JOURNAL OF CLINICAL AND BIOMEDICAL SCIENCES

CASE REPORT

OPEN ACCESS

Received: 17-06-2024
Accepted: 31-10-2025
Published: 21-04-2026

Citation: Karthik K, Karthik S, Krishnappa J, Reddy VRS, Srikanth C. A Rare Case Report of Atypical Presentation of Acute Disseminated Encephalomyelitis (ADEM). 2026; 16(1):84-86. https://doi.org/10.58739/jcbs/v16i1.24.51

* **Corresponding author.**
csrikanth5019@gmail.com

Funding: None

Competing Interests: None

Published By Sri Devaraj Urs Academy of Higher Education, Kolar, Karnataka

ISSN
Print: 2231-4180
Electronic: 2319-2453

A Rare Case Report of Atypical Presentation of Acute Disseminated Encephalomyelitis (ADEM)

Karthik K[1], Karthik S[2], Krishnappa J[3], Reddy V R S[4], Srikanth C[5*]

1 III rd Year Post Graduate, Department of Paediatrics, SDUMC, Kolar, Karnataka, India.
2 Assistant Professor, Department of Paediatrics, SDUMC, Kolar, Karnataka, India.
3 Professor, Department of Paediatrics, SDUMC, Kolar, Karnataka, India.
4 Professor and HOD, Department of Paediatrics, SDUMC, Kolar, Karnataka, India.
5 Associate Professor, Department of Paediatrics, SDUMC, Kolar, Karnataka, India.

Abstract

Acute Disseminated Encephalomyelitis (ADEM) is an inflammatory condition that causes demyelination in the central nervous system (CNS). It usually occurs in children and is characterized by multifocal brain lesions and encephalopathy. An 11-year-old male presented with a 15-day history of fever, pain abdomen for 1 week, neck pain with inability to walk for 3 days, and loss of bladder control for 1 day. Systemic examination done revealed hypotonia in the lower limbs with hyporeflexia. MRI brain with spine done revealed - scattered T1 iso-hypo and T2 hyperintensity involving spinal cord, suggestive of Acute Disseminated Encephalomyelitis. The child was started on supportive management and Intravenous Methyl Prednisolone. The child clinically improved, partially attained bladder control and was discharged with tapering doses of oral steroids. On follow up the child attained complete bladder control and was able to walk completely in the following 3 months. ADEM can manifest atypically with hypotonia in the lower limbs with loss of bladder control. Neuroimaging and a high index of suspicion are crucial for the diagnosis and treatment of these individuals.

Keywords: Encephalomyelitis, Neuroimaging, Methyl Prednisolone

1 Introduction

ADEM is an "inflammatory demyelinating illness of the central nervous system[1], usually manifesting in children and is linked to multifocal brain lesions and encephalopathy." The diagnostic evaluation includes neuroimaging to determine the extent of the CNS involvement, along with spinal and serum fluid tests to investigate viral and inflammatory etiology. Minor pleocytosis with increased protein may be seen in CSF analysis. Pediatric ADEM is one of a collection of conditions known as acquired demyelinating syndromes (ADSs)[2], which are defined by a sudden or gradual onset of neurological impairments associated with signs of inflammatory demyelination of CNS, including the optic nerves. Here we report an atypical presentation of ADEM in an 11-year-old child.

2 Case Report

An 11-year-old child presented with complaints of fever for 15 days, pain abdomen for 1 week, neck pain with inability to walk for 3 days and urinary incontinence for 1 day. Systemic examination done revealed hypotonia in lower limbs with

hyporeflexia and intact sensory system. MRI brain with spine done revealed - scattered T1 iso-hypo and T2/FLAIR white matter hyperintense areas noted in bilateral frontal, temporal and left parietal lobe , the spinal cord had T2 hyperintensity noted involving the spinal cord extending from C2 to D4 vertebral body level predominately involving central and posterior cord with mild cord swelling, suggestive of ADEM. The child was started on Intravenous Methyl Prednisolone, the urinary bladder was catheterized and other supportive management was provided. The child improved gradually over a period of 3 months with a tapering dose of oral steroids. The child attained complete bladder control and was able to walk again independently.

