CASE REPORT RHOMBENCEPHALITIS POSSIBLY CAUSED BY MYCOPLASMA PNEUMONIAE

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We report a rare case of encephalitis that is not often described in clinical settings in neurology. Our case was 11-year-old female patient who had presented with features of meningoencephalitis, but not responded to the conventional treatment. Her magnetic resonance imaging revealed lesions in thalami, cerebellum and brainstem. The differentials in this age were infective and inflammatory causes of meningoencephalitis and acute disseminated encephalomyelitis (ADEM). Paraneoplastic was another differential. Mycoplasma serology came out positive. As a result, diagnosis of mycoplasma pneumoiae associated Rhombencephalitis was made based on diagnosis of exclusion.

Keywords: Mycoplasma pneumoiae; Meningoencephalitis; Rhombencephalitis.

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INTRODUCTION

Mycoplasma pneumonaie is considered one of the frequent causative pathogens of respiratory tract infection in human being. The median age of Mycoplasma pneumonaie (M.pnumonaie) infection is 16 years and about 15% prevalence is reported in seasonal peaks in this age.¹ Apart from respiratory tract infection, central nervous system (CNS) is most frequent affected system during any phase of Mycoplasma infection.² Of various antibodies found, IgM is associated with neurologic disease.² About 10% of Mycoplasma pnuemoniae patients get admitted due to neurological complications and considered a leading cause of encephalitis in older children and adolescents³, however the pathophysiology of CNS involvement in unknown. Rhombencephalitis or brain stem encephalitis is a syndrome of multiple causes including infections, autoimmune and paraneoplastic. Mycoplasma is rare cause of encephalitis and reported prevalence is about 5%. We report a young girl who had atypical CNS involvement brainstem of and cerebellum (Rhombencephalitis) likely caused by M.pneumniae in the absence of respiratory system involvement.

CASE REPORT

An eleven-year-old young girl presented with one-week history of high-grade fever followed by seizures and decreased level of consciousness. There was no preceding history of travel abroad or recent vaccination. She had normal birth history, developmental milestones and complete vaccination history. On examination signs of meningeal irritation were positive and GCS was 11/15 (E4 V4 M3). Tone was increased in lower limbs with brisk reflexes and bilateral positive Babinski signs. Systemic examination was found unremarkable. At this point a working diagnosis of meningoencephalitis was made and was commenced on intravenous acyclovir, ceftriaxone, vancomycin, sodium valproate and fluids. LP was performed which showed WCC 8/mm³ (90% lymphocytes) protein 44 mg/dl, glucose 88mg/dl. MRI brain showed bilateral asymmetrical (abnormal signals) within thalami, midbrain along with focal altered signals within the cortical grey matter of cerebral hemisphere and cerebellum with prominent lateral ventricles.

Having completed 10 days of intravenous antibiotics and antivirals without any clinical improvement, she was referred to our neurology unit. At this stage she continued to have complex partial seizures and being encephalopathic. She had dystonic posturing of upper limbs. CSF was repeated with WCC 85 mm³ (lymphocytes 85%), protein 90 mg/dl and glucose 64 mg/dl. Serum glucose 90 mg/dl. CSF gram stain, AFB and geneXpert for TB were all negative.

Other normal or negative investigations included CBC, RFTs, LFTs, chest X-ray, covid screen, CSF HSV1, II, anti NMDA(N-methyl-D-aspartate) antibodies, HIV, hepatitis B and C. ESR was 25. In view of the MR brain findings of rhombencephalitis and CSF showing lymphocytic pleocytosis with normal glucose mycoplasma related rhombencephalitis was considered. Mycoplasma serology was sent and she was started on empirical Methylprednisolone injection for possible associated autoimmune phenomena. Her serum mycoplasma antibodies (IgM) came back positive. She was started on injectable macrolides (azithromycin). Patient started getting better over next 4-5 days. Her conscious level, seizures and dystonic posturing of the upper limbs steadily improved. Intravenous azithromycin was given for 10 days. IV methylprednisolone was given for 5 days followed by reducing dose of oral steroids over next 20 days. She was back to her normal self in 8 weeks' time.

