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CASE REPORT

From the lung to brain: an atipical apresentation - case report

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Abstract

Mycoplasma pneumoniae is a common pathogen of respiratory infections in the pediatric age group, and can affect the central nervous system (CNS) in rare cases, presenting with manifestations such as encephalitis and meningoencephalitis. This report describes the case of an 11-month-old infant, previously healthy, who presented with clinical symptoms of fever, vomiting, and diarrhea. Subsequently, the infant developed severe neurological signs, including drowsiness, muscle weakness, and loss of developmental milestones. Three weeks prior, the child had respiratory symptoms suggestive of a viral infection. Analysis of the cerebrospinal fluid (CSF) showed lymphocytic pleocytosis and increased protein levels. Magnetic resonance imaging of the brain revealed cortical sulcus filling, associated with diffuse pachymeningeal and leptomeningeal thickening and enhancement, compatible with meningitis. The diagnosis was confirmed by serological tests, with the detection of IgM antibodies against *M. pneumoniae*. The diagnosis of meningoencephalitis caused by *M. pneumoniae* is challenging and often difficult, as the disease can present in an unspecific manner. This case highlights the importance of considering *M. pneumoniae* as a cause of neurological complications in children with a history of respiratory infection.

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INTRODUCTION

Mycoplasma pneumoniae is a common etiological agent of respiratory tract diseases in children, and it can affect the central nervous system (CNS) in up to 7% of hospitalized patients, causing encephalitis, meningoencephalitis, and acute disseminated encephalomyelitis.[1,2] However, the incidence of CNS complications and unfavorable outcomes is below 0.1%.[2]

Neurological involvement caused by M. pneumoniae is confirmed in most cases through serological tests, molecular testing, and imaging studies.[2,3]

This case report aims to describe the history of an infant who developed meningoencephalitis due to M. pneumoniae following clinical manifestations of respiratory infection, and to raise awareness about the possibility of severe complications associated with the infection.

DESCRIPTION

An 11-month-old female infant, previously healthy and with an up-to-date vaccination schedule, sought hospital care due to persistent and intermittent fever for approximately 12 days, associated with vomiting and diarrheal episodes. Ten days prior, she had developed drowsiness, hypoactivity, loss of muscle strength in both upper and lower limbs, difficulty walking, impaired balance, and regression of developmental milestones (stopped crawling and sitting without support). The mother reported that three weeks before symptom onset, the child had experienced a flu-like syndrome with fever, cough, and rhinorrhea.

Upon hospital admission, the patient was in fair general condition, afebrile (35.8°C), hypoactive, hyporesponsive, without meningeal signs, heart rate of 115 bpm, oxygen saturation of 98% on room air, and respiratory rate of 31 breaths/min. On neurological assessment, reflexes were globally preserved, muscle tone was decreased, and there was no spontaneous limb movement, along with poor trunk and neck support. The infant was referred to a semi-intensive care unit for monitoring, where she remained for 4 days without significant changes, and was then transferred to the pediatric ward for further diagnostic investigation.

Initial laboratory tests upon admission showed: hemoglobin 8.2 g/dL, hematocrit 25%, total leukocytes 10,200/mm³, bands 1%, segmented neutrophils 54%, eosinophils 6%, lymphocytes 30%, and platelets 242,000/mm³; C-reactive protein 16.31 mg/dL; normal electrolytes and transaminases. A head CT scan was performed before lumbar puncture and revealed signs of mild communicating hydrocephalus, with no evidence of intracranial hypertension. The first cerebrospinal fluid (CSF) analysis was compatible with lymphocytic meningitis (Table 1), with negative cultures for aerobic bacteria and fungi. Polymerase chain reaction (PCR) testing was requested for herpes simplex virus 1 and 2 and enterovirus/parechovirus, both undetectable; and the rapid test for Cryptococcus neoformans was nonreactive.

Based on the neurological abnormalities observed during the physical examination and the CSF results, the initial diagnostic hypothesis was meningoencephalitis of indeterminate etiology (viral or bacterial). Therefore, treatment with acyclovir and ceftriaxone was started. However, given the severity of the neurological symptoms, the possibility of Guillain-Barré syndrome (GBS) was also considered, and human immunoglobulin (IVIg) was administered. Nonetheless, there was no significant change in the clinical condition after treatment.

