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Case Report

A Rare Case of Adult-Onset Acute Disseminated Encephalomyelitis Presenting with Seizure and Acute Confusion: A Case Report and Literature Review

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ABSTRACT

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated inflammatory demyelinating disorder of the central nervous system, typically encountered in the pediatric population. Adult-onset ADEM is uncommon and poses a diagnostic challenge, particularly when presenting with seizure as a cardinal feature and when initially mimicking infectious meningoencephalitis. A 33-year-old previously healthy man presented with a first-ever generalized tonic–clonic seizure preceded by a 1-week history of frontal headache and acute-onset confusion. Infectious meningoencephalitis was initially suspected, and empiric antimicrobials were started. Initial laboratory investigations are summarized in **Table 1**. Cerebrospinal fluid analysis showed lymphocytic pleocytosis with elevated protein, while multiplex polymerase chain reaction testing for common viral, bacterial, and fungal pathogens was negative, as shown in **Table 2**. Brain magnetic resonance imaging revealed extensive, poorly marginated T2/fluid-attenuated inversion recovery hyperintense lesions involving the subcortical white matter, basal ganglia, bilateral thalami, midbrain, and cerebellar peduncle, as shown in **Figures 1 to 4**. Extensive infectious, autoimmune, and paraneoplastic workups were negative, including serum myelin oligodendrocyte glycoprotein immunoglobulin G and aquaporin-4 immunoglobulin G antibodies. The patient was treated with high-dose intravenous methylprednisolone and intravenous immunoglobulin, leading to rapid and complete clinical recovery. A follow-up magnetic resonance imaging was not performed because the patient achieved complete clinical recovery and remained asymptomatic during follow-up. Adult ADEM should remain a differential diagnosis in young adults presenting with a first-time seizure and encephalopathy, even without preceding infection or vaccination. Early recognition, exclusion of infectious mimics, and timely immunotherapy with corticosteroids, with consideration of intravenous immunoglobulin in severe cases, are critical for favorable outcomes.

Key words: Acute disseminated encephalomyelitis, case report, adult, seizure, encephalopathy

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated inflammatory demyelinating disease of the central nervous system (CNS), classically characterized by an acute polyfocal neurological syndrome with encephalopathy and multifocal demyelinating lesions on magnetic resonance imaging (MRI). [1] Although ADEM is more commonly described in children, adult-onset cases are increasingly recognized and may be more diagnostically challenging because of overlap with multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), infectious encephalitis, autoimmune encephalitis, vasculitis, and neoplastic mimics. [2,3]

The International Pediatric Multiple Sclerosis Study Group criteria define ADEM as a first polyfocal demyelinating CNS event associated with encephalopathy, defined as altered mental status or behavioral change not explained by fever. [4] Although these criteria were developed for pediatric demyelinating disease, they remain clinically useful when approaching suspected ADEM in adults, particularly because encephalopathy helps distinguish ADEM from a first demyelinating event of MS. [3,4]

Seizures are a recognized manifestation of ADEM but are less commonly emphasized in adult presentations. In adults, a first-ever seizure with acute confusion frequently prompts an initial workup for infectious meningoencephalitis, structural brain lesions, metabolic abnormalities, or autoimmune encephalitis. This case adds to the adult ADEM literature by describing a seizure-predominant presentation without a clear infectious or vaccination prodrome, initially managed as suspected meningoencephalitis, with rapid recovery after early combined immunotherapy using corticosteroids and intravenous immunoglobulin (IVIG). [2,3]

CASE REPORT

A previously healthy 33-year-old man presented to the emergency department with a first-ever generalized tonic–clonic seizure lasting approximately 7 minutes. One week before admission, he developed a persistent frontal headache without associated fever, neck stiffness, vomiting, trauma, or other meningeal symptoms. One day before the presentation, his family noticed an acute change in his mental status characterized by intermittent confusion and disorientation. There was no history of preceding upper respiratory tract infection, gastrointestinal illness, recent travel, sick contacts, or recent vaccination. His past medical, medication, social, and family histories were otherwise unremarkable, with no prior seizures, substance use, or known autoimmune disease.

On arrival, his vital signs were within normal limits: temperature—36.8°C, blood pressure—118/72 mmHg, heart rate—76 beats/min, respiratory rate—16 breaths/min, and oxygen saturation—98% on room air. The initial clinical history supported encephalopathy at onset, based on the family's report of acute confusion and disorientation before presentation. By the time of the detailed neurological examination after stabilization in the emergency department, the patient had improved and was alert and fully oriented, with fluent speech and intact cranial nerves. Motor strength, sensation, coordination, and gait were normal, with no focal neurological deficits or meningeal signs. Kernig and Brudzinski signs were negative.

