Case report

Cerebellar syndrome with meningoencephalitis due to *Mycoplasma pneumoniae*

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Abstract

We report a 24-year-old woman with a *Mycoplasma* pneumonitis associated with subacute meningoencephalitis and acute cerebellitis that caused a cerebellar atrophy. Electroencephalogram showed diffuse dysfunction in the brain. There was few white blood cell but normal glucose and protein in the cerebrospinal fluid (CSF). Brain MRI showed bilateral atrophy of cerebellum.

Keywords: *Mycoplasma pneumoniae*; Cerebellar atrophy; Meningoencephalitis

Introduction

Neurological syndromes due to *Mycoplasma pneumoniae* infection are occasionally reported in adults, usually in the post-infection period [1]. Up to 7% of patients hospitalized with *M. pneumoniae* may have central nervous system (CNS) symptoms [2]. The cases suffered from demyelinating disorders of the central nervous system [1], middle cerebral artery thrombosis [1], myelitis [3-8], encephalopathy [9], acute disseminated encephalomyelitis [10], encephalitis [2,11], aseptic meningitis, polyradiculitis, cerebellar ataxia [2], acute cerebellitis and cerebellar atrophy [12] and hydrocephalus [13] due to *M. pneumoniae* infection.

Recent infection with *M. pneumoniae* was considered as a risk factor for cerebrovascular and CNS demyelinating disease [1]. We report a 24-year-old woman who developed early neurological complications associated with skin rashes before respiratory manifestations of *M. pneumoniae* occur.

Case history

A 24-year-old woman was admitted with fever, chills, scanning speech, nausea, vomiting and dizziness that had begun three days before her hospitalization. On physical examination, nothing much was found. She didn't complain of any respiratory symptoms. There were neck rigidity, abnormal cerebellar tests (ataxia, heel to shin test, wide base gate, and finger to nose) ,and she was hypotonic at the time of admission. Brain CT scan without contrast was normal. In cerebrospinal fluid (CSF), the number of WBC was eight cells (90% neutrophil, 10% lymphocyte), but the glucose and protein were normal.

The pruritic macula-papular rashes appeared on the second day after admission which distributed on the whole of the body (face, neck, chest, abdomen, and extremities). Repeated lumbar punctures showed similar results. On the fourth day after admission, the cough appeared and her chest X-ray showed the peribronchial infiltration. Electroencephalogram (EEG) reported a generalized slow background in the range of 6-7 cycles per second (CPS) that waves dominantly. A few degree of alpha activity in the range of eight CPS were seen in some occasion which was correlated with clinical diagnosis of diffuse cerebral dysfunction. Brain MRI with and without contrast showed bilateral atrophy of cerebellum.

Cold agglutinin tests quantitative and qualitative were positive with the titre of 1/128. Azithromycin was administered for 14 days; as a result, fever and diarrhea were ceased. Desquamation of rash was begun; the cerebellar tests changed to be normal and patient could talk fluently. Clinical recovery has been completed within six weeks.

Discussion

Neurological disorders associated with *M. pneumoniae* infection [1,10,14,15], which usually happens in post infectious period [1,9,14], and sometimes accompanied by active chest infection were reported [3, 16]. But in this case, symptoms and signs of *M. pneumonitis* appeared after cerebellitis. In her CSF, the number of cells increased and there were not changes in glucose and protein. This is compatible with Tokisawa *et al.* [9] and Socan *et al.* [14].

Acute cerebellitis, cerebellar atrophy, encephalopathy, acute disseminated encephalomyelitis, encephalitis, and meningoencephalitis associnated with *M. pneumoniae* infection have been reported by many investigators [2-3,9-13], but reports on *M. pneumoniae* induced subacute meningoencephalitis with acute cerebellitis are few. The mechanism of damage caused by *M. pneumoniae* remains unclear [2,4,17].

The isolation of *M. pneumoniae* from the CSF undoubtedly confirms the invasion to the CNS or, at least, to the CSF [7]. Direct invasion, neurotoxin production, or an immune-mediated mechanism has been proposed [2,6].

The present case was not evaluated to isolate *Mycoplasma* by PCR or culture and diagnosis was based on her serology test results. Serologic testing is still primarily used by clinicians in order to establish the diagnosis of *Mycoplasma* infection. However, serologic testing is not appropriate technique to detect the infection. This is because PCR confirmed the CNS infections in cases with negative serologic test results [18]. Presently, PCR for detection of *M. pneumoniae* sequences has been recommended for better detection especially in neurologic manifestations [19]. Clinical recovery occurred in our case within three to six weeks, which is compatible with Coleman *et al.* [13] and Socan *et al.* [14] experiences. *M. pneumoniae* infection should be considered in those patients with CNS manifestations of unknown cause, particularly if they appear in association with primary atypical pneumonias.

Conflict of interest statement: All authors declare that they have no conflict of interest.

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