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Severe Fulminant Acute Disseminated Encephalomyelitis (ADEM) in an 18-Year-Old

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Abstract

Background: Acute disseminated encephalomyelitis (ADEM) is a rare demyelinating disease of the central nervous system usually preceded by an infection. While mainly a childhood disease, ADEM carries a worse prognosis in adults. We present a case of severe fulminant ADEM in an 18-year-old patient resulting in rapid neurological injury. **Case Report**: An 18-year-old woman presented to our emergency room with fever and an altered level of consciousness followed by a generalized tonic-clonic seizure. Cerebrospinal fluid (CSF) findings were initially suggestive of meningitis; however, subsequent neuroimaging and laboratory evaluation favored a diagnosis of acute disseminated encephalomyelitis. Despite treatment with broad-spectrum antimicrobials and high-dose corticosteroids, the patient experienced rapid neurological deterioration, culminating in extensive and irreversible brain injury. **Conclusion**: Prompt diagnosis and treatment of ADEM, particularly in the adult population, remains a significant clinical challenge. Our case underscores the need for heightened vigilance and continued research to assist clinicians in the time-sensitive identification of this severe demyelinating disease.

Keywords

Encephalomyelitis, Demyelinating Syndromes, Neuroimmunology, Molecular Mimicry

1. Introduction

Acute disseminated encephalomyelitis (ADEM) is a rare immune-mediated demyelinating disorder of the central nervous system, most commonly associated with a preceding infection [1]. Usually seen in childhood following viral infections, it has a more severe course and is associated with a worse outcome in adults [2]. Here, we describe an 18-year-old patient with a fulminant case of ADEM re-

sulting in rapid neurological deterioration.

2. Case Report

An 18-year-old woman with no significant medical history presented to our emergency room after a generalized tonic-clonic seizure following a two-day history of fever and confusion. She had no history of recent travel or substance use and had no sick contacts.

On evaluation in the ER, she was found to be drowsy but arousable, aphasic, and unable to follow simple commands. She had erythema of the oropharynx but no neck stiffness. After a CT head revealed no abnormalities, a lumbar puncture was performed, and she was administered empiric antimicrobials to treat meningitis along with anti-epileptic agents. Cerebrospinal fluid (CSF) analysis showed an elevated white cell count with a neutrophilic predominance; CSF protein was abnormally high, and CSF glucose was within the normal range (Table 1). Despite the normal glucose, the overall CSF profile was initially concerning for meningitis, and antimicrobial therapy was therefore continued. Aside from mild leukocytosis, complete blood counts, serum chemistry, toxicology screen, and respiratory viral panel were all unremarkable.

Table 1. Cerebrospinal fluid analysis.

CSF analysis	
Color	Colorless
Turbidity	Clear
Protein	479 mg/dL
Glucose	108 mg/dL
Concurrent blood glucose	190 mg/dL
Red blood cells	53/mm^3
Nucleated cells	274/mm^3
Polymorphonuclear cells	76 %
Lymphocytes	5 %
Mononuclear cells	19 %

Due to increasing obtundation and hypoxemia, the patient underwent tracheal intubation later the same day. An MRI of the brain showed multiple bilateral periventricular white matter lesions (Figure 1(A), Figure 1(D), Figure 1(E) and Figure 2(A)) along with areas of increased T2 signal with restricted diffusion (Figure 1(B) and Figure 1(C)). Also seen were small areas of hypointensity on susceptibility-weighted images consistent with microhemorrhages (Figure 2(B)). MRI of the spine showed multifocal areas of demyelination in the cervical and thoracic spinal cord (Figure 3 and Figure 4). High-dose steroids were initiated at this point due to suspicion for ADEM and rapid progression of the disease. A CT

of the abdomen and pelvis showed mesenteric and retroperitoneal lymphadenopathy with no evidence of an occult malignancy.