Initial laboratory investigations showed a white blood cell count of 9.4 × 10^3/μL, with mild neutrophilia at 8.3 × 10^3/μL and lymphocytes at the lower limit of normal at 0.8 × 10^3/μL. Hemoglobin was 13.2 g/dL, and platelets were 252 × 10^3/μL. Renal function, serum electrolytes, liver function tests, thyroid-stimulating hormone, vitamin D, and vitamin B12 levels were within normal limits. C-reactive protein (CRP) was elevated at 34 mg/L, with a reference range of 0 to 5 mg/L, while procalcitonin was normal at 0.05 ng/mL. The initial blood investigations are summarized in **Table 1**. The elevated CRP initially supported an infectious or inflammatory process but was considered nonspecific, particularly in the setting of negative microbiological testing and normal procalcitonin.

Given the acute presentation with seizure and encephalopathy, infectious meningoencephalitis was initially considered the primary working diagnosis. After initial neuroimaging excluded an acute hemorrhagic or space-occupying lesion, lumbar puncture was performed. Empiric intravenous ceftriaxone and acyclovir were started early after cerebrospinal fluid (CSF) sampling to provide broad coverage for bacterial and herpetic meningoencephalitis. Levetiracetam was also initiated for seizure prophylaxis.

CSF analysis showed an inflammatory profile with lymphocytic-predominant pleocytosis. The CSF white blood cell count was 95 cells/μL, consisting of 80% lymphocytes, 2% neutrophils, and 18% macrophages. The CSF red blood cell count was 1 cell/μL. CSF protein was elevated at 0.74 g/L, while CSF glucose was within normal limits at 3.3 mmol/L. No CSF oligoclonal bands were detected. CSF aerobic and anaerobic cultures, acid-fast bacilli smear and culture, tuberculosis polymerase chain reaction, and multiplex polymerase chain reaction testing for common viral, bacterial, and fungal pathogens were all negative. The CSF multiplex polymerase chain reaction results are summarized in **Table 2**.

CSF multiplex PCR analysis was negative for common viral, bacterial, and fungal pathogens, including herpes simplex virus, varicella-zoster virus, enterovirus, and major causes of community-acquired bacterial meningitis.

Electroencephalography was performed after the presenting seizure during the early inpatient evaluation. It demonstrated generalized slowing without epileptiform discharges, consistent with diffuse cerebral dysfunction. No electrographic seizures or features suggestive of ongoing subclinical seizure activity were identified.

A non-contrast computed tomography scan of the head showed no acute intracranial abnormality. A brain MRI was

Table 1: Blood analysis.

Laboratory parameter	Result	Reference range	Units
White blood cell count	9.4	4.0–10.0	×10^3 cells/μL
Neutrophils	8.3	1.5–8.0	×10^3 cells/μL
Lymphocytes	0.8	0.8–4.0	×10^3 cells/μL
Hemoglobin	13.2	13.6–18.0	g/dL
Platelets	252	150–450	×10^3 cells/μL
Creatinine	84	62–106	μmol/L
Blood urea nitrogen	3.6	2.5–7.8	mmol/L
Sodium	139	135–145	mmol/L
Potassium	4.2	3.5–5.1	mmol/L
Adjusted calcium	2.38	2.20–2.60	mmol/L
Alanine aminotransferase	11	0–40	U/L
Aspartate aminotransferase	16	0–40	U/L
C-reactive protein	34	0–5	mg/L
Procalcitonin	0.05	<0.50	ng/mL
Thyroid-stimulating hormone	2.3	0.4–4.0	μIU/mL
Vitamin D	35	30–100	ng/mL

Table 2: CSF multiplex PCR analysis.