On the 7th day of hospitalization, the patient developed worsening respiratory patterns, prompting a new infectious evaluation. A second lumbar puncture was performed (Table 1), showing infectious worsening. In addition, the infant underwent a chest CT scan, which revealed multiple nodular parenchymal opacities in both lung fields, predominantly peripheral and basal, associated with peripheral ground-glass attenuation (Figure 1).

Due to the absence of improvement in neurological symptoms with the instituted therapy, a brain magnetic resonance imaging (MRI) scan was performed, which showed filling of the cortical sulci associated with pachymeningeal and leptomeningeal thickening and enhancement (Figure 2). Multiple subcentimetric nodular foci of post-contrast enhancement were also observed, randomly distributed, both juxtacortical and intraparenchymal, in the supra- and infratentorial compartments

Given the CSF results, the severity of the case, and the pulmonary involvement, antibiotic therapy was changed, and meropenem and linezolid were initiated.

On the 14th day of hospitalization, with persistent neurological symptoms, a neuroaxis MRI was performed, which showed multiple intramedullary lesions diffusely distributed (cervical, thoracic, and conus), with edema of the adjacent medullary tissues, associated with diffuse lepto- and pachymeningeal enhancement, as well as enhancement of the cauda equina roots along their course in the vertebral canal—findings consistent with meningitis, myelitis, and polyradiculitis.

After a detailed review of the infant's clinical course, due to the prolonged respiratory condition, radiological findings, and atypical neurological involvement, serology for M. pneumoniae was requested, and azithromycin and pulse therapy with methylprednisolone for 3 days were initiated, considering a possible immune and inflammatory involvement of the CNS.

Serum serology for M. pneumoniae was positive by chemiluminescence testing (IgM 27 UA/mL; reference value ≥ 10). A diagnosis of meningoencephalitis secondary to M. pneumoniae was established, and azithromycin was continued to complete antibiotic therapy, leading to progressive improvement of the neurological condition.

The patient was discharged after 21 days of hospitalization, with recovery of limb strength and trunk and neck support, and was referred for outpatient follow-up.

Table 1. Cerebrospinal fluid (CSF) results.

CSF Analysis	1st sample	2nd sample
Cell count	76/mm³	180/mm³
Polymorphonuclear cells	3%	6%
Lymphocytes	90%	92%
Proteins	185,8 mg/dL	305 mg/dL
Glucose	19 mg/dL	12 mg/dL