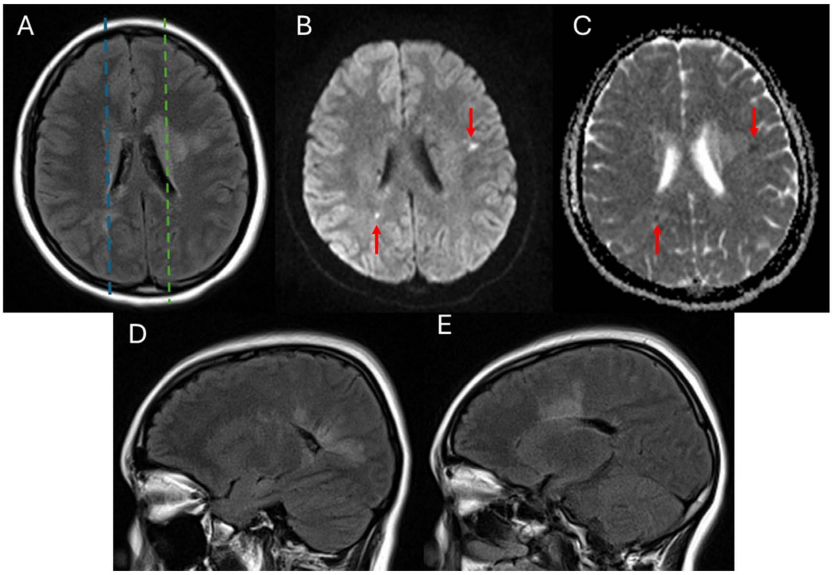

Figure 1. Abnormal T2 hyperintensities and diffusion restriction on MRI brain. ((A) - (C), Axial slices of T2 FLAIR (A), diffusion-weighted imaging (B), and apparent diffusion coefficient (C) at the same level, showing small areas of diffusion restriction (red arrows) within the larger areas of T2 hyperintensities. (D)-(E), Sagittal T2 FLAIR at the level of the blue dotted line (D) and green dotted line (E) in A, further demonstrating the periventricular distribution of T2 hyperintensities.

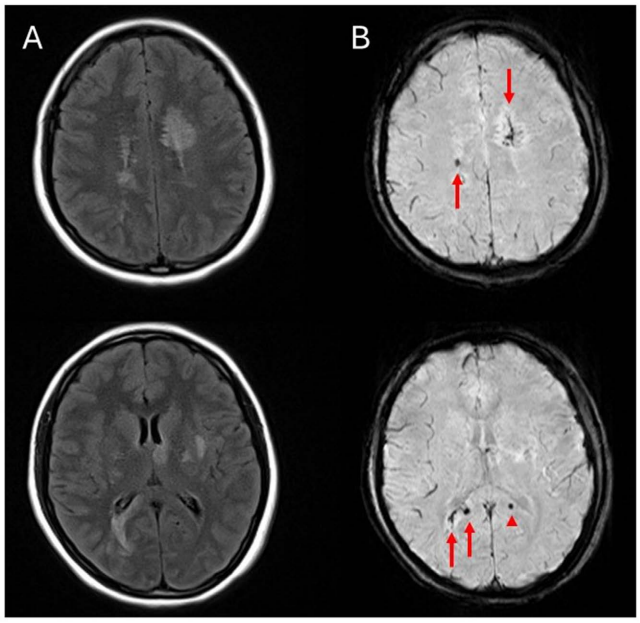

Figure 2. Microhemorrhages in the areas of T2 hyperintensities. (A) and (B), Axial slices of T2 FLAIR sequences (A) and susceptibility-weighted imaging (B) at two different levels demonstrate the extent and variability of periventricular and juxtacortical T2 hyperintensities (A), with areas of hypointensity (B) inside (arrows) and outside (arrowhead) the areas of T2 hyperintensity.