Pathogen tested	Result
Cytomegalovirus PCR	Negative
Human herpesvirus 6 PCR	Negative
Herpes simplex virus 1 PCR	Negative
Herpes simplex virus 2 PCR	Negative
Varicella zoster virus PCR	Negative
Parechovirus PCR	Negative
Enterovirus PCR	Negative
Escherichia coli K1	Negative
Haemophilus influenzae	Negative
Listeria monocytogenes	Negative
Neisseria meningitidis	Negative
Streptococcus agalactiae	Negative
Streptococcus pneumoniae	Negative
Cryptococcus neoformans/gattii	Negative

obtained early during admission after initial stabilization and was the pivotal diagnostic investigation. MRI demonstrated extensive multifocal T2/fluid-attenuated inversion recovery (FLAIR) hyperintense lesions involving the right frontal cortical/subcortical region and left parietal cortical/subcortical region, as shown in **Figure 1**. Additional lesions were identified in the right basal ganglia and bilateral thalami, as shown in **Figure 2**; the left midbrain, as shown in **Figure 3**, and the left cerebellar peduncle, as shown in **Figure 4**. The lesions were poorly marginated and involved both subcortical white matter and deep gray nuclei. No contrast enhancement, restricted diffusion, hemorrhage, or significant mass effect was identified. No typical MS-pattern lesions such as Dawson's fingers, dominant periventricular ovoid lesions, or corpus callosum lesions were reported. MRI of the cervical and thoracic spine revealed no abnormalities.

An extensive autoimmune, infectious, and paraneoplastic workup was subsequently performed to exclude alternative inflammatory, vasculitic, infectious, and autoimmune encephalitic etiologies. Antinuclear antibody, antineutrophil cytoplasmic antibody, extractable nuclear antigen panel, anti-double-stranded DNA, antiphospholipid antibodies, autoimmune encephalitis panel, HIV serology, syphilis serology, and hepatitis serologies were all negative.

As the CSF multiplex polymerase chain reaction, cultures, and other infectious investigations returned negative, and as MRI findings were more suggestive of inflammatory demyelination than infection, empiric ceftriaxone and acyclovir were discontinued. The absence of fever, meningeal signs, positive cultures, viral polymerase chain reaction positivity, restricted diffusion, hemorrhage, or imaging features typical of encephalitis further reduced the likelihood of infectious meningoencephalitis.

Collectively, the clinical presentation of acute encephalopathy and seizure, inflammatory CSF findings, absence of CSF oligoclonal bands, and MRI appearance of extensive bilateral poorly demarcated lesions involving both white matter and deep gray nuclei favored ADEM over MS. Autoimmune encephalitis was also considered; however, the negative autoimmune encephalitis panel, absence of a typical limbic-predominant MRI pattern, lack of persistent psychiatric or behavioral syndrome, and monophasic clinical course argued against it. Primary CNS vasculitis was considered unlikely

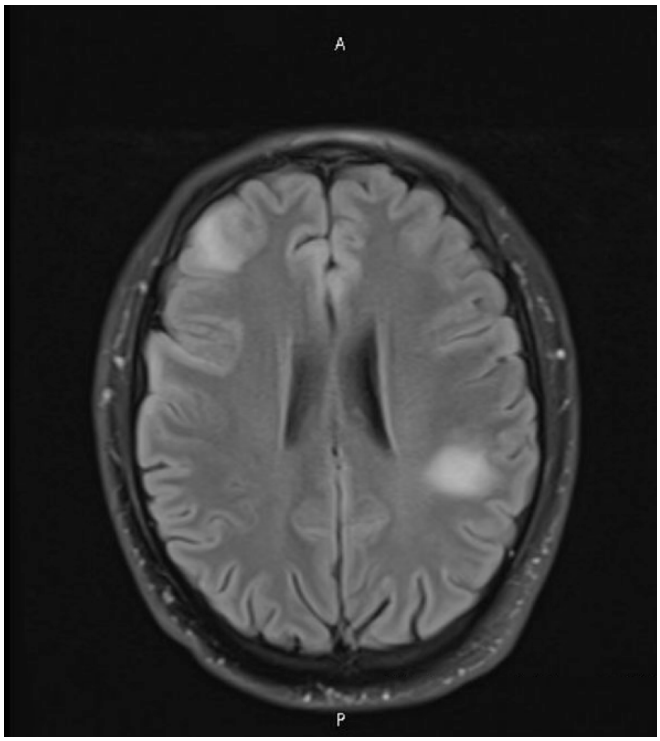

Figure 1: T2/FLAIR hyperintensities are noted in the right frontal cortical/subcortical region and left parietal cortical/subcortical region.

