CASE REPORT

Acute Mycoplasma Pneumoniae Encephalitis in an Adult

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ABSTRACT

Mycoplasma pneumonia is an atypical bacterium that causes mild respiratory tract infections, especially in the upper respiratory system. Mycoplasma pneumoniae infection is infrequently associated with various CNS manifestations such as encephalitis, meningoencephalitis, myelitis, Guillain-Barre syndrome and acute disseminated encephalomyelitis (ADEM). Here we report a rare case of mycoplasma encephalitis in an adult who presented with a first episode of seizure following fever and neck stiffness for one week. Mycoplasma pneumoniae antibody titer was markedly elevated at >1:320 and MRI brain revealed encephalitic changes with a lesion in the splenium. Interestingly, there was no associated respiratory infection and his stay in the hospital was also complicated by SIADH. The patient improved after treatment with a macrolide antibiotic.
Malaysian Journal of Medicine and Health Sciences (2022) 18(5) 222-224. doi:10.47836/mjmhs18.5.31

Keywords: Mycoplasma pneumoniae, Encephalitis, Hyponatremia, Magnetic resonance imaging, Boomerang sign

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INTRODUCTION

Mycoplasma pneumonia is an atypical bacterium that causes mild respiratory tract infections especially in the upper respiratory system. It typically affects children and young adults, accounting for 40% of community-acquired pneumonia in this population. The incidence of mycoplasma pneumoniae declines with the advancement of age, less frequently affecting the adults (1). Rarely, mycoplasma pneumonia infection is associated with extrapulmonary manifestations, in which the involvement of the central nervous systems (CNS) is the most frequent. Here we report a case of mycoplasma encephalitis in an immunocompetent adult male.

CASE REPORT

A 16-year-old man presented to the emergency department with the first episode of a generalized tonic-clonic seizure. Before the episode, he had been experiencing fever associated with neck stiffness for a one-week duration.

Upon arrival, he was alert and orientated with a full GCS. Vitals examination was normal but he was febrile at 38.5C. Physical examinations including neurological examination were unremarkable apart from neck rigidity. Chest x-ray was clear. Initial plain CT brain was performed and concluded as normal. Blood investigations are unremarkable including white cell count which was normal at 6.8 x 10 9/L. Lumbar puncture which was done on the day of admission showed colorless fluid with normal opening pressure, protein 733 mg/100 ml and glucose 2.07 mmol/L (serum glucose 5.63 mmol/L). No cell count was detected. Other findings include negative India ink, AFB, cryptococcal antigen, viral and fungal culture. Viral screening of CMV, HSV1 and HSV2 DNA were negative. The anti-N-methyl-D-aspartate receptor was also sent and came back negative.

The patient was treated empirically with intravenous Ceftriaxone and Acyclovir while awaiting the results of investigations. Electroencephalogram (EEG) showed moderate generalized slowing of 5-6 c/s theta activity with no epileptiform changes observed consistent with diffuse encephalopathy. He remained well in the ward and became afebrile after the commencement of empirical treatment. However, on day 3, neurological examination revealed focal paresis of 4/5 grade on the right lower limb with hyperreflexia. We proceeded with MRI brain which revealed a hyperintense splenial lesion

on T2 weighted and FLAIR image (Fig 1A). The lesion was isointense on T1 weighted image without enhancement (Fig 1B). It also appeared to be a high signal on diffusion-weighted image (DWI), however, there was no low signal in the ADC sequence (no diffusion restriction). In addition, there was also a focal enhancement noted at the subcortical white matter at the left parietal lobe on T1 post-gadolinium sequence (Fig 1C) and normal on T2 weighted image (Fig 1D). No leptomeningeal enhancement was noted. Mycoplasma pneumoniae antibody titer was markedly elevated at >1: 320 (normal value <1:40), upon which his antibiotics were changed to azithromycin. However, PCR for mycoplasma DNA in the CSF was not sent due to logistic reasons. Despite the change of antibiotic, he suddenly became confused and agitated on the following day. His inflammatory markers were not raised but serum hyponatremia was low at 109 mmol/L. This was markedly reduced compared to his serum sodium level on admission which was normal (142 mmol/L). Syndrome of inappropriate antidiuretic hormone secretion (SIADH) was diagnosed based on the low serum osmolality and increased urine sodium concentration. He was managed with fluid restriction and hypertonic saline infusion after which the serum sodium normalized. The patient made an uneventful recovery after the completion of the antibiotic. His right lower limb weakness completely resolved and he developed no further seizure episodes. Upon review in the clinic 6 months later, the patient appeared well with no residual deficits.

DISCUSSION

Even though Mycoplasma pneumoniae infection can be associated with various CNS manifestations such as myelitis, Guillain-Barre syndrome and acute disseminated encephalomyelitis (ADEM), they are usually found in childhood population(2). Of these, encephalitis and meningitis are the most common. Patients may present acutely with meningism, high fever, seizures and reduced consciousness. If the infection involves the brainstem, clinical presentations may include ataxia or ophthalmoplegia. It is estimated that CNS manifestations are present in one out of 1,000 patients with Mycoplasma pneumoniae infections (3). Interestingly, there is no pulmonary involvement in this case. The patient predominantly presented with meningism, fever and seizures. He denied respiratory tract symptoms such as cough and the chest x-ray was clear. Even though there were a few case reports describing adult mycoplasma pneumoniae-encephalitis previously, CNS manifestations without the involvement of the respiratory system are indeed rare. This patient also developed focal paresis of the right leg which corresponds to the left parietal lobe lesion found on the MRI Brain consistent with the clinical suspicion of focal encephalitis. Motor weakness is found in 7% of reported cases of mycoplasma encephalitis, apart from other neurological manifestations such as abnormal speech, ataxia, delirium, abnormal behavior and peripheral nerve paralysis (4).

