World Journal of Clinical Case Reports and Case Series

Case Report

Fatal Acute Disseminated Encephalomyelitis after Infection: A Rare Case Report

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Abstract

Acute Disseminated Encephalomyelitis (ADEM) is an acute inflammatory demyelinating disease that extensively affects the white matter of the brain and spinal cord. It usually follows an infection, such as rash or vaccination. Brain Magnetic Resonance Imaging (MRI) show multiple scattered low signals on T1 and high signal lesions on T2 in the white matter of the brain and spinal cord. Here, we report a case, a 55-year-old man with progressive aggravation in lower limb weakness following infection. The imaging showed acute demyelination and spinal cord swelling.

Keywords: Acute disseminated encephalomyelitis; Infection; Spinal cord; Brain; Central nervous system

Introduction

Acute Disseminated Encephalomyelitis (ADEM) is a rare autoimmune demyelinating disease of the central nervous system. It is reported that ADEM is following various bacterial and viral infections and vaccination [1]. *Mycoplasma pneumoniae* is a common cause of Community-Acquired Pneumonia (CAP). In patients with *Mycoplasma pneumoniae* infection, the incidence of Central Nervous System (CNS) complications was up to 0.1% [2]. ADEM is rarely seen after *Mycoplasma pneumoniae* infection; therefore, we report a case of a patient with fatal ADEM.

Case Presentation

A 55-year-old male whose chief complaint was the weakness of both lower limbs worsened progressively for 13 days. According to family's description, we known a week before weakness in both lower limbs the patient had a medical history of fever, and general fatigue and sweating. The patient had weakness in both lower limbs and went to the basic hospital for treatment, but did not see significant improvement after treatment, then the weakness of both lower limbs worsened progressively and urine retention occurred. The possibility of myelitis was considered by local hospital.

The patient was admitted with a temperature of 37.5°C and a pulse of 89 beats per minute. Specialist physical examination showed that the patient was lethargic, abdominal wall reflex was not induced, and muscle strength of both lower limbs was 0. The patient's head Computed Tomography (CT) showed large symmetric low-density lesions in the lateral ventricles and central of hemioval (Figure 1). We were tentatively considering a diagnosis of myelitis. Lumbar puncture showed light red cerebrospinal fluid outflow, red blood cell counts of 5940X106/L, nucleated cell counts of 3X106/L. In order to distinguish from spinal vascular disease and puncture injury, lumbar puncture was performed again the next day, and there was still light red cerebrospinal fluid, red blood cell count was 4050X106/L, and nucleated cell count was 0. The antibody of autoimmune encephalitis 24 items, cerebrospinal fluid immunoidine and protein analysis were negative. The patient's head MRI indicated bilateral hemioval center, radiating crown area, basal ganglia area, lateral brain voiding-mass area, midbrain, pontine and pontine arm with slightly longer symmetrical T2 signals and equal T1 signals, FLAIR slightly higher signals, and DWI diffusion limited (Figure 2). MRI of the patient's cervical, thoracic, and lumbar vertebrae indicated enhanced T2 signal of brain stem and spinal cord swelling (Figure 3). The patient received nucleic acid resistance test results of Coronavirus Disease-19 (COVID-19), influenza A virus, influenza B virus was negative. All cultures including blood, urine and CSF were sterile. Nine respiratory examinations indicated mycoplasma infection. Began to use antibiotics, antivirals, mannitol and other supportive drugs for him. According to the results of lumbar puncture and imaging, spinal vascular disease cannot be excluded. After consultation with the Interventional department and Imaging department, spinal vascular disease was not considered for this time and acute demyelinating disease was considered at present. A high-dose methylprednisolone therapy at 500 mg/day was initiated. The general condition of the patient did not significantly improve. Terribly, the patient had autonomic nervous dysfunction, frequent changed in body temperature and heart rate, and excessive paroxysmal sweating above the umbilical plane. We recommended intravenous immunoglobulin, but the family refused to use it, then we transferred him to Intensive Care Unit (ICU) for further treatment. The patient developed central hyperthermia and dyspnea at night. Although appropriate rescue measures were taken as soon as possible, tracheotomy and ventilator support were performed, the disease continued to deteriorate due to the large lesion area and obvious spinal cord swelling, and finally died due to severe respiratory center involvement.

Discussion

Acute Disseminated Encephalomyelitis (ADEM) was an acute inflammatory immune-mediated demyelinating disease that extensively affected the white matter of the brain and spinal cord. ADEM usually appeared after a viral infection or vaccination [3], but bacterial infections caused by *mycoplasma*, *chlamydia*, *legionella*,

Citation: Wang L, Chen K, Zhao L, Lei C. Fatal Acute Disseminated Encephalomyelitis after Infection: A Rare Case Report. World J Clin Case Rep Case Ser. 2024;4(2):1026.

Publisher Name: Medtext Publications LLC

Manuscript compiled: Jun 13th, 2024

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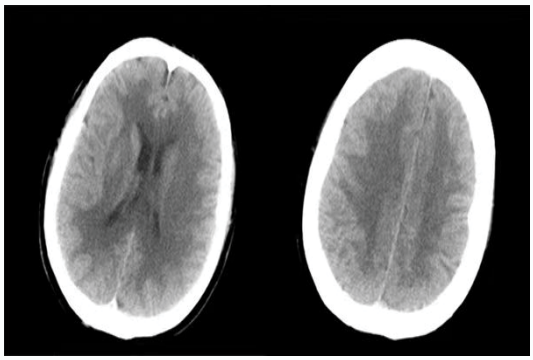

Figure 1: Computed tomography showing large symmetrical low-density foci in the lateral ventricle and center of the hemioval.