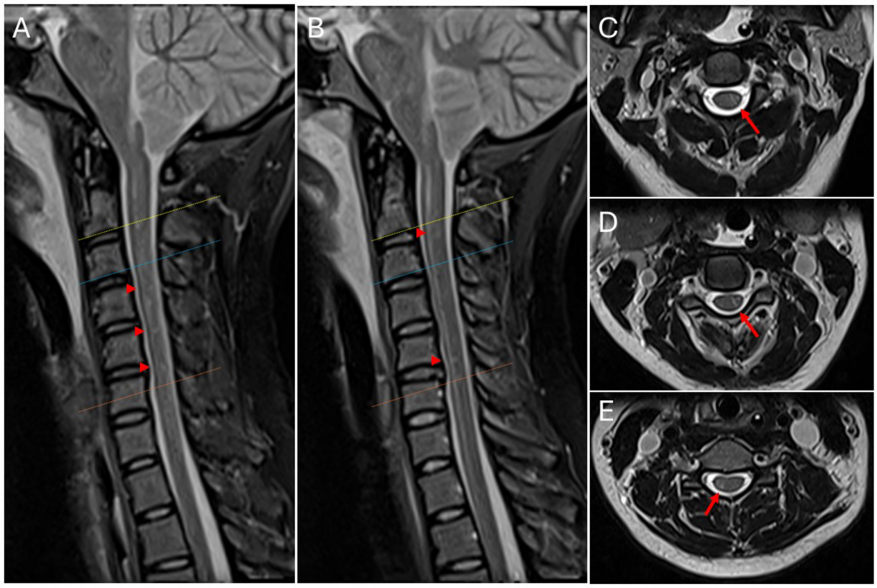

Figure 3. Patchy T2 hyperintensities in the cervical spine on MRI. (A) and (B), Sagittal T2 STIR sequences in different sagittal planes show patchy areas of T2 hyperintensities (arrowheads). (C)-(E), Axial T2 sequences at the levels of the yellow (C), blue (D), and orange (E) dotted lines show areas of T2 hyperintensities (arrows).

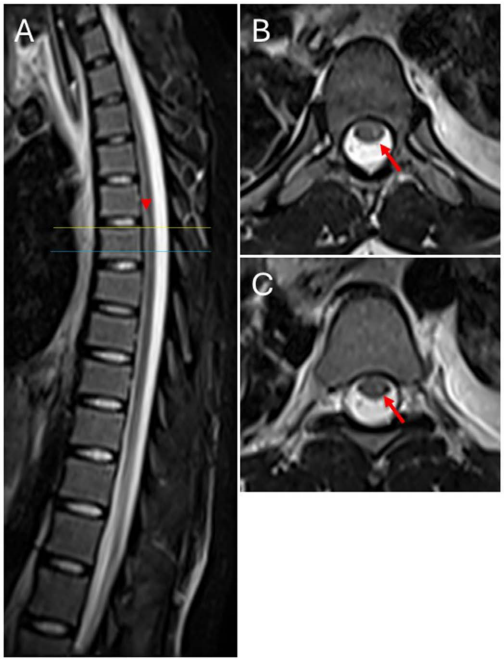

Figure 4. T2 hyperintensities in the thoracic spine on MRI. A, Sagittal T2 sequence showing subtle T2 hyperintensity of the anterior spinal cord (arrowhead). (B) and (C), Axial T2 sequences at the levels of the yellow (B) and blue (C) dotted lines show areas of T2 hyperintensity in the left hemicord (arrows).

The next morning, the patient's neurologic exam deteriorated with loss of cranial nerve reflexes, absence of spontaneous respiratory effort, and a lack of motor response. An urgent CT head revealed diffuse cerebral edema, with loss of 'gray-

white' differentiation (Figure 5). Hypertonic saline was administered to reduce intracranial pressure; however, there was no clinical improvement. The patient's family declined a decompressive hemicraniectomy and other invasive interventions. The patient was subsequently declared brain dead.

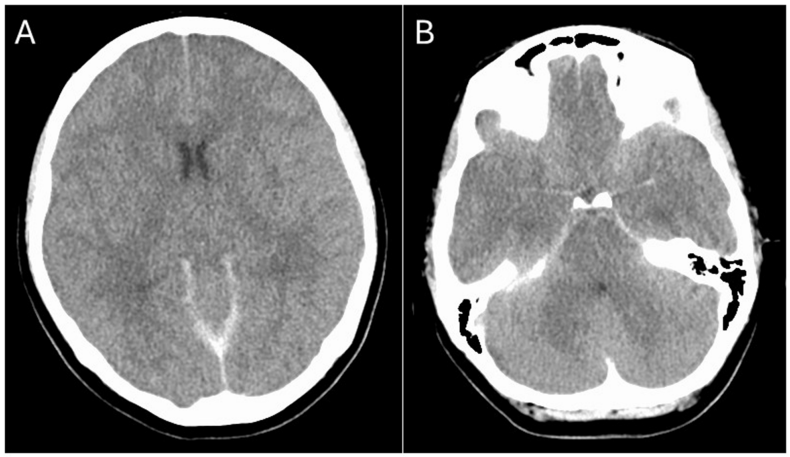

Figure 5. Diffuse cerebral edema on CT head. A-B, Axial sequences at different levels showing loss of gray-white differentiation ((A), (B)) and pseudosubarachnoid hemorrhage (B).

