

Severe Post-Measles Encephalitis in a Young Adult: Clinico-Biological Discordance and Neurocritical Care Challenges

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Abstract

Case Report

Post-measles encephalitis (PME) is a rare inflammatory demyelinating complication in young adults, whose global resurgence necessitates increased vigilance in intensive care settings. We report the case of an 18-year-old patient admitted for febrile encephalopathy (Glasgow Coma Scale [GCS] 9), complicated by status epilepticus and persistent akinetic mutism. Despite a marked systemic inflammatory response (CRP 89 mg/L, leukocytes 20,650/mm³), cerebrospinal fluid (CSF) analysis was strictly normal, illustrating a major clinico-biological discordance. Brain magnetic resonance imaging (MRI) was the key diagnostic modality, revealing diffuse cortical and subcortical T2/FLAIR hyperintensities suggestive of an ADEM-like pattern. This observation highlights that normal CSF findings should not exclude the diagnosis in the presence of critical neurological symptoms. Management relies on intensive neuroprotective strategies, strict seizure control, and early imaging, while vaccination remains the only effective preventive measure against these devastating forms.

Keywords: Measles, Neurocritical Care, Akinetic Mutism, Cerebrospinal Fluid, MRI, ADEM.

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1. INTRODUCTION

The global resurgence of measles, documented by the WHO and public health authorities since 2020, has re-exposed young adults to severe systemic complications [1]. Post-measles encephalitis (PME) typically occurs 1 to 14 days after rash onset and results from an immune-mediated demyelinating process rather than direct viral invasion of the brain parenchyma [2].

While pediatric forms are well described, adult presentations tend to be more severe, with over 15% of neurological complications requiring intensive care admission [3]. Prognosis depends on early neuroprotection and the management of associated multiorgan dysfunction [4, 5].

2. CASE PRESENTATION

An 18-year-old patient with a history of transient psychiatric disturbances (undocumented delusional episodes) was admitted to the intensive care unit for acute alteration of consciousness (initial GCS: 9), in the context of clinically and serologically confirmed measles.

On admission, laboratory findings showed a marked inflammatory syndrome (CRP 89 mg/L) and leukocytosis (20,650/mm³). Initial etiological workup excluded common co-infections (HIV, HBV, HCV, and syphilis).

Cerebrospinal fluid (CSF) analysis was paradoxically normal, with no pleocytosis or significant protein elevation. Brain MRI revealed diffuse cortical and subcortical hyperintense lesions on T2 and FLAIR sequences, without abnormal contrast enhancement.

Electroencephalography (EEG) showed generalized slowing with theta-delta activity, consistent with global cerebral dysfunction.

The clinical course was marked by generalized seizures progressing to status epilepticus, requiring orotracheal intubation, mechanical ventilation, and deep sedation (propofol and midazolam). Vasopressor support with norepinephrine was required for 48 hours.

After sedation withdrawal and successful extubation, the patient developed persistent akinetic mutism, characterized by spontaneous eye opening and

preserved visual tracking, but complete absence of motor and verbal initiation, despite a stabilized GCS of 13/15.

3. DISCUSSION

This case highlights the diagnostic and therapeutic challenges of PME in adults, a condition with complex pathophysiology requiring comparison with recent literature.

Epidemiology and Resurgence Context

Since 2024, a 50% increase in measles-related hospitalizations has been reported in certain regions, predominantly affecting unvaccinated or partially vaccinated young adults [1-10]. Compared to pediatric populations, adults present with more severe encephalitic forms, including higher rates of status epilepticus and respiratory complications [2-11].

This epidemiological trend necessitates reconsidering measles in the differential diagnosis of

acute encephalopathy in adults, even in the absence of meningeal signs [1-13].

The Cerebrospinal Fluid (CSF) Paradox

The most striking finding in our case is the normal CSF analysis. Recent literature (2020–2025) reports that 10–18% of imaging-confirmed inflammatory encephalitis cases may initially present with normal cytological and biochemical CSF findings [6-12].

This phenomenon is particularly described in PME, where inflammation predominantly affects perivascular white matter without associated meningeal involvement [12]. In neurocritical care, such clinico-biological discordance should never delay diagnosis or the initiation of aggressive treatment [5-13].

Comparison with Recent Neurological Literature (2020–2026)

Table 1: Clinical and paraclinical comparison between our case and recent literature

Characteristics	Our Case	Adult ADEM/PME Series (2020–2025)
Median age	18 years	22–35 years
Initial GCS	9	8–11
Normal CSF	Yes (100%)	12–18%
Seizure complication	Status epilepticus	30–45%
Akinetic mutism	Present	Rare (<5%)
Neurological sequelae	Persistent (mutism)	25% incomplete recovery

Post Measles encephalitis vs ADEM (Acute Disseminated Encephalomyelitis).

POST-MEASLES ENCEPHALITIS

T2WI

Diffuse cortical and subcortical hyperintensities

FLAIR

Diffuse cortical and subcortical hyperintensities

DWI (b1000)

No definite diffusion restriction

ADC

No low ADC

NO ABNORMAL ENHANCEMENT

T1WI + C

ADEM vs VIRAL ENCEPHALITIS: IMAGING CLUES

ADEM (Typical)

- Multifocal, bilateral, asymmetric
- Predominantly subcortical white matter
- Often spares cortex
- May show patchy enhancement
- Usually no diffusion restriction

VIRAL ENCEPHALITIS

- Often involves medial temporal lobes, insula, cingulate
- Cortical and subcortical
- DWI restriction common
- Usually no enhancement

Example

Example (HSV)

FLAIR

DWI (b1000)

IMPRESSION: Diffuse cortical and subcortical T2/FLAIR hyperintensities without diffusion restriction or abnormal enhancement. Imaging features are more in keeping with post-infectious (post-measles) encephalitis / ADEM spectrum. No evidence of viral encephalitis pattern.

Akinetic Mutism: Neuroanatomical Insights

Akinetic mutism observed in our patient is a rarely reported complication in PME. Functional neuroimaging studies suggest that this condition results from disruption of fronto-striatal circuits and the anterior cingulate cortex [14].

Unlike herpetic encephalitis, which primarily affects the temporal lobes, PME appears to induce diffuse dysfunction of neuronal networks involved in motivation and