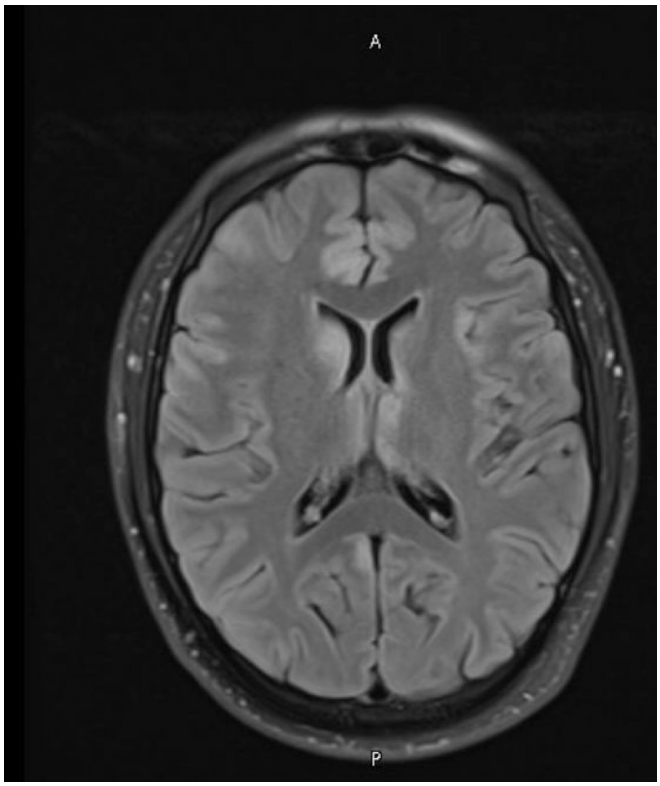

Figure 2: T2/FLAIR hyperintense lesions are identified in the right basal ganglia and bilateral thalami.

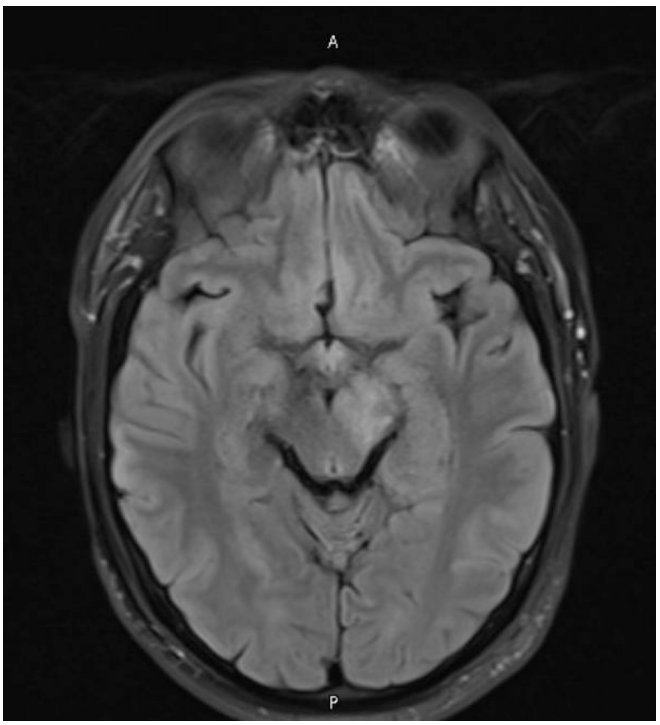

Figure 3: Focal T2/FLAIR hyperintensity is seen in the left midbrain.

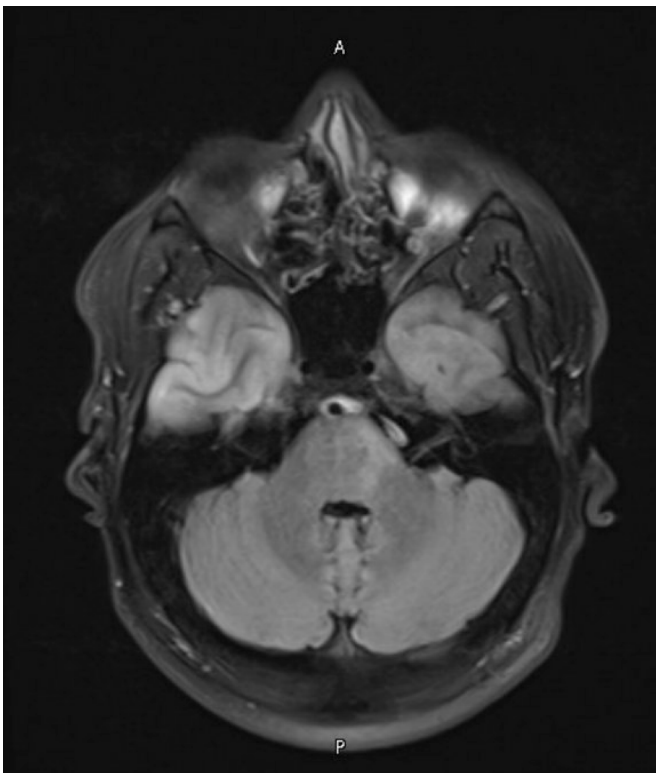

Figure 4: T2/FLAIR hyperintensity is observed in the left cerebellar peduncle.