A comprehensive panel of serum and CSF studies—including autoimmune antibody testing, CSF cultures, serologic tests, metagenomic next-generation sequencing for DNA and RNA viruses, bacteria, fungi, and parasites, as well as blood cultures—eventually returned negative, with the exception of Epstein–Barr virus (EBV) serology indicating a recent infection, likely a few weeks prior to presentation (Table 2).

3. Discussion

The earliest descriptions of ADEM seem to date back to the 18th century [3]. Although its etiology has not been definitively established, the condition is believed to arise from molecular mimicry, typically following infection, resulting in autoimmune-mediated demyelination of the central nervous system [4].

The 2012 update from the International Pediatric Multiple Sclerosis Study Group (IPMSSG) established consensus definitions and diagnostic criteria for pediatric acquired demyelinating syndromes, including ADEM [5]. Adults and children with ADEM often differ in their clinical presentations, with adults more commonly exhibiting long-tract neurological signs, whereas children typically present with fever, encephalopathy, and meningeal features [6]. In the absence of formal adult-specific criteria, the IPMSSG criteria have been applied in the evaluation of ADEM in adult patients [7]. However, as adults with ADEM less commonly present with encephalopathy, the applicability of these criteria to adult patients is limited. Furthermore, given the rarity of ADEM in adults and the predominantly retrospective nature of available studies, formal adult-specific diagnostic criteria are not well established [8].

Table 2. Comprehensive list of CSF and serum studies. Note that all studies were negative except for the EBV serology panel, which indicated a recent infection in the prior 2 - 3 months, and positive parvovirus IgG, which indicated a prior exposure.

Comprehensive CSF and serum studies	
CSF Studies	**CSF Encephalopathy Antibody Panel**
Herpes Simplex Type 1 PCR Herpes Simplex Type 2 PCR Enterovirus PCR Cryptococcal antigen West Nile IgG and IgM VDRL Meningitis/Encephalitis Panel by PCR E. coli Heomophilus influenza Listeria monocytogenes Neisseria meningitidis Streptococcus agalactiae Streptococcus pneumoniae Cytomegalovirus Enterovirus HSV 1 & 2 Human herpesvirus 6 Human parechovirus Varicella zoster virus Cryptococcus neoformans/gattii DelveBio mNGS No DNA viruses, RNA viruses, bacteria, fungi, or parasites were detected.	1) *α*-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor antibody (AMPA-R Ab) 2) Amphiphysin Ab (Amphiphysin Ab) 3) Contactin-associated protein-like 2 antibody (CASPR2-IgG) 4) Collapsin response mediator protein 5 antibody (CRMP-5-IgG) 5) Gamma-aminobutyric acid type B receptor Ab (GABA-B-R Ab) 6) Glutamic acid decarboxylase 65 Ab (GAD65 Ab) 7) Glial fibrillary acidic protein antibody (GFAP Ab) 8) Leucine-rich glioma-inactivated 1 antibody (LGI1-IgG) 9) Metabotropic glutamate receptor 1 antibody (mGluR1 Ab) 10) N-Methyl-D-aspartate receptor Ab (NMDA-R Ab) 11) Purkinje cell cytoplasmic antibody type 1 (Anti-Yo) (PCA antibody type 1) 12) Purkinje cell cytoplasmic antibody type 2 (Anti-Ri) (PCA antibody type 2) 13) Purkinje cell cytoplasmic antibody type Tr (Anti-Tr) (PCA Type Tr) 14) Dipeptidyl-peptidase-like protein 6 antibody (DPPX Ab) 15) Neuronal intermediate filament antibody (NIF Ab) 16) IgLON family member 5 Ab (IgLON5 Ab) 17) Neurochondrin Antibody (Neurochondrin Ab) 18) Septin-7 Ab (Septin-7 Ab) 19) Anti-glial nuclear antibody type 1 (AGNA-1 Ab) 20) Anti-neuronal nuclear antibody type 1 (Anti-Hu) (ANNA antibody type 1) 21) Anti-neuronal nuclear antibody type 2 (Anti-Ri) (ANNA antibody type 2) 22) Anti-neuronal nuclear antibody type 3 (ANNA antibody type 3) 23) Tripartite motif-containing protein 46 antibody (TRIM46 Ab) 24) Phosphodiesterase 10A Antibody (PDE10A Ab)
Serum Serology	
Complement C3 and C4 Complement CH50 functional assay ANA Screen Varicella IgM and IgG CMV IgG and IgM HIV Screen Bartonella henselae IgG and IgM Rubeola IgM CMV IgG and IgM Borrelia burgdorferi Mycoplasma IgG and IgM Rabies antibody screen Parvovirus IgG (positive) and IgM were negative Epstein–Barr virus antibody panel: VCA IgG: Positive VCA IgM: Positive EBNA IgG: Positive EA-D IgG: Negative	

