have been known since the 1940s.[1] Encephalitis is the most common complication, and occurs most often in children. A review by Lerer and Kalavsky[2] found that 53% of patients with central nervous system (CNS) disease associated with *M. pneumoniae* were between the ages 6 years and 20 years. It is estimated that between 5% and 10% of acute childhood encephalitis in Europe and North America is attributable to *M. pneumoniae*.[3] There is a spectrum of clinical manifestations such as aseptic meningitis, peripheral neuropathy, transverse myelitis, cranial nerve palsies, cerebellar ataxia, acute transverse myelitis, and acute disseminated encephalomyelitis.[4] The incidence of these manifestations has been estimated to be between 0.06% and 0.1%,[5] but CNS symptoms are reported in up to 7% of patients in the hospital setting with confirmed *M. pneumoniae* infection.[6] Long-term neurological problems have been noted in 20%–60% of cases, with severe disease resulting in mental retardation, brain atrophy, hydrocephalus, epilepsy, visual changes, and global neurologic deficits with brainstem dysfunction and cerebellar ataxia.[7] Two studies have shown that between 24% and 30% of patients require intensive care treatment, with mortality rates of between 8% and 10%.[8,9]

Case Report

The index patient presented with a 2-week history of headaches, fevers, and dry cough. She then developed 3 days of diplopia, ataxia, and painful peripheral neuropathy. Her examination revealed an ataxic gait with normal muscle power, reflexes, and skin sensation. There was diplopia but no papilledema or other significant examination findings. A chest radiograph showed no abnormalities. Initial computed tomography imaging of her brain showed a single hypodense white matter lesion in the left frontal lobe. A subsequent magnetic resonance imaging scan (MRI) of the brain demonstrated a corresponding T2 and fluid-attenuated inversion recovery (FLAIR) hyperintense lesion in the left frontal lobe (Fig. 1). This raised the suspicion of acute demyelinating encephalomyelitis. Given the patient's age and symptoms, a diagnosis of multiple sclerosis was also considered.

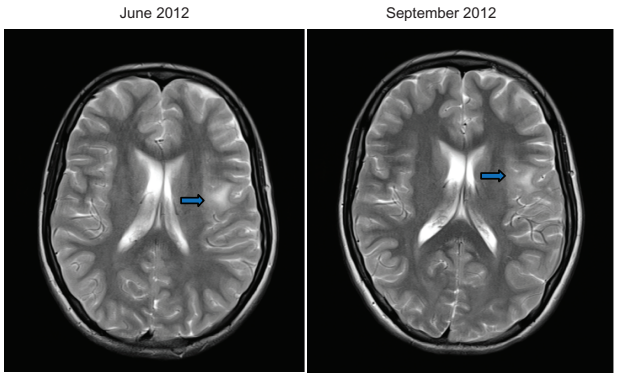

Figure 1. T2 axial images demonstrating the left frontal lobe demyelinating lesion.

Table 1 shows the results of the cerebrospinal fluid (CSF) analysis.

The patient was started on empirical intravenous acyclovir. Other investigations revealed a positive total *M. pneumoniae* serology immunoglobulin (Ig)M antibody titer of >1,280. The diagnosis of *M. pneumoniae*-related CNS disease was made based on the patient's clinical symptoms, positive serology, and the mononuclear pleocytosis found in the CSF. This is typically in the range of 10–200 cells/μL for *M. pneumoniae*-associated CNS infections.[10] Routine CSF gram stain and bacterial cultures are characteristically negative. One review of 58 cases showed a diagnosis was made in 84% of patients with positive serology, 14% with CSF polymerase chain reaction (PCR), and 2% with CSF antibody determination.[11] Our laboratory was able to evaluate the CSF for the open reading frame (ORF) 521 nuclear capsid protein gene with PCR, which returned a negative result.

Acyclovir was ceased 3 days into hospital admission, and the patient was prescribed a course of oral doxycycline. The patient's symptoms resolved over a period of 1 week of observation in hospital. Further analysis of the CSF with isoelectric focusing revealed oligoclonal bands of IgG in the CSF that was not present in the serum. The IgG index (CSF/serum) was 0.90 (<0.70). Two subsequent cranial MRI scans of the patient were obtained over a period of 3 months in the outpatient setting, and they showed the stable appearance of the left frontal lobe lesion. MRI of the spine did not show any demyelinating plaques. The patient had no recurrence of symptoms.

Discussion

The positive oligoclonal bands in the CSF are likely related to the immunological phenomena that are commonly linked to *M. pneumoniae* infections. It has been known since the 1970s that polyclonal B-cell activation and antibodies to brain tissue have been demonstrated in *M. pneumoniae* infections.[12] In 1996, Nishimura et al.[13] reported three cases of *M. pneumoniae* encephalitis with associated anti-galactocerebroside antibodies. In this patient, a moderately positive anti-ganglioside GQ1b antibody was found. Komatsu et al[14] had also reported this association in 1998. This supports the hypothesis that demyelination and neurologic dysfunction in this entity could be mediated by the immune system. The pathogenesis of *M. pneumoniae* CNS disease is unclear, but it may be related to direct invasion (proven during brain biopsy culture[15] or nucleic acid hybridization[16]) or autoimmunity (immune complexes, cross-reacting antibodies, or a neurotoxin effect).[17] This case highlights a rare cause of encephalitis in a patient presenting to a tertiary-level adult teaching hospital.

Table 1. Cerebrospinal fluid analysis on admission.

WBC	61×10^6/L (<5)
	Polymorphs 2%
	Mononuclear 98%
RBC	2×10^6/L (<5)
Glucose	3.0 mmol/L (2.2–3.9)
Protein	300 mg/L (150–500)
No organisms seen on gram stain	
M. Pneumonia DNA (NAA) Not detected	
India Ink: Negative	
Cryptococcal Ag (latex) Non-reactive	
HSV 1 DNA (NAA) Not detected	
HSV 2 DNA (NAA) Not detected	
Varicella Zoster DNA (NAA) Not detected	
CMV DNA (NAA) Not detected	
EBV DNA (NAA) Not detected	

Author Contributions

Wrote the first draft of the manuscript: HT. Contributed to the writing of the manuscript: HT. Agree with manuscript results and conclusions: HT, AA, CB. Made critical revisions and approved final version: HT. All authors reviewed and approved of the final manuscript.

DISCLOSURES AND ETHICS

As a requirement of publication the authors have provided signed confirmation of their compliance with ethical and legal obligations including but not limited to compliance with ICMJE authorship and competing interests guidelines, that the article is neither under consideration for publication nor published elsewhere, of their compliance with legal and ethical guidelines concerning human and animal research participants (if applicable), and that permission has been obtained for reproduction of any copyrighted material. This article was subject to blind, independent, expert peer review. The reviewers reported no competing interests.

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