ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 08 | August - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ADULT-ONSET ACUTE DISSEMINATED ENCEPHALOMYELITIS FOLLOWING FEBRILE ILLNESS: A CASE REPORT

Internal Medicine

Swaroop KS* Junior resident 3 Department of General Medicine, SBKS Medical College & Hospital, Vadodara, Gujarat, India*Corresponding Author

Aanal Patel Junior resident 2 Department of General Medicine, SBKS Medical College & Hospital, Vadodara, Gujarat, India

Tamanna Charpot Senior resident Department of General Medicine, SBKS Medical College & Hospital, Vadodara, Gujarat, India

ABSTRACT

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated demyelinating disorder that mostly affects children, with adult presentations remaining less common. We report a rare adult presentation of ADEM in a 20-year-old woman who presented with a five-day history of fever followed by rapidly progressive bilateral lower limb weakness, a generalised tonic-clonic seizure, and altered sensorium. Due to a significant drop in sensorium after the seizure, she was put on ventilatory support. MRI brain and spine showed multifocal T2/FLAIR hyper-intense lesions involving the cerebral hemispheres, basal ganglia, corpus callosum, periventricular white matter, brainstem, and cerebellum, along with longitudinally extensive spinal cord changes. CSF revealed mild lymphocytic pleocytosis with a negative infectious work-up. She improved dramatically after pulse therapy with methylprednisolone (1 g daily for five days), with significant clinical improvement noted at follow-up. This case underscores the importance of considering ADEM in adults presenting with severe post-febrile neurological deterioration and highlights the value of prompt immunotherapy.

KEYWORDS

Acute Disseminated Encephalomyelitis, Demyelination, Encephalopathy, Methylprednisolone

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated demyelinating disease that typically presents with multifocal neurological deficits and encephalopathy (Anilkumar & Foris, 2024). It mainly affects children, but adult cases are being recognised more often, and can be just as severe (Corrêa et al., 2024; Dy et al., 2025). In adults, ADEM is often hard to tell apart from other demyelinating disorders. Multiple Sclerosis (MS), Neuromyelitis Optica Spectrum Disorder (NMOSD), and Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) can all look similar on imaging and present with overlapping symptoms (Banwell et al., 2023). A few features pointed us toward ADEM: a single (monophasic) episode, significant encephalopathy at onset, bilateral deep grey matter lesions, and a quick steroid response (Anilkumar & Foris, 2024; Paolilo et al., 2020). The 2017 McDonald criteria define dissemination in space in MS as involvement of four characteristic areas — periventricular, juxtacortical/cortical, infratentorial, and spinal cord — along with dissemination in time (Thompson et al., 2018). Our patient's imaging did not fit this pattern, further favouring ADEM over MS.

Case Report

A 20-year-old woman presented with a five-day history of fever and rapidly progressive bilateral lower limb weakness. She then developed a generalised tonic-clonic seizure and altered sensorium. At the peripheral centre, GCS assessed was E2V1M4; she was intubated and put on ventilatory support before referral to our hospital. At admission, the patient was febrile, pulse rate was 160 beats per minute, blood pressure was 100/60 mmHg, and oxygen saturation was 98% on ventilator support. Neurological examination was limited by sedation but showed bilaterally reactive pupils. Motor examination revealed increased tone in the lower limbs. Deep tendon reflexes were exaggerated at bilateral knees with no ankle clonus and bilateral extensor plantars. Other systemic examination was unremarkable.

Initial laboratory investigations showed Hb 10.9 g/dl, TLC 40,000/mm³, platelets 300,000/mm³, ESR 25 mm/hr, and CRP 125 mg/L. Empirical antibiotics were started suspecting meningoencephalitis. CSF analysis showed 2 cells (100% lymphocytes) with normal biochemistry and negative CBNAAT for tuberculosis. Blood culture, CSF cultures, endotracheal tube culture, and urine cultures resulted in no growth. Contrast-enhanced MRI of the brain showed multifocal T2/FLAIR hyperintense lesions involving the cerebral hemispheres, deep grey matter, brainstem, cerebellum, while spinal MRI demonstrated longitudinally extensive T2 hyper-intensities. By day 5 of ICU admission, the leukocyte count had decreased and CRP had dropped to 11 mg/L; with infection less likely, ADEM was favoured over primary infection and other differential diagnoses. AQP4 and MOG antibodies were sent and later tested negative.