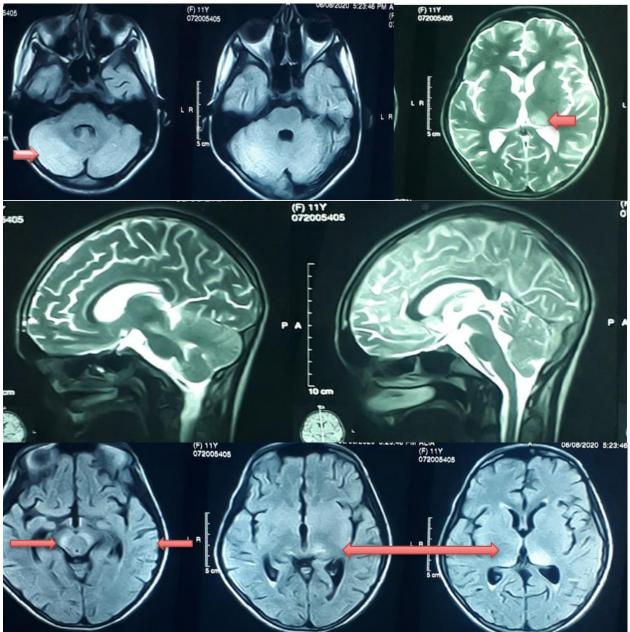


Figure-1: MRI brain demonstrating bilateral asymmetrical signals within thalami, midbrain along with focal altered signals within the cortical gray matter of cerebral hemisphere and cerebellum with prominent lateral ventricles

DISCUSSION

Mycoplasma pneumoniae is respiratory pathogen that is cell wall deficient and mainly infects the respiratory system. Its incubation period is 2–4 weeks and transmitted by respiratory droplets.⁴ *M.pneumoniae* is known to cause a wide variety of extra pulmonary diseases, including CNS, skin, heart, haematological system, gastrointestinal and musculosketal system.⁵ However CNS is considered most common site after respiratory system and up to 7% patients admitted with mycoplama infections develop CNS complications. *Mycoplasma pneumonia* cause different neurological manifestations including aseptic meningitis, encephalitis, polyradiculitis, myelitis, cerebellar ataxia, optic neuritis, Guillain Barre Syndrome, transverse myelitis and epilepsy.^{5–7} The mechanism of CNS involvement with mycoplasma infection remains unclear, however direct invasion, neurotoxin production and immune mediation are proposed mechanisms.⁸ In children encephalitis is

the most common presentation of mycoplasma infection and reported incidence is about 5 to 7%. In our case the primary symptoms were of encephalitis.

There are many causes of brain stem and cerebellum involvement, including inflammatory and infectious disease e-g multiple sclerosis, Lyme disease, Behcet disease, Bickerstaff encephalitis, Sarcoidosis, Listeria Rhombencephlites, tuberculosis etc but it is rarely related to mycoplasma infection.^{9,10}

The diagnosis of mycoplasma CNS infection can be difficult as the pathogen is rarely grown from CSF and is made indirectly through detection of IgM antibodies in serum and positive PCR from nasopharyngeal swab¹¹. The diagnosis is either confirmed or likely. The confirmed diagnosis of mycoplasma CNS infection is by PCR detection of the organism in CSF, which is reported only in 2–14% of the cases.¹² The likely diagnosis is supported by positive serological evidence, positive IgM in acute or IgG in later stages. MRI brain in which there will be enhanced signals more in white matter on T2 weighted images and brain stem changes However MRI brain might be normal in up to 49% of cases of encephalitis.¹³ Macrolides is the first line treatment for mycoplasma infection, but it may or may not be effective in CNS infection as macrolides lack the ability of blood brain barrier penetration.¹⁴ In cases where the immunological phenomena is suspected, corticosteroids and intravenous immunoglobulin (IVIGS) are used. Plasma exchange is used for transverse myelitis and polyradiculitis.

This case revealed favourable evaluation of mycoplasma rhombencephlites and patient responded very well to intravenous macrolides and steroids.^{9,15}

CONCLUSION

Encephalitis is very broad and potential lethal disease and many aetiologies remain unclear. Mycoplasma should be considered in children with encephalitis in children particularly in cases where MRI brain shows brain stem changes even in the absence of respiratory symptoms.

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