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Case Study

Acute Disseminated Encephalomyelitis in Childhood: A Rare Immune-Mediated Demyelinating Disorder

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ABSTRACT

Acute Disseminated Encephalomyelitis (ADEM) is an autoimmune disorder having demyelination in the brain and spinal cord due to inflammation in response to an infection or vaccination. It shows prevalence rate of 0.07-0.9/100,000 children/year. A 5 years old boy was admitted to the paediatric department with the complaints of fever for 9 days, generalized fatigue and malaise, diplopia, dehydration and loose stools. His blood report shows a positive Leptospira IgM and Epstein-Barr Virus (EBV) IgM. He was diagnosed with contrast Magnetic Resonance Imaging MRI-Brain, lumbar puncture which shows the features of ADEM. He was treated with Intravenous (IV) antibiotics, IV methyl prednisolone and other supportive treatments. This case highlights the importance of early recognition and diagnosis of ADEM and the usage of prompt steroid therapy to improve outcomes and prevent long term neurological sequelae..

INTRODUCTION

Acute Disseminated Encephalomyelitis (ADEM) is a rare, immune-mediated, inflammatory demyelinating disorder affecting the central nervous system (CNS). It is also known as post-infectious encephalomyelitis [1,10,11]. This condition is most frequently seen in children and young adults with a reported incidence of 0.07 to 0.9 per 100,000 children per year. It was also reported in peoples of age above 18 years [2]. The occurrence of the disease is spread over the colder months of a year and males are prone to ADEM than females.

It is often considered to be a post infectious disease of CNS. It is often triggered by viral or bacterial infections, and less commonly by certain vaccinations. Most associated organisms include cytomegalovirus, EBV, Herpes Simplex Virus, Human Herpes Virus, influenza Virus, Mycoplasma pneumonia, Leptospira etc [3]. It can also see both in children and adults with 8-21 days

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of immunization following are the vaccines that cause ADEM rabies vaccine (most common), measles, tetanus, diphtheria, small pox, hepatitis, human papillomavirus, influenza and poliomyelitis [4,5].

The exact mechanism of ADEM is not well defined but it is expected to be an inflammation of the brain that occurs as a result of vaccination or infection [6,12]. Due to the triggers of environmental stimulus, there may occur either a cell mediated response or due to the production of antibodies that cross-react with myelin auto antigens in the brain and spinal cord which cause the demyelination of myelin sheath which is the characteristic feature of ADEM [7,13]. It is also reported that it may occur due to increased vascular permeability and congestion in the CNS [8,9].

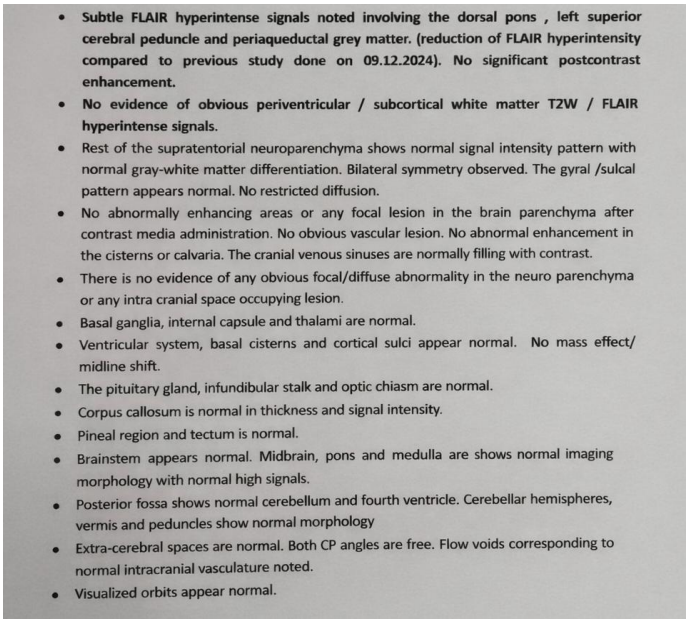
Most critical diagnostic tool used in ADEM are MRI- Brain and spinal cord, Cerebrospinal fluid (CSF) analysis, Blood tests that include inflammatory mediators like C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR), Complete Blood Count (CBC), Anti- MOG antibodies, Anti- ds DNA, Electroencephalogram (EEG), Infectious workup along with clinical manifestations. The first line treatment for ADEM includes high dose IV corticosteroids, IV immunoglobulins, plasmapheresis and other supportive care.