because of the nonvascular distribution of lesions, lack of infarcts or hemorrhage, negative systemic autoimmune workup, and favorable response to demyelination-directed immunotherapy. Dedicated vessel imaging or brain biopsy was not performed because the clinical and radiological findings, together with subsequent improvement, did not strongly support vasculitis to justify invasive testing. Acute hemorrhagic leukoencephalitis, a fulminant hemorrhagic variant of ADEM, was excluded clinically and radiologically because there was no hemorrhage, rapid neurological deterioration, severe mass effect, or malignant cerebral edema.

To exclude antibody-mediated demyelinating syndromes that may mimic ADEM, serum testing for myelin oligodendrocyte glycoprotein immunoglobulin G (MOG-IgG) and aquaporin-4 immunoglobulin G (AQP4-IgG) antibodies was performed. The available laboratory report documented negative MOG-IgG and AQP4-IgG results; however, the exact assay platform was not specified in the available report and should be confirmed with the reference laboratory if required by the journal. Given the severity of the encephalopathic presentation and extensive multifocal CNS involvement, adjunctive IVIG was administered early alongside corticosteroids rather than waiting for steroid failure. The patient received high-dose intravenous methylprednisolone 1 g daily for 5 days, together with IVIG at 0.4 g/kg/day for 5 days.

The patient demonstrated rapid clinical improvement, with complete resolution of confusion and no further seizures. Subsequent MOG-IgG and AQP4-IgG testing returned negative, making MOGAD and NMOSD less likely. He returned to baseline cognition with complete resolution of encephalopathy and no residual focal neurological deficits. Formal neuropsychological testing was not performed during admission; however, bedside cognitive assessment before discharge showed normal orientation, attention, language, and memory by clinical examination.

He was discharged home neurologically stable on levetiracetam. At 4-month follow-up, he remained entirely symptom-free, with no recurrent seizures, cognitive complaints, headaches, visual symptoms, motor deficits, sensory symptoms, or gait disturbance. Bedside cognitive assessment at follow-up remained normal, and levetiracetam was successfully discontinued. A follow-up MRI was not performed because the patient had complete clinical recovery, no residual neurological deficits, and no recurrent symptoms during follow-up. Although the absence of follow-up MRI limits radiological confirmation of lesion regression, the patient's clinical recovery and absence of relapse during 8 months of follow-up strongly supported a monophasic ADEM course. Continued clinical follow-up was advised, with repeat MRI recommended if new neurological symptoms develop or if relapse is suspected. The overall clinical course and management milestones are summarized in **Table 3**.

DISCUSSION

Overview of ADEM

ADEM is an acute inflammatory demyelinating disorder of the CNS, generally considered monophasic and presumed to be immune-mediated. It is thought to result from a transient autoimmune response against myelin antigens, potentially triggered by molecular mimicry or nonspecific immune activation after infection or vaccination. [1,3] Although commonly described in children, adult ADEM is well recognized and may have a more severe clinical course and less favorable recovery than pediatric ADEM in some series. [2,5]

Table 3: Clinical timeline.

Time point	Clinical event/investigation	Management/outcome
One week before admission	Persistent frontal headache	No fever, meningism, trauma, or vomiting
One day before admission	Acute confusion and disorientation were noticed by the family	Encephalopathy present at onset
Day of admission	First-ever generalized tonic–clonic seizure lasting approximately 7 minutes	Levetiracetam initiated
Early admission	CT head negative; lumbar puncture showed lymphocytic pleocytosis and elevated protein.	Empiric ceftriaxone and acyclovir were started for suspected meningoencephalitis.
Early admission	EEG showed generalized slowing without epileptiform discharges	No evidence of subclinical seizures
Early admission	Brain MRI showed multifocal T2/FLAIR hyperintense lesions involving white matter and deep gray nuclei.	The demyelinating process favored
After a negative infectious workup	CSF cultures, PCR studies, TB testing, and autoimmune workup were negative	Antimicrobials discontinued
Treatment phase	High-dose intravenous methylprednisolone and IVIG were administered for 5 days.	Rapid clinical improvement
Discharge	Neurologically stable and back to baseline cognition	Discharged on levetiracetam
4 months	No recurrent seizures or cognitive symptoms	Levetiracetam discontinued
8 months	No relapse or new neurological symptoms	Continued clinical follow-up advised; repeat MRI recommended if symptoms recur.