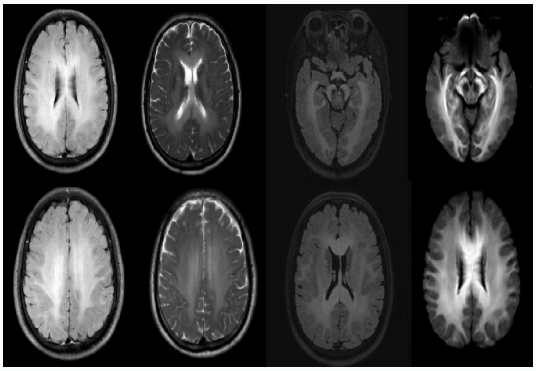

Figure 2: The MRI indicated bilateral hemioval center, radiating crown area, basal ganglia area, lateral brain voiding-mass area, midbrain, pontine and pontine arm with slightly longer symmetrical T2 signals and equal T1 signals, FLAIR slightly higher signals, and DWI diffusion limited.

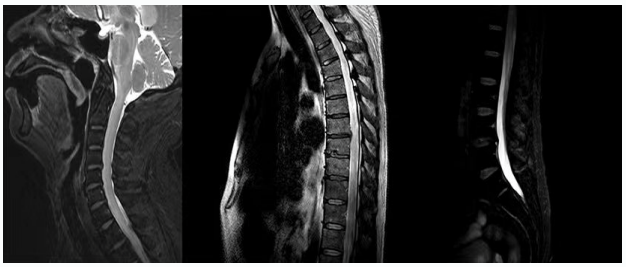

Figure 3: Magnetic resonance imaging showed increased T2 signal in diffuse swelling of brain stem and spinal cord.

campylobacter, and *streptococcus* occurring was rare. Patients usually had prodromal symptoms, such as fever, muscle pain, headache, or nausea and vomiting. There was usually an incubation period of 7 to 14 days between prodromal symptoms and the onset of neurological symptoms [4]. Late encephalopathy symptoms usually occurred 4-21 days after prodromal symptoms and included restlessness, drowsiness, hallucinations, confusion, and sensory changes. In addition, there were focal or multifocal neurological signs such as paraplegia, hemiplegia, and cerebral nerve palsy [1]. The mechanism of ADEM may be that the body was over activated the immune function after virus infection and vaccination, which lead to auto-immune reaction. Meanwhile some hidden antigens were released by the body, which misrecognized these antigens and resulted in immune attack against its own myelin sheath.

In this case, the patient was treated in a primary hospital two weeks before the onset of the disease, and the details of the conversion of prodromal symptoms to the nervous system were not known. However, after a week of respiratory symptoms, he did develop drowsiness, and paraplegia and other symptoms. According to the positive serological test results of *Mycoplasma pneumoniae* in the patient, the course of the patient's disease, and the symptoms of central nervous system infection and the radiological examination results, which fully proved that the infection was *Mycoplasma pneumoniae*?

Mycoplasma pneumoniae infection, as the primary infection, first affected the patient's respiratory tract, leading to acute influenza-like tracheobronchitis, followed by ADEM syndrome as the second stage of the disease. However, the pathogenesis of ADEM syndrome remains unclear [5]. At present, due to the lack of biomarkers for acute diffuse myelitis, its diagnosis requires imaging support. The white matter and gray matter may be impaired in patients with ADEM, which showed abnormally hyperintense on acute MRI T2-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) images. Lesions were usually bilateral, asymmetrical, larger than 2 cm, and poorly defined, which should be taken to distinguished it from demyelinating diseases such as multiple sclerosis. Currently, ADEM patients had a wide range of clinical features, whose diagnosis required careful exclusion of other diseases, and was a lack of biomarkers [6-8]. The histological features of ADEM were perivenous inflammatory infiltration and perivenous demyelination [9,10].

First-line treatment for ADEM usually included intravenous methylprednisolone, and intravenous immunoglobulin was often suitable for patients who had not responded to initial intravenous steroids. Plasmapheresis was usually the last resort treatment for fulminant disease ADEM [6,11]. The poor outcome of ADEM typically included poor functional outcomes, which was associated with spinal involvement, older age, and elevated markers of inflammation in the cerebrospinal fluid [8].

Conclusion

Acute disseminated encephalomyelitis caused by *Mycoplasma pneumoniae* was uncommon. At present, its treatment only relied on clinical experience, and lacked a lot of clinical data support. ADEM after *Mycoplasma pneumoniae* infection was rare and few patients die from it, so we would like to report this case. In summary, signs of encephalopathy and neurological symptoms followed pneumonia and patients with *Mycoplasma pneumoniae* infection and subsequent immune-mediated demyelination should be considered for diagnosis of the disease.

Funding

This study was supported by the Basic project of Yunnan Province (202101AT070142) and National Natural Science Foundation of China (82060230).

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