Its rapid onset and varied neurological manifestations can mimic other serious conditions. Making early recognition and prompt treatment is essential to prevent long-term deficits and helps to emphasize the importance of differentiating ADEM from other demyelinating and infectious encephalopathies.

CASE REPORT

A 5 years old boy was admitted to the paediatric department with the complaints of fever for 9 days, generalized fatigue and malaise, diplopia, dehydration and loose stools. On his physical examinations the neurological signs are reduced responsiveness, mild ataxia, bilateral adduction with nystagmus. Systemic findings show mild tachycardia and no signs of meningeal irritation.

Laboratory Findings: On CSF analysis (Lumbar Puncture) protein 46mg/dl (mildly elevated), glucose 70mg/dl(normal), total cell counts 50cells(elevated), polymorphs 25%(elevated) and lymphocytes 75%(normal). From the gene expert for Tuberculosis, it is negative for *mycobacterium tuberculosis*. From the blood investigations the *Leptospira* IgM and *Epstein-Barr Virus* (EBV) IgM positive and inflammatory marker ESR 23mm/hr (mildly elevated). In the neuroimaging (MRI Brain- Contrast Enhanced) [Figure 1] the findings were hypertensive lesions in the subcortical white matter, predominantly in the parietal and occipital lobes. No signs of tuberculosis meningitis or encephalitis. These all features consistent with ADEM.



- **Subtle FLAIR hyperintense signals noted involving the dorsal pons , left superior cerebral peduncle and periaqueductal grey matter. (reduction of FLAIR hyperintensity compared to previous study done on 09.12.2024). No significant postcontrast enhancement.**
- **No evidence of obvious periventricular / subcortical white matter T2W / FLAIR hyperintense signals.**
- Rest of the supratentorial neuroparenchyma shows normal signal intensity pattern with normal gray-white matter differentiation. Bilateral symmetry observed. The gyral /sulcal pattern appears normal. No restricted diffusion.
- No abnormally enhancing areas or any focal lesion in the brain parenchyma after contrast media administration. No obvious vascular lesion. No abnormal enhancement in the cisterns or calvaria. The cranial venous sinuses are normally filling with contrast.
- There is no evidence of any obvious focal/diffuse abnormality in the neuro parenchyma or any intra cranial space occupying lesion.
- Basal ganglia, internal capsule and thalami are normal.
- Ventricular system, basal cisterns and cortical sulci appear normal. No mass effect/ midline shift.
- The pituitary gland, infundibular stalk and optic chiasm are normal.
- Corpus callosum is normal in thickness and signal intensity.
- Pineal region and tectum is normal.
- Brainstem appears normal. Midbrain, pons and medulla are shows normal imaging morphology with normal high signals.
- Posterior fossa shows normal cerebellum and fourth ventricle. Cerebellar hemispheres, vermis and peduncles show normal morphology
- Extra-cerebral spaces are normal. Both CP angles are free. Flow voids corresponding to normal intracranial vasculature noted.
- Visualized orbits appear normal.

Figure 1 CE MRI BRAIN

Treatment course and Progress: Day 1-3 initiated high dose **IV METHYL PREDNISOLONE 450mg Once Daily** to reduce inflammation. **IV CEFTRIAXONE 750mg twice daily** given as empirical antibiotic and **CAPSULE DOXYCYCLINE 100mg twice daily** for the treatment of leptospirosis with other symptomatic treatment and supportive measures. Day 4-6 significant improvement in neurological function, fever and diplopia resolved but on eye examination horizontal eye movements restricted and vertical movements are normal. After the 7th day patient was stable and started oral tapering of corticosteroids and discharged with follow up instructions.