Treatment

The patient was initially managed with empirical antibiotics and supportive care for suspected meningoencephalitis. Following MRI findings suggestive of ADEM and after neurology consultation, she was started on pulse therapy with high-dose intravenous methylprednisolone (1 g daily for five days).

Outcome and Follow-Up

Her sensorium improved within two days of starting pulse steroids, and she was successfully extubated with return of full consciousness and orientation. Marked neurological improvement followed completion of the full five-day steroid course. Over the next two weeks, power in both lower limbs improved from 0/5 to 2/5 on the Medical Research Council (MRC) scale. She was discharged on day 15 on Tablet Omnacortil (prednisolone) 40 mg once daily, with advice for follow-up after two weeks.

At follow-up two weeks post-discharge, the patient reported significant improvement. She was able to walk with the support of a walking stick and had no new complaints. She was advised to continue physiotherapy and return for another review after two weeks.

DISCUSSION

The MRI findings — bilateral, asymmetric T2/FLAIR hyperintensities involving white and deep grey matter — supported ADEM (Almaghrabi et al., 2021; Koelman et al., 2019). CSF findings, as is typical in ADEM, were largely nonspecific: mild lymphocytic pleocytosis with a negative infectious workup (Aftab et al., 2024; Campbell et al., 2023).

MOGAD and NMOSD both needed ruling out, since either would have changed management and follow-up (Sechi et al., 2024; Wingerchuk et al., 2015). AQP4 and MOG antibodies were both negative, supporting ADEM over a relapsing, antibody-driven disorder such as NMOSD or MOGAD; even so, a single episode can occasionally turn out to be the first attack of relapsing-remitting MS, so repeated follow-up is needed to confirm the illness stays monophasic (Geraldes et al., 2024; Sechi et al., 2024). High-dose IV corticosteroids remain first-line for ADEM and led to good improvement here, consistent with the literature (Anilkumar & Foris, 2024; Corrêa et al., 2024).

CONCLUSION

This case highlights why ADEM should be considered in adults who develop acute encephalopathy with rapidly progressive paraplegia and altered sensorium needing ventilatory support to protect the airway, after a febrile illness. Early diagnosis and prompt high-dose steroid therapy led to significant recovery in our patient. Long-term follow-up remains important, both to watch for relapse and to confirm this monophasic presentation does not evolve into relapsing-remitting MS.

Learning Points

- ADEM should be considered in adults who develop severe encephalopathy with rapidly progressive paraplegia and altered sensorium needing ventilatory support to protect the airway.
- A monophasic course, prominent encephalopathy, asymmetrical involvement of areas in both white and grey matter with LETM of spinal cord, and a quick steroid response help distinguish ADEM from MS.
- AQP4 and MOG antibody testing should be routine in adult ADEM cases, since NMOSD and MOGAD have different prognoses and treatment approaches.
- Steroid-resistant cases may need IVIG or plasma exchange, and should raise suspicion for MOG-associated disease overlap.

Acknowledgements

The authors thank the Department of Neurology and the ICU team at SBKS Medical College & Hospital, Vadodara, for their support in the management of this patient.

Conflict of Interest

The authors declare no conflicts of interest.

Guarantor

Dr Swaroop KS is the guarantor of this article and accepts full responsibility for the integrity of the work, as a whole, from inception to published article.

Patient Consent

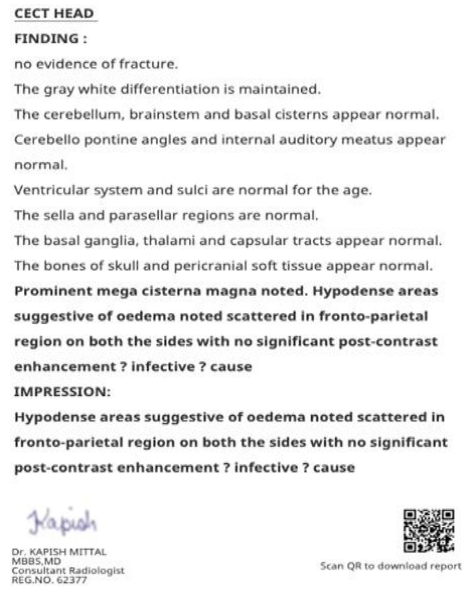
Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Ethical Approval

This case reort was conducted in accordance with the ethical principles of the Declaration of Helsinki (revised 2013). Institutional ethics committee waiver was obtained for publication of this case report.