Adults with ADEM tend to have a more severe clinical course and worse outcomes than children, including longer hospital stays, increased need for intensive care, and higher mortality rates [9]. This may be a result of more intense inflammatory responses, greater blood–brain barrier disruption, and a higher likelihood of catastrophic variants such as acute hemorrhagic leukoencephalitis, in which edema, vascular necrosis, and hemorrhage contribute to irreversible brain injury [10]. Prompt recognition of ADEM is particularly crucial in adults and relies on a high index of clinical suspicion, in the context of a preceding infection, followed by multifocal neurological deficits, characteristic neuroimaging findings, and the exclusion of certain alternative diagnoses.

CSF findings in ADEM tend to be non-specific. Early in the course, CSF pleocytosis may show a neutrophilic predominance, shifting to a lymphocytic pattern as the disease progresses. CSF protein is usually only mildly elevated [4].

Early in the course, CT imaging is usually normal. MRI of the brain is the imaging modality of choice, and typical findings include multifocal, bilateral, hyperintense, poorly demarcated white matter lesions on T2-weighted images. These lesions are typically smaller when the MOG antibody is negative, and larger when the antibody is positive in ADEM patients [11]. Spinal cord involvement is described in a third of patients, with large, confluent lesions typically involving multiple segments and more likely to occur in patients with positive MOG antibody [11].

There is considerable overlap between ADEM, acute fulminant multiple sclerosis (MS), myelin oligodendrocyte glycoprotein antibody-associated disorder (MOG-AD), and neuromyelitis optica spectrum disorder (NMOSD) [1]. Although these inflammatory demyelinating disorders arise from distinct pathophysiological mechanisms, they frequently share overlapping clinical features. Notably, ADEM, MOG-AD, and NMOSD are often preceded by recent infections, while fever and encephalopathy are common in ADEM, MOG-AD, and fulminant MS [12]. A positive MOG antibody can be seen with nearly 40% of adults with ADEM [13] and up to 65% of children with ADEM [14]. While ADEM presents as a monophasic illness, MOG-AD typically exhibits a relapsing clinical pattern [15].

On the cusp of adulthood, our patient presented with antecedent EBV infection, fevers, erythema of the oropharynx and lymphadenopathy. She did not have a history of relapsing demyelinating disease. Her clinical presentation (fever, encephalopathy and rapid neurological deterioration), CSF profile, and neuroimaging strongly pointed to ADEM as the underlying diagnosis. Although the CSF findings could be suggestive of meningitis, the imaging features and negative microbiologic studies make this less likely. The size and appearance of the lesions on MRI brain and spine were consistent with a negative MOG antibody; however, this was not sent prior to the patient's demise. Despite treatment with empiric antimicrobials and high-dose corticosteroids, her condition deteriorated rapidly, progressing to irreversible brain injury within 48 hours of hospitalization.

4. Conclusion

Given its rarity and the absence of adult-specific diagnostic criteria, the diagnosis and management of ADEM in adults remain challenging. Timely MRI of the brain and spine, particularly in patients presenting with focal neurological deficits, may be the most helpful diagnostic modality for distinguishing ADEM from infectious meningitis. This case, along with the existing literature, highlights the need for continued research, increased clinical awareness, and the development of diagnostic criteria tailored to adult patients with ADEM.

Data Availability

The data supporting the findings of this case report is available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Abbreviations

Ab: Antibody;

CSF: Cerebrospinal Fluid;

PCR: Polymerase Chain Reaction;

IgG: Immunoglobulin G;

IgM: Immunoglobulin M;

VDRL: Venereal Disease Research Laboratory;

mNGS: metagenomic Next-Generation Sequencing;

VCA: Viral Capsid Antigen;

EBNA: Epstein-Barr Nuclear Antigen;

EA-D: Early Antigen-Diffuse component.