The diagnosis remains clinical–radiological and requires careful exclusion of mimics. Encephalopathy is a key diagnostic feature and helps distinguish ADEM from MS, particularly when multifocal demyelinating lesions are present. [4] In our case, encephalopathy was evident at onset through acute confusion and disorientation reported by the family. However, the patient had partially improved by the time of the detailed neurological examination.

Why This Case Is Unusual

This case is unusual for several reasons. First, the patient was 33 years old, whereas ADEM is predominantly a pediatric disorder and is uncommon in adults. [1,2] Second, there was no identifiable preceding infection, vaccination, or systemic illness, which made the diagnosis less immediately apparent. Third, the presenting event was a first-ever generalized tonic–clonic seizure, which initially shifted the diagnostic focus toward infectious meningoencephalitis, structural brain disease, or autoimmune encephalitis. Seizures have been reported in adult ADEM cohorts but are not usually the dominant presenting feature, making this presentation clinically important. [2,5]

The case, therefore, adds to the adult ADEM literature by emphasizing that seizure-predominant ADEM may occur without a clear prodrome, and that early MRI recognition can redirect management from prolonged antimicrobial therapy toward immunotherapy after infectious mimics are reasonably excluded.

MRI Characteristics

MRI was central to the diagnosis. The lesions were extensive, multifocal, poorly marginated, and involved both supratentorial white matter and deep gray matter, including the thalami and basal ganglia. Deep gray matter involvement, particularly bilateral thalamic involvement, supports ADEM over a typical first presentation of MS. [4,6] In contrast, MS more commonly demonstrates discrete ovoid periventricular lesions, callososeptal interface involvement, Dawson's fingers, and persistent lesion accrual over time. [4,6]

The absence of contrast enhancement in this case does not exclude active inflammatory demyelination. Enhancement in ADEM is variable and may be absent despite clinically active disease. The absence of restricted diffusion, hemorrhage, or significant mass effect also made infarction, abscess, neoplasm, and acute hemorrhagic leukoencephalitis less likely. [7]

Differential Diagnosis

The initial differential diagnosis was broad and included infectious meningoencephalitis, MS, MOGAD, NMOSD, autoimmune encephalitis, primary CNS vasculitis, and acute hemorrhagic leukoencephalitis.

Infectious meningoencephalitis was appropriately considered at presentation because of seizure, headache, encephalopathy, CSF pleocytosis, and elevated CRP. However, the absence of fever and meningeal signs, normal procalcitonin, negative CSF cultures, negative CSF multiplex polymerase chain reaction, negative tuberculosis studies, and MRI pattern of demyelination rather than encephalitis made infection unlikely. Empiric antimicrobials were therefore discontinued once microbiological testing and MRI findings supported a noninfectious inflammatory demyelinating diagnosis.

MS was considered because of multifocal CNS lesions; however, the presence of encephalopathy, poorly demarcated lesions, bilateral deep gray matter involvement, absence of typical periventricular ovoid MS lesions, absence of CSF

oligoclonal bands, and complete clinical recovery favored ADEM. [4,6]

MOGAD and NMOSD were important considerations because both can mimic ADEM and may require different long-term management. MOG-IgG and AQP4-IgG were negative, making these diagnoses less likely. The assay type was not specified in the available report; however, current recommendations emphasize the importance of cell-based assays for MOG-IgG testing, and this should be confirmed with the reference laboratory if possible. [8,9,10]

Autoimmune and paraneoplastic encephalitis were considered because of seizures and encephalopathy. However, the negative autoimmune encephalitis panel, lack of a limbic-predominant MRI pattern, absence of persistent psychiatric or memory-dominant syndrome, and subsequent complete clinical recovery argued against this diagnosis. Primary CNS vasculitis was also unlikely because there were no infarcts, hemorrhage, vessel-territory lesions, systemic vasculitic features, or supportive autoimmune markers. Vessel imaging and brain biopsy were not pursued because the overall clinical course and imaging pattern favored demyelination, and the patient improved rapidly with immunotherapy.