Figure Legends

ADEM Case Report – High-Resolution Images & Figure Legends
Adult-Onset Acute Disseminated Encephalomyelitis Following Febrile IllnessSwaroop KS et al. | SBKS Medical College & Hospital, Vadodara



CECT HEAD

FINDING :

no evidence of fracture.

The gray white differentiation is maintained.

The cerebellum, brainstem and basal cisterns appear normal.

Cerebello pontine angles and internal auditory meatus appear normal.

Ventricular system and sulci are normal for the age.

The sella and parasellar regions are normal.

The basal ganglia, thalami and capsular tracts appear normal.

The bones of skull and pericranial soft tissue appear normal.

Prominent mega cisterna magna noted. Hypodense areas suggestive of oedema noted scattered in fronto-parietal region on both the sides with no significant post-contrast enhancement ? infective ? cause

IMPRESSION:

Hypodense areas suggestive of oedema noted scattered in fronto-parietal region on both the sides with no significant post-contrast enhancement ? infective ? cause

Kapish

Dr. KAPISH MITTAL
MBBS,MD
Consultant Radiologist
REG.NO. 62377

Scan QR to download report

Figure 1

[Table/Fig 1]: Non-contrast CT brain (CECT Head report) showing bilateral frontoparietal hypodensities suggestve of cerebral oedema.

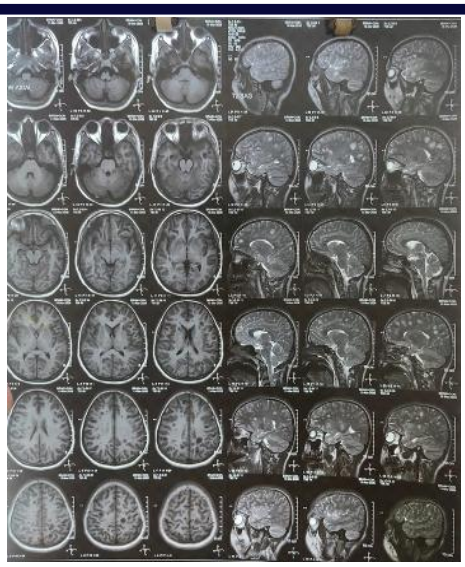


Figure 2

[Table/Fig 2]: MRI brain (plain) – multiple spherical / multifocal T2/FLAIR hyperintense lesions involving bilateral cerebral hemispheres, basal ganglia, corpus callosum, periventricular white matter, brainstem and cerebellum.

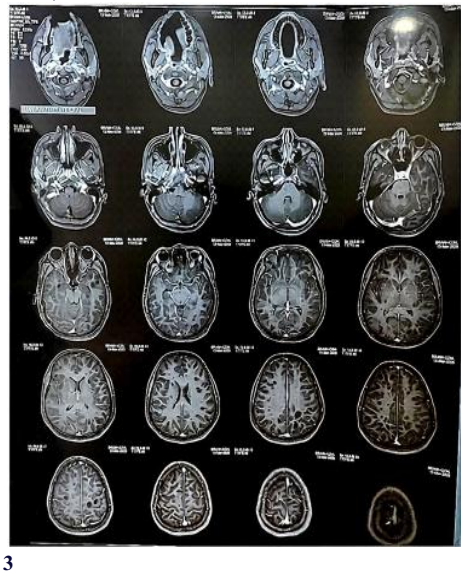


Figure 3

[Table/Fig 3]: MRI brain (contrast) showing subtle enhancement of demyelinating lesions.