Furthermore, our patient also developed SIADH during his treatment in the hospital. To our knowledge, there was only one case report that described mycoplasma pneumoniae infection in adults causing encephalitis and SIADH in the absence of pulmonary lesion (5). In our case, the CNS manifestation is associated with a splenial lesion and encephalitic changes on MRI Brain. A splenial lesion can be found in various conditions such

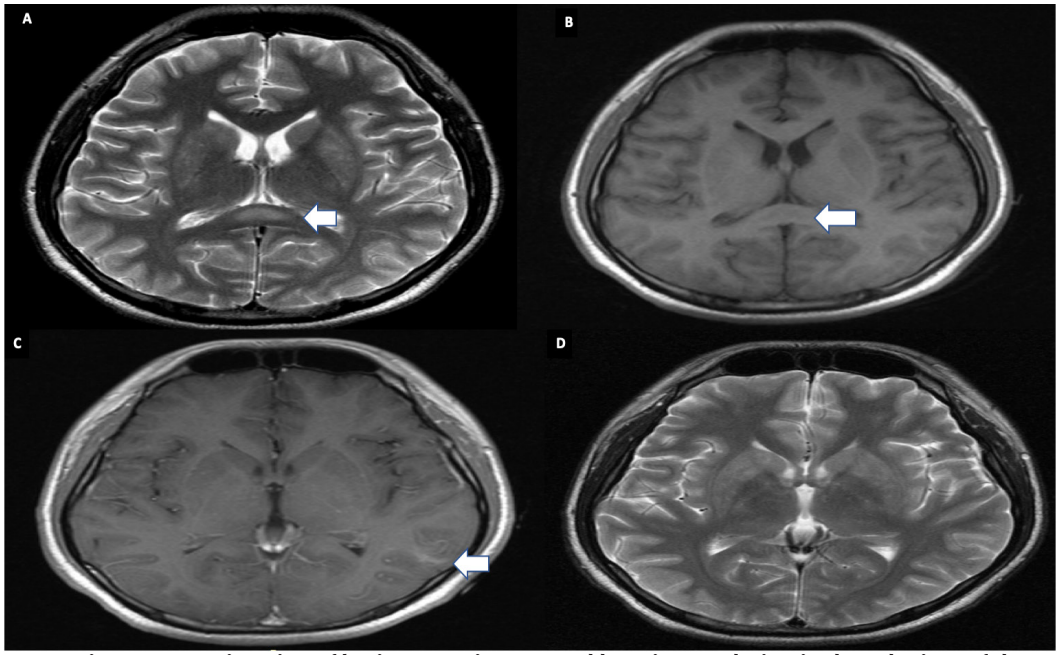

Figure 1: Magnetic resonance imaging of brain. Hyperintense and hypointense lesion in the splenium of the corpus callosum on T2-weighted image and T1 weighted images respectively (A, B). Focal enhancement noted at the subcortical white matter at the left parietal lobe noted on T1 post-gadolinium sequence but absent on T2-weighted image (C, D).

as anti-epileptic (AED) toxicity, post-ictal, demyelinating diseases, metabolic causes such as hypoglycemia and hyponatremia and post anoxic event (4). Our patient was prescribed sodium valproate on admission, however, the possibility of anti-epileptic toxicity was ruled out as his serum sodium valproate level was normal at 86.6 umol/L. The MRI scan was also performed on the third-day post his last seizure activity and hyponatremia was discovered after that. There were also no other supporting features to suggest demyelinating conditions such as multiple sclerosis and ADEM. Encephalitis may be the main cause of the hyperintensity of the splenium. The finding of focal encephalitis at the left parietal lobe also supports this. Mild encephalitis/encephalopathy with a reversible splenial lesion (MERS), a clinic-radiological syndrome had been previously reported in several case reports of Mycoplasma pneumoniae infection although it mainly pertains in paediatric cases. Unfortunately, we were not able to repeat the MRI Brain to confirm the resolution of the splenial lesion due to cost issue. Regardless, there is no clinical or radiologic signs that specifically point to mycoplasma infection of the CNS system.

The diagnosis of Mycoplasma pneumoniae infection is made by serology test. A fourfold or greater rise in titer is indicative of a recent infection (2). Unfortunately, the detection of Mycoplasma pneumoniae DNA by PCR technique in the CSF is low. This test is also not widely available, especially in limited-resource settings. Furthermore, both serology and PCR tests are limited in terms of sensitivity and specificity (3). Therefore, clinical suspicion with supportive neuroimaging findings is of utmost importance in establishing the diagnosis. Supplementary investigations include electroencephalogram (EEG) in which there were diffuse encephalopathy changes in this patient. Several case reports have previously reported similar findings (5). In children, Mycoplasma associated encephalopathy (MPAE) is associated with focal encephalopathies which may be detected using EEG.

CONCLUSION

Managing acute mycoplasma encephalitis can be challenging as it can be associated with other complications such as hyponatremia and encephalopathy. Treating practitioners are to be made aware of these complications to improve morbidity and reduce mortality.

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