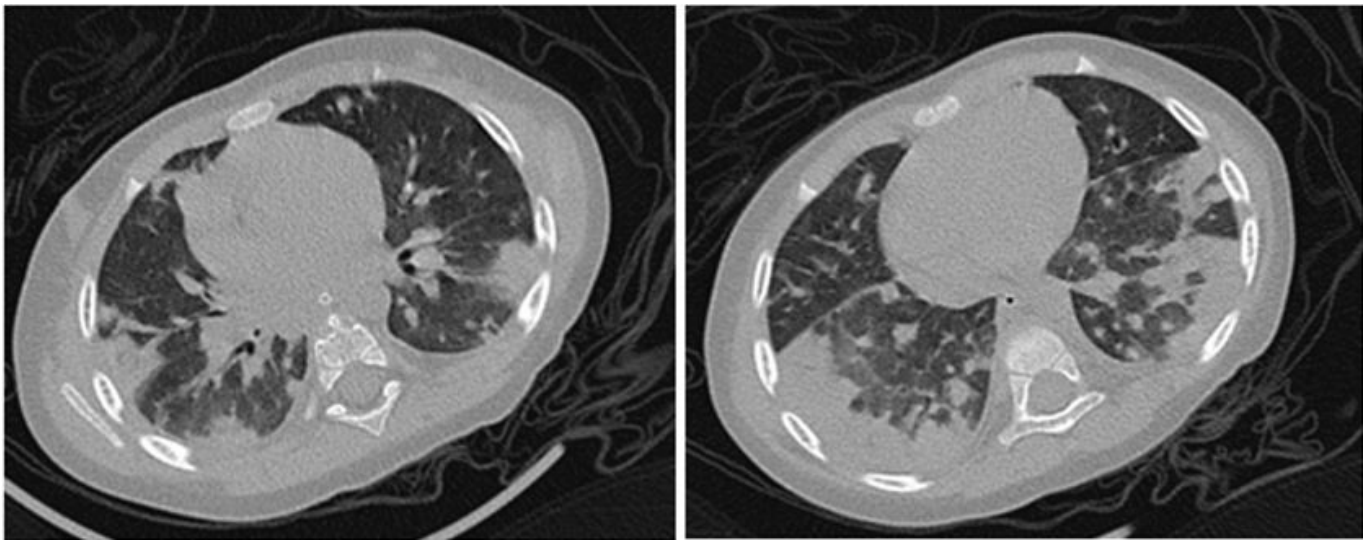


Figure 1. Chest CT scan showing multiple nodular parenchymal opacities in both lung fields.

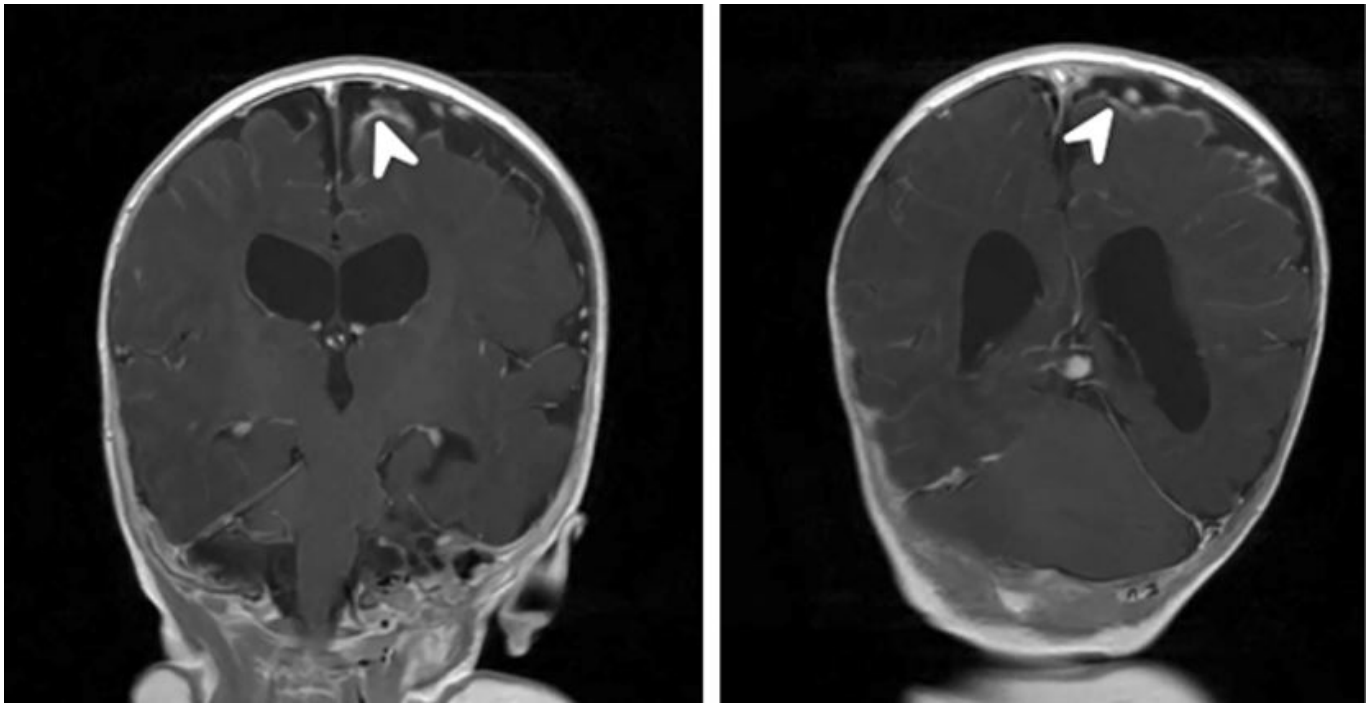


Figure 2. Coronal T1 post-contrast brain MRI. The arrows show pachymeningeal enhancement.