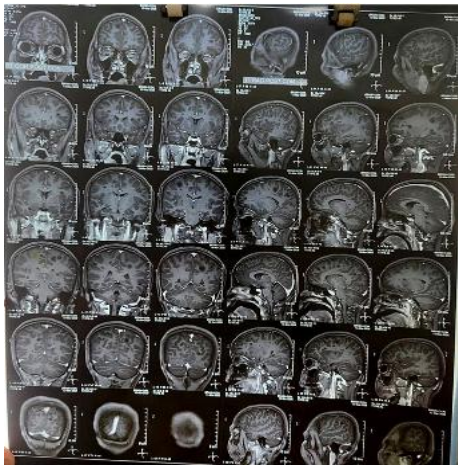


Figure 4

[Table/Fig 4]: MRI brain (post-contrast) showing multifocal enhancing lesions consistent with acute disseminated encephalomyelitis.

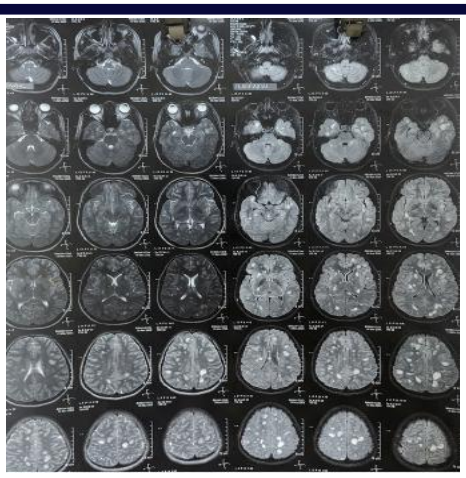


Figure 5
[Table/Fig 5]: MRI brain (FLAIR) demonstrating multifocal hyperintense lesions involving bilateral cerebral hemispheres, deep grey matter, and brainstem.

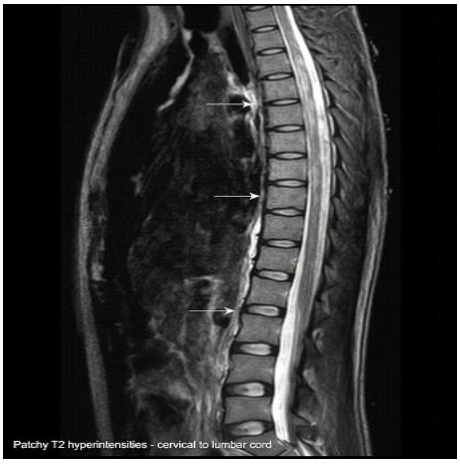


Figure 6
[Table/Fig 6]: Sagittal T2-weighted MRI of the spine demonstrating patchy T2 hyperintensities (white arrows) extending from the cervical to the lumbar spinal cord (Longitudinally Extensive Transverse Myelitis – LETM).

NON FASTING Report Status Final

SPL TEST

Specimen :	**Coll.**		
SERUM SPL	22/03/2026 11:00	Received at Lab	
Test Parameter		**Result(s)**	**Biological Reference Interval (Adult)**
Antibody against aquaporin-4, Serum			
Specimen		**Serum**	
Antibodies against Aquaporin-4 (AQP-4)			
Antibody against aquaporin-4 (AQP-4)		**Not Detected**	Not Detected (Indirect Immunofluorescence)
Test note		.	

Substrate : Aquaporin-4 transfected cells, EU 90
Dilution: 1:10 for serum, 1:1 for CSF

This is an indirect Immunofluorescence test.
This is a qualitative test to detect human antibodies of IgG type against aquaporin-4
Detection of this antibody supports the diagnosis of demyelinating diseases of CNS
The detection of this antibody is specific for NMO, Neuromyelitis Optica (Specificity: 91 - 100 %)
They can be detected in 60 - 80 % of patients who fulfil the clinical diagnostic criteria for NMO.

Associated test - Anti MOG antibody

Figure 7
[Table/Fig 7]: Aquaporin-4 (AQP4) antibody report – Not Detected.

NON FASTING Report Status Final

SPL TEST

Specimen :	**Coll.**		
SERUM SPL	22/03/2026 11:00	Received at Lab	
Test Parameter		**Result(s)**	**Biological Reference Interval (Adult)**
*** Antibodies against MOG**			
***Antibodies against myelin-oligodendrocyte glycoprotein (MOG)**			
*Specimen		**Serum**	
*Antibodies against MOG		**Not Detected**	Not Detected (Indirect Immunofluorescence)
*Test note		.	