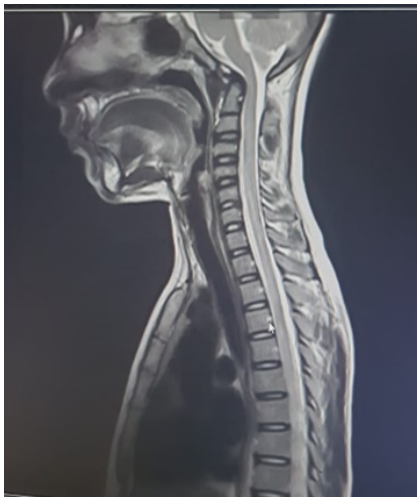

Fig. 1: MRI SPINE-Long segment, Linear, Intramedullary T2 hyperintensity noted in SC-C2 to D4 vertebral body with mild cord swelling

3 Discussion

ADEM is mainly diagnosed through clinical and radiological evaluation since there are no specific laboratory investigations to confirm the same. “The International Pediatric Multiple Sclerosis Study Group defines ADEM with the following criteria”[3]: a) “a polyfocal clinical CNS condition presumed to have an inflammatory demyelinating cause”, b) “exclusion of meningitis”, c) “no new clinical or MRI findings appearing 3 months or more after the onset”, and d) “an abnormal brain MRI during the acute phrase (3 months)”. Typically, clinical presentation and MRI lesions associated with ADEM will improve after short immunotherapy. Most of acute neurological deficit resolve by 3 months post-attack. Associated brain lesions show significant improvement or complete resolution on neuroimaging. To assess the progression of ADEM lesion, it is recommended for a follow-up MRI brain at 3–6 months and confirm the regression in a typical ADEM pattern.

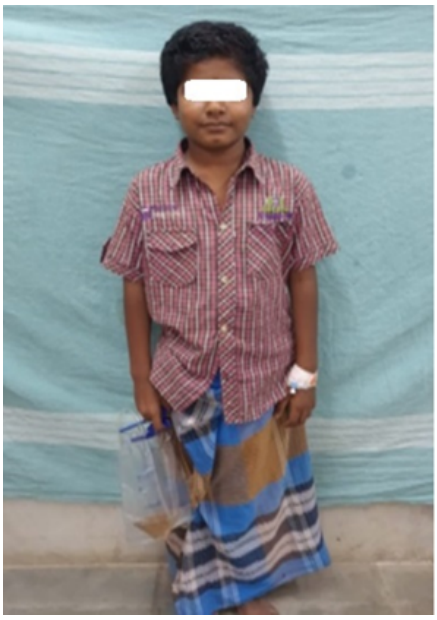

Fig. 2: Child with partial bladder control after 1 month of treatment

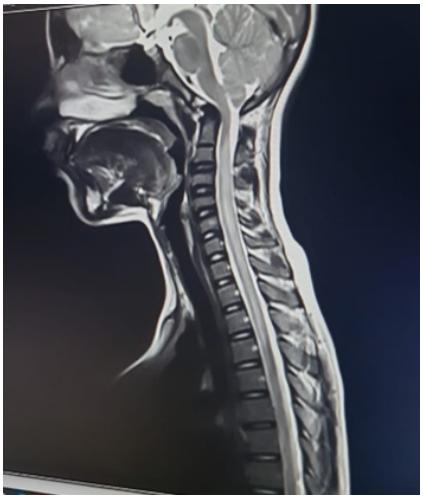

Fig. 3: Reduction in cord swelling as compared to previous MRI

To address any possible neurological or psychosocial effects of the ADEM, long-term follow-up is essential[1]. According to research conducted by Paolilo *et al.*[2], around 25% of children hospitalized with ADEM require admission to the intensive care unit. The mortality rate for ADEM has been reported to be between 1% and 3%. Despite variations in evaluation methods across different ADEM studies, the majority of patients (50-80%) achieve full recovery with a normal neurological examination.