Acute hemorrhagic leukoencephalitis was excluded because the patient did not have a fulminant deteriorating course, hemorrhagic lesions, malignant edema, or severe mass effect. [7]

Treatment Rationale

There are no large randomized controlled trials guiding ADEM treatment, and management is largely based on observational evidence and expert consensus. High-dose intravenous methylprednisolone for 3 to 5 days is widely used as first-line therapy. [3,11,12] IVIG and plasma exchange are generally considered for severe, steroid-refractory, or rapidly progressive cases. [11,12]

In this patient, IVIG was started early and concurrently with corticosteroids because of the extensive multifocal brain involvement, encephalopathic presentation, and concern for severe immune-mediated demyelination. This approach was chosen to accelerate immunomodulation while infection was actively excluded. The patient's rapid clinical recovery supported an inflammatory immune-mediated process. Plasma exchange was not required because the patient improved promptly with corticosteroids and IVIG. In cases with poor response or clinical deterioration, plasma exchange remains an important escalation therapy. [11,12]

Elevated CRP in the Diagnostic Workup

The elevated CRP of 34 mg/L initially contributed to concern for infection and justified early empiric antimicrobial coverage. However, CRP is nonspecific and may rise in systemic inflammatory states, after seizures, or with intercurrent inflammation. In this case, the normal procalcitonin, absence of fever, negative CSF microbiology, and MRI pattern argued against bacterial infection. Therefore, the CRP elevation was interpreted as a nonspecific inflammatory marker rather than evidence of bacterial meningoencephalitis.

Prognosis and Follow-Up

Adult ADEM generally has a favorable prognosis when recognized and treated early, but outcomes may be less favorable than in children, and long-term follow-up is important. [5,12] Our patient achieved complete clinical recovery, discontinued antiseizure therapy after 4 months, and remained asymptomatic during 8 months of follow-up. A follow-up MRI was not performed because the patient had no recurrent symptoms, no residual neurological deficits, and complete clinical recovery. This represents a limitation of the report, as radiological regression could not be documented. However, the absence of clinical relapse during follow-up strongly supports a monophasic ADEM course. Continued clinical surveillance is important, and repeat MRI should be performed if new neurological symptoms occur or if there is concern for evolution into MS, MOGAD, or another relapsing demyelinating disorder. [5,12]

Literature Comparison

Adult ADEM series have shown that adult patients may experience more severe disease courses than children, including higher rates of hospitalization, intensive care requirements, incomplete recovery, and mortality. [5] Compared with more severe adult presentations involving prominent motor deficits, coma, or spinal cord involvement, our patient had a seizure- and encephalopathy-predominant presentation without persistent focal neurological deficits. The complete clinical recovery suggests a favorable subset of adult ADEM. The early use of combined corticosteroid and IVIG therapy may have contributed to rapid recovery, although causality cannot be established from a single case. [5,11,12]

Clinical Takeaway

In a young adult presenting with acute encephalopathy, first-time seizure, inflammatory CSF findings, and multifocal poorly marginated T2/FLAIR lesions involving both white matter and deep gray nuclei, ADEM should be considered even without a clear prodrome. Early MRI, prompt CSF analysis to exclude infection, targeted antibody testing for MOG-IgG and AQP4-IgG, and timely immunotherapy can lead to excellent outcomes. [3,4,8,11]

CONCLUSIONS

This case demonstrates that ADEM, although rare in adults, can present acutely with a generalized seizure and encephalopathy without a preceding infectious or vaccination trigger. The diagnosis was supported by multifocal poorly marginated T2/FLAIR lesions involving the subcortical white matter, thalami, basal ganglia, midbrain, and cerebellar peduncle; inflammatory CSF findings; absence of oligoclonal bands; and exclusion of infectious, autoimmune, MOGAD, and NMOSD mimics. Early treatment with high-dose corticosteroids and IVIG resulted in complete clinical recovery. A follow-up MRI was not performed because the patient remained clinically well without recurrent neurological symptoms; however, this is acknowledged as a limitation because radiological regression could not be documented. Continued long-term follow-up is important to confirm a

monophasic course and exclude later evolution into MS, MOGAD, or another relapsing demyelinating disorder.

KEY CLINICAL MESSAGES

Adult ADEM should be considered in a seizure with encephalopathy: Although uncommon, adult-onset ADEM can present with a first-time generalized seizure and acute confusion, even without a preceding infection or vaccination.

MRI is central to diagnosis: Multifocal, poorly marginated T2/FLAIR hyperintense lesions involving both white matter and deep gray nuclei, especially the thalami and basal ganglia, are highly informative but must be interpreted with clinical and CSF findings.

Exclude important mimics: CSF analysis, infectious testing, autoimmune/paraneoplastic evaluation, and serum MOG-IgG and AQP4-IgG testing are important to differentiate ADEM from infection, MS, MOGAD, NMOSD, autoimmune encephalitis, and vasculitis.