Substrate : Myelin-oligodendrocyte glycoprotein (MOG) transfected cells, EU 90
Dilution: 1:10 for serum, 1:1 for CSF

This is an indirect Immunofluorescence test.
This is a qualitative test to detect human antibodies of IgG type against MOG
Detection of MOG antibody (in addition to AQP-4) supports the diagnosis of demyelinating diseases of CNS
Paediatric patients mostly show MOG antibodies
Presence of MOG antibody (along with AQP-4) enables detection of neuromyelitis optica spectrum disorders (NMOSD)
An important differential diagnoses, such as multiple sclerosis
MOG antibodies are seen in 5-8 % cases of NMOSD, but prevalence rises in AQP-4 negative patients

Associated test - Anti AQP-4 antibody, (NMO)

Figure 8
[Table/Fig 8]: Myelin oligodendrocyte glycoprotein (MOG) antibody report – Not Detected.

REFERENCES

1. Anilkumar, A. C., & Foris, L. A. (2024). Acute disseminated encephalomyelitis. In StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
2. Corrêa, J. R., Silva, A. P., Coelho, J., Gonçalves, R., & Estevão, D. (2024). Adult onset acutedisseminated encephalomyelitis: a case report. Cureus, 16(10), e72487.
3. Dy, J. S. H., Ng, A. R., & Pilotin, R. (2025). Adult-onset acute disseminated encephalomyelitis in elderly Filipino patients: a case report and review of the literature. Cureus, 17(9), e92138.
4. Banwell, B., Bennett, J. L., Marignier, R., Kim, H. J., Brilot, F., Flanagan, E. P., et al. (2023). Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: international MOGAD Panel proposed criteria. Lancet Neurology, 22(3), 268–282.
5. Paolilo, R. B., Deiva, K., Neuteboom, R., Rostasy, K., & Lim, M. (2020). Acute disseminated encephalomyelitis: current perspectives. Children (Basel), 7(11), 210.
6. Thompson, A. J., Banwell, B. L., Barkhof, F., Carroll, W. M., Coetzee, T., Comi, G., et al. (2018). Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. Lancet Neurology, 17(2), 162–173.
7. Almaghrabi, N., Albeshri, M., Alyahya, M., & Alghamdi, S. (2021). Adult onset acute disseminated encephalomyelitis. Radiology Case Reports, 16(8), 2205–2209.
8. Koelman, D. L. H., Chahin, S., Mar, S. S., Venkatesan, A., Hoganson, G. M., Yeshokumar, A. K., et al. (2019). Acute disseminated encephalomyelitis in adults: characteristics, diagnosis and treatment. Current Opinion in Neurology, 32(3), 385–392.
9. Aftab, H., Ahmed, R., Tariq, T., Shafiq, S., Khan, M. H., & Bashir, M. A. (2024). Clinical pattern, neuroimaging findings and outcome of acute disseminated encephalomyelitis in children: a retrospective study. Pakistan Journal of Medical Sciences, 40(7), 1479–1484.
10. Campbell, D., Wong, G. S., Park, H., & McLeod, G. (2023). An adult case of adenovirus-associated acute disseminated encephalomyelitis. Case Reports in Neurological Medicine, 2023, 5528198.
11. Sechi, E., Cacciaguerra, L., Chen, J. J., Mariotto, S., Bannoud, N., Dinoto, A., et al. (2024). Updates in NMOSD and MOGAD diagnosis and treatment: a tale of two CNS autoimmune inflammatory disorders. Neurologic Clinics, 42(1), 77–114.
12. Wingerchuk, D. M., Banwell, B., Bennett, J. L., Cabre, P., Carroll, W., Chitnis, T., et al. (2015). International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology, 85(2), 177–189.
13. Geraldes, R., Bukhari, W., Brex, P. A., & Brownlee, W. (2024). Neuromyelitis optica spectrum disorders and myelin oligodendrocyte glycoprotein antibody-associated disease. Continuum (Minneapolis, Minn.), 30(4), 1052–1087.
14. Najjar, A. A., Abu Mayaleh, M. A., Halaykh, R. M., Amleh, M. Y., & Atawnah, S. I. (2025). Adult-onset acute disseminated encephalomyelitis mimicking complex migraine: a CARE-compliant case report. Medicine (Baltimore), 104(20), e42458.