Immunotherapy is regarded as a conventional treatment and may help patients recover more quickly and have better results than in the past, yet there isn't enough conclusive data to support this theory[4]. Children with ADEM typically recover completely, usually in 4-6 weeks.

According to research by Li *et al.*[5], the clinical presentation of ADEM varies based on the areas affected by demyelination. Majority of the patients showed a polyfocal clinical presentation with multifocal neurological deficits. Early symptoms included fever, headache, nausea and vomiting. Since fever is uncommon in other demyelinating diseases like MS, its presence can support an ADEM Diagnosis. Motor deficits such as hemiplegia, paraplegia, and quadriplegia, observed over three-fifths of patients are the most common clinical features, suggesting frequent involvement of pyramidal tract lesions. Ataxia is another common clinical feature, which is less frequent in Multiple Sclerosis.

Intravenous immunoglobulins, plasma exchange, and corticosteroids are the current therapy options for ADEM. The initial treatment involves administrating a high dosage of IV corticosteroids, specifically methylprednisolone, for three to five days at a dose of 20 to 30 mg/kg per day, with a maximum dose of 1 g per day. This should be followed by three to six weeks of oral prednisolone. If a patient does not respond to steroid therapy, other treatment options for ADEM include plasma exchange and IV immunoglobulin, administered at a dose of 2 grams/kg over 2-4 days. ADEM generally has an excellent long-term prognosis.

A case report by Balamurugesan *et al.*[6] on a rare occurrence of acute disseminated encephalomyelitis (ADEM) triggered by *Escherichia coli* infection, involved a 6-year-old girl who developed neurological symptoms following a urinary tract infection, ultimately leading to the diagnosis of ADEM through MRI findings. Hence ADEM can present atypically and should be kept as one of the differential diagnoses in children presenting with neurological symptoms.

4 Conclusion

ADEM can manifest atypically with lower-limb hypotonia or loss of bladder control. A high degree of suspicion and neuroimaging are essential for identifying and managing these individuals.

5 Acknowledgement

I would like to thank patient and his attenders for their cooperation. I would like to thank department of paediatrics for providing me this opportunity and constant support.

References

1. Wang CX. Assessment and Management of Acute Disseminated Encephalomyelitis (ADEM) in the Pediatric Patient. *Pediatric Drugs*. 2021;23(3):213-221. Available from: 10.1007/s40272-021-00441-7
2. Paolilo R, Deiva K, Neuteboom R, Rostásy K, Lim M. Acute Disseminated Encephalomyelitis: Current Perspectives. *Children*. 2020;7(11):210. Available from: 10.3390/children7110210
3. Krupp LB, Tardieu M, Amato MP, Banwell B, Chitnis T, Dale RC, *et al*. International Pediatric Multiple Sclerosis Study Group criteria for pediatric multiple sclerosis and immune-mediated central nervous system demyelinating disorders: revisions to the 2007 definitions. *Multiple Sclerosis Journal*. 2013;19(10):1261-1267. Available from: 10.1177/1352458513484547
4. Pohl D, Alper G, Haren K, Kornberg A, Claudia F. Acute disseminated encephalomyelitis. Updates on an inflammatory CNS syndrome. *Neurology*. 2016;87(9_Supplement_2):38-45. Available from: 10.1212/wnl.0000000000002825
5. Li K, Li M, Wen L, Wang Q, Ding X and Wang J. Clinical Presentation and Outcomes of Acute Disseminated Encephalomyelitis in Adults Worldwide: Systematic Review and Meta-Analysis. *Frontiers in Immunology*. 2022;13:1-10. Available from: 10.3389/fimmu.2022.870867
6. Balamurugesan K, Ponprabha R, Davis P. An interesting case of acute disseminated encephalomyelitis following E. coli infection. *Journal of Family Medicine and Primary Care*. 2019;8(1):292-295. Available from: 10.4103/jfmpc.jfmpc_402_18