DISCUSSION

Acute Disseminated Encephalomyelitis (ADEM) is an immune-mediated demyelinating disorder of the central nervous system, predominantly affecting the pediatric population. It typically follows a viral or bacterial infection and, less commonly, vaccination. The pathogenesis is believed to involve an autoimmune response triggered by molecular mimicry, leading to inflammation and demyelination of the white matter of the brain and spinal cord.

In the present case, a 5-year-old child developed neurological symptoms following a febrile illness with serological evidence of **Leptospira IgM and Epstein–Barr virus (EBV) IgM** positivity, supporting the post-infectious etiology of ADEM. Several studies have reported EBV as a significant trigger for immune-mediated demyelinating disorders, including ADEM, particularly in children. Leptospira-associated neurological complications, though uncommon, have also been reported, making this case clinically notable due to the coexistence of both infectious triggers.

The clinical presentation of ADEM is highly variable and may include altered sensorium, ataxia, cranial nerve palsies, visual disturbances, and motor deficits. In this case, the patient presented with diplopia, ataxia, and reduced responsiveness, which are consistent with previously reported pediatric ADEM cases. Gastrointestinal symptoms such as loose stools and dehydration further complicated the clinical picture, potentially delaying neurological suspicion.

Magnetic Resonance Imaging (MRI) of the brain remains the cornerstone for diagnosis. The presence of multifocal hyperintense lesions in the subcortical white matter, particularly involving the parietal and occipital lobes, strongly supported the diagnosis of ADEM. Cerebrospinal fluid analysis revealed mild pleocytosis and elevated protein levels, which, although non-specific, further supported an inflammatory demyelinating process. The exclusion of tuberculosis and infectious encephalitis was crucial to avoid misdiagnosis and inappropriate management.

High-dose intravenous corticosteroids are considered the first-line treatment for ADEM due to their potent anti-inflammatory and immunosuppressive effects. In this case, early initiation of intravenous methylprednisolone resulted in significant neurological improvement within days, consistent with literature reports demonstrating favourable outcomes with prompt steroid therapy. Adjunctive antimicrobial therapy was appropriately administered to address the underlying infectious triggers.

This case emphasizes the importance of early diagnosis, timely neuroimaging, and prompt initiation of immunosuppressive therapy. Delayed recognition may result in persistent neurological deficits or progression to other demyelinating disorders, such as multiple sclerosis.

CLINICAL SIGNIFICANCE

This case highlights the clinical importance of considering Acute Disseminated Encephalomyelitis in pediatric patients presenting with acute neurological symptoms following an infectious illness. The coexistence of Leptospira and EBV infection underscores the role of multiple infectious triggers in precipitating immune-mediated demyelination.

Early differentiation of ADEM from infectious encephalitis, meningitis, and other demyelinating disorders is essential, as management strategies differ significantly. Prompt MRI evaluation and cerebrospinal fluid analysis are critical for accurate diagnosis. The favourable response to early corticosteroid therapy in this case reinforces the need for rapid intervention to prevent long-term neurological sequelae.

CONCLUSION

Acute Disseminated Encephalomyelitis is a rare but potentially serious immune-mediated demyelinating disorder of the central nervous system, predominantly affecting children. It commonly follows viral or bacterial infections and presents with acute-onset neurological deficits that can mimic other life-threatening conditions.

This case illustrates a classic presentation of post-infectious ADEM with dual infectious triggers and highlights the diagnostic value of MRI brain imaging and cerebrospinal fluid analysis. Early initiation of high-dose intravenous corticosteroids resulted in significant clinical improvement and prevented long-term neurological complications.

In conclusion, maintaining a high index of suspicion for ADEM in children presenting with acute neurological symptoms, early diagnostic workup, and prompt immunosuppressive therapy are crucial for favourable outcomes. Long-term follow-up is recommended to monitor recovery and exclude recurrence or progression to chronic demyelinating disorders.

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