Atypical Neurological Manifestations of Mycoplasma Pneumoniae: Isolated Abducens Nerve Palsy and Masseter Spasms in a Healthy 22-Year-Old

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INTRODUCTION

Mycoplasma pneumoniae (M. pneumoniae) is a bacterial pathogen commonly responsible for respiratory infections, such as atypical pneumonia, though it has also been associated with a range of extrapulmonary manifestations, including neurological complications. These manifestations, most commonly documented in children, can implicate both the central and peripheral nervous systems with findings of meningitis, encephalitis, ataxia, and nerve palsies, to name a few. The pathophysiology behind these neurological manifestations is not fully understood but is thought to involve both direct bacterial invasion and immune-mediated cross-reactivity between the pathogen and neural tissues.[1] Clinical findings often include an elevated white blood cell count with lymphocytic predominance in cerebrospinal fluid (CSF), increased protein, and normal glucose, which help distinguish M. pneumoniae-related meningitis from other bacterial infections. These infections can be successfully managed with central nervous system-penetrating antibiotics such as fluoroquinolones or tetracyclines. Recognizing M. pneumoniae as a potential agent of focal and nonfocal neurological symptoms is critical for preventing disease progression and long-term complications.

CASE PRESENTATION

A 22-year-old man with no significant medical history presented to the emergency department (ED) with double vision and difficulty speaking. He reported worsening speech over the past two days due to "jaw tightness," followed by the onset of binocular horizontal diplopia the day before presentation. Two weeks prior, the patient had returned to Rhode Island after vacationing in Florida. Soon after, he developed a cough and congestion, which resolved within a few days but was followed by bilateral ear pain, hearing loss, and posterior eye pressure. His primary care provider (PCP) diagnosed bilateral otitis media, for which he started a 10-day course of amoxicillin three days before his ED visit. Subsequently, he developed fever (to 101°F), nausea, vomiting, hyporexia, fatigue, headache, and neck pain. He then developed worsening but painless jaw tightness and double vision, prompting his visit to the ED.

On examination, the patient appeared lethargic and was mildly hypothermic (96.7°F/35.9°C), with a blood pressure of 118/53 mmHg, heart rate of 92 bpm, respiratory rate of 18 breaths per minute, and oxygen saturation of 98% on room air. Head and neck examination revealed visible bilateral masseter muscle spasms with restricted jaw movement. Neurologically, he was alert and oriented to person, place, time, and situation, and able to follow two-step commands; the only focal deficit was a medially deviated right eye with impaired abduction. He otherwise exhibited full strength, sensation to light touch, and 2+ reflexes in all extremities.

Initial labs showed leukocytosis (WBC 15.6) with 87% segmented neutrophils. A respiratory pathogen panel detected M. pneumoniae, and further testing revealed elevated M. pneumoniae IgM antibodies (3.09; normal 0–0.90). Lumbar puncture showed 66 and 64 nucleated cells per cubic millimeter in the first and last tubes, respectively, with 96% lymphocytes, normal glucose (57 mg/dL), elevated protein (81 mg/dL), and no oligoclonal bands. PCR testing for HSV I and II, along with a comprehensive meningoencephalitis panel, was negative (**Table 1**). Chest X-ray, CTA ELVO, and CT venogram of the head and neck were unremarkable, reducing concern for pneumonia, stroke, or cavernous sinus thrombosis. MRI revealed abnormal signal intensity of the CSF in the basal cisterns, consistent with abnormally proteinaceous CSF; FLAIR signal abnormalities in the bilateral frontal and parietal white matter and splenium of the corpus callosum were consistent with diffuse encephalitis.

Given the patient's positive M. pneumoniae serology, and a broadly negative meningoencephalitis panel, a diagnosis of M. pneumoniae meningoencephalitis was made. He was admitted for inpatient management, treated with CNS-penetrating levofloxacin and doxycycline in combination with prednisone, and made a full recovery over 14 days.