DISCUSSION

The clinical case described was compatible with acute meningoencephalitis, with M. pneumoniae as the etiological agent. In this context, the persistent respiratory symptoms that began about three weeks before the neurological condition and worsened during the clinical course were decisive for clinical suspicion.

Neurological involvement associated with M. pneumoniae infection, although rare, has been described in the literature for more than 40 years. It is important to note that, as in the case presented, respiratory symptoms precede neurological involvement in up to 67% of cases.[4] Patients with respiratory symptoms and a prodromal period longer than 7 days, as observed in this case, often exhibit an immunological response to M. pneumoniae, demonstrated by positive serum IgM levels, although this response is not always observed in the CSF.[2,5] Diagnostic challenges may therefore delay the initiation of appropriate treatment.

The pathophysiology of M. pneumoniae meningoencephalitis remains uncertain, but it is believed to result from an immune-mediated process or direct invasion of the pathogen into the central nervous system.[2]

In this context, based on CSF characteristics, other diagnostic hypotheses were excluded, including Guillain-Barré syndrome (GBS). GBS is characterized by albuminocytologic dissociation, observed as increased CSF protein without pleocytosis. In the present case, the CSF showed elevations in both leukocytes and protein. In addition, the patient did not show significant improvement after IVIg administration, making GBS less likely.

Regarding infectious etiologies, viral or bacterial origins are the most prevalent and have a high potential to trigger outbreaks.[6] Although the history and physical examination findings were not sufficient to exclude CNS infections, they aided in differentiating between meningitis and encephalitis caused by the most common pathogens. CSF characteristics also suggested atypical CNS involvement. The history of prior respiratory illness with worsening pneumonia was indicative of M. pneumoniae infection, likely contributing to both clinical presentations.

The diagnosis of meningoencephalitis due to M. pneumoniae is not commonly the first suspicion in children with neurological manifestations of infectious origin, which frequently leads to diagnostic and therapeutic delays.

Empirical treatment for meningoencephalitis was initiated, covering bacterial and viral agents, while awaiting culture results. In this case, the patient initially received ceftriaxone and acyclovir. However, no neurological improvement was observed. The respiratory worsening despite broad-spectrum antibiotics raised concern for atypical pneumonia caused by M. pneumoniae, prompting the addition of azithromycin.

MRI findings showing diffuse pachymeningeal and leptomeningeal enhancement led to the introduction of methylprednisolone, aimed at reducing inflammatory activity. Significant clinical improvement was observed within 48 hours of this combination. The suspected immune-mediated origin of the condition suggested that corticosteroid therapy could be beneficial.[2]

Fan et al. observed that in 87 pediatric patients treated with azithromycin combined with human immunoglobulin or corticosteroid therapy, as in the present case, hospitalization time was shorter and symptom control was better compared with the group treated with azithromycin alone.[1]

After one year of follow-up, the child showed full recovery of mobility and muscle tone, although delayed speech development persisted. According to Christie et al. (2007), neurological sequelae occur in 48–64% of cases.[7]

This case highlights the importance of considering the possibility of extrapulmonary manifestations of M. pneumoniae infection. In addition to neurological involvement, cases of autoimmune hemolytic anemia, thrombocytopenia, and acute kidney injury associated with this pathogen have been reported.[8,9]

In conclusion, CNS involvement associated with M. pneumoniae infection should be considered in patients with neurological symptoms during or after respiratory tract infection. The clinical response in this patient to methylprednisolone demonstrates the role of immunological mechanisms in the pathophysiology of the neurological complications caused by this pathogen. Corticosteroid therapy, together with macrolide treatment, should be considered a first-line approach for patients with severe neurological manifestations of M. pneumoniae infection.

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