Early immunotherapy can improve outcome: High-dose corticosteroids remain in first-line therapy. IVIG may be considered early in severe presentations or used as escalation therapy when response to corticosteroids is incomplete.

Long-term follow-up is essential: Even after completing clinical recovery, continued clinical follow-up is recommended to confirm a monophasic course and detect possible evolution to MS, MOGAD, or another relapsing demyelinating disease. Repeat MRI should be considered if new neurological symptoms occur or if relapse is suspected.

CONSENT

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

AUTHORS' CONTRIBUTION

All authors have significantly contributed to the work, whether by following the case at the bedside, conducting literature searches, drafting, revising, or critically reviewing the article. They have given their final approval of the version to be published, have agreed with the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

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CONFLICT OF INTEREST

None.

REFERENCES

1. Menge T, Hemmer B, Nessler S, Wiendl H, Neuhaus O, Hartung HP. Acute disseminated encephalomyelitis: An update. Arch Neurol. 2005;62(11):1673–1680. http://doi.org/10.1001/archneur.62.11.1673
2. Schwarz S, Mohr A, Knauth M, Wildemann B, Storch-Hagenlocher B. Acute disseminated encephalomyelitis: A follow-up study of 40 adult patients. Neurology. 2001;56(10):1313–1318. https://doi.org/10.1212/WNL.56.10.1313
3. Tenembaum S, Chitnis T, Ness J, Hahn JS; International Pediatric MS Study Group. Acute disseminated encephalomyelitis. Neurology. 2007;68(16 Suppl 2):S23–S36. https://doi.org/10.1212/01.wnl.0000259404.51352.7f
4. Krupp LB, Tardieu M, Amato MP, Banwell B, Chitnis T, Dale RC, et al; International Pediatric Multiple Sclerosis Study Group. International Pediatric Multiple Sclerosis Study Group criteria for pediatric multiple sclerosis and immune-mediated central nervous system demyelinating disorders: Revisions to the 2007 definitions. Mult Scler. 2013;19(10):1261–1267. http://doi.org/10.1177/1352458513484547
5. Ketelslegers IA, Visser IER, Neuteboom RF, Boon M, Catsman-Berrevoets CE, Hintzen RQ. Disease course and outcome of acute disseminated encephalomyelitis is more severe in adults than in children. Mult Scler. 2011;17(4):441–448. http://doi.org/10.1177/1352458510390068
6. Verhey LH, Branson HM, Shroff MM, Callen DJA, Sled JG, Narayanan S, et al; Canadian Pediatric Demyelinating Disease Network. MRI parameters for prediction of multiple sclerosis diagnosis in children with acute CNS demyelination: A prospective national cohort study. Lancet Neurol. 2011;10(12):1065–1073. http://doi.org/10.1016/S1474-4422(11)70250-2
7. de Seze J, Debouverie M, Zephir H, Lebrun C, Blanc F, Bourg V, et al. Acute fulminant demyelinating disease: A descriptive study of 60 patients. Arch Neurol. 2007;64(10):1426–1432. http://doi.org/10.1001/archneur.64.10.1426
8. Jarius S, Paul F, Aktas O, Asgari N, Dale RC, de Seze J, et al. MOG encephalomyelitis: International recommendations on diagnosis and antibody testing. J Neuroinflammation. 2018;15(1):134. http://doi.org/10.1186/s12974-018-1144-2
9. Reindl M, Waters P. Myelin oligodendrocyte glycoprotein antibodies in neurological disease. Nat Rev Neurol. 2019;15(2):89–102. http://doi.org/10.1038/s41582-018-0112-x
10. Banwell B, Bennett JL, Marignier R, Kim HJ, Brilot F, Flanagan EP, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. Lancet Neurol. 2023;22(3):268–282. https://doi.org/10.1186/s12974-018-1144-2
11. Pohl D, Alper G, Van Haren K, Kornberg AJ, Lucchinetti CF, Tenembaum S, et al. Acute disseminated encephalomyelitis: Updates on an inflammatory CNS syndrome. Neurology. 2016;87(9 Suppl 2):S38–S45. http://doi.org/10.1212/WNL.0000000000002825
12. Paolilo RB, Deiva K, Neuteboom R, Rostasy K, Lim M. Acute disseminated encephalomyelitis: Current perspectives. Children (Basel). 2020;7(11):210. http://doi.org/10.3390/children7110210