DISCUSSION

This case of Mycoplasma pneumoniae-associated meningitis in a 22-year-old highlights the diverse neurological complications that can arise from M. pneumoniae infection. The patient initially presented with mild respiratory symptoms, including cough, congestion, and otitis media, which is consistent with prior reports indicating that M. pneumoniae infections often start with mild respiratory prodromes before progressing to more severe extrapulmonary manifestations.[2,3] However, the progression to neurological

Table 1. Bloodwork and notable lab findings on admission

Component	Result
CBC	
WBC	15.6 (H)
Hemoglobin	13.7
Hematocrit	40.4
Platelets	286
BMP	
Glucose	102
BUN	12
Creatinine	1.03
Na+	141
K+	3.7
Cl–	109
HCO3–	19 (L)
Anion Gap	13
Ca2+	9.1
Respiratory Pathogen Panel	
SARS-CoV-2, Coronavirus, Adenovirus, C. Pneumoniae, Human Metapneumovirus, Human Rhinovirus/ Enterovirus, Influenza A, Influenza B, Parainfluenza Viruses 1-4, RSV A, RSV B	Not Detected
Mycoplasma pneumoniae	Detected
Infectious	
Mycoplasma pneumoniae IgM antibody	3.09
Treponema pallidum IgG IgM Ab	<0.2
CSF	
CSF Nuc Cell Last Tube/CCM	64 (H)
Lymphocytes CSF (Manual)%	96
Glucose, CSF	57
Protein, CSF	81 (H)
Oligoclonal Bands Number, CSF	Matching
Oligoclonal Bands, CSF	Negative
E. Coli K1, H. Influenzae, L. Monocytogenes, N. Meningitidis, S. Agalactiae, S. Pneumoniae, Cryptococcus Neoformans, Cytomegalovirus, Enterovirus, HSV-1, HSV-2, HHV-6, VZV, Eastern Equine Encephalitis Ab, Western Equine Encephalitis Ab, California Encephalitis Ab, St. Louis Encephalitis Ab	Not Detected

symptoms in this case – particularly abducens nerve palsy and jaw tightness – is noteworthy, as these symptoms are rarely reported in association with M. pneumoniae.[4]

Reported cases of M. pneumoniae-related nerve palsies have typically involved pediatric or older populations. In a similar case of abducens nerve palsy in a 69-year-old, that patient lacked preceding respiratory symptoms, which contrasts with our patient's initial respiratory illness before neurological symptoms emerged.[5] Additionally, our patient lacked skin findings or arthralgias and demonstrated concurrent signs of encephalitis, suggesting that M. pneumoniae infections may manifest in different sequences. Some cases may present with primarily neurological symptoms, while others follow a more typical respiratory prodrome. Moreover, the presence of masseter muscle spasms in our patient introduces a novel aspect to the clinical picture. This feature, not commonly reported in prior cases of M. pneumoniae infections, suggests a potential neuromuscular hyperexcitability associated with the pathogen, adding complexity to the understanding of M. pneumoniae-induced neurological manifestations.

The pathogenesis of M. pneumoniae-associated neurological complications remains multifaceted, involving both direct bacterial invasion and immune-mediated responses. In our case, the elevated CSF protein, normal glucose levels, and lymphocytic pleocytosis suggest the possibility of an inflammatory or immune-mediated process, rather than direct bacterial invasion,[almost all infections of the CNS produce a pleocytosis, whether immune related or not].[6,7] The MRI findings, including FLAIR abnormalities in the splenium of the corpus callosum, support the idea of an inflammatory process consistent with M. pneumoniae-associated encephalitis rather than a primary ischemic event.[8]

While vascular complications, such as stroke or aneurysms, have been associated with M. pneumoniae infections, this patient did not experience such events.[9,10] This may suggest that vascular inflammation in M. pneumoniae-associated neurological disease could present without overt cerebrovascular events. The absence of other meningoencephalitis pathogens on PCR testing strengthens the conclusion that M. pneumoniae was the primary causative agent.[11,12]

Treatment with CNS-penetrating antibiotics (levofloxacin and doxycycline) and prednisone resulted in rapid symptom resolution, supporting the efficacy of fluoroquinolone and steroid-based regimens for managing M. pneumoniae-related neurological complications.

In sum, this case emphasizes the importance of recognizing M. pneumoniae as a potential cause of neurological disease, even in the absence of severe respiratory symptoms. The patient's presentation with isolated abducens nerve palsy, masseter spasms, and encephalitis highlights the diverse and unpredictable manifestations of M. pneumoniae infections. This case reinforces prior reports on the pathogen's ability to induce both direct infection and immune-mediated neurological damage.

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Disclosures

The authors have no conflicts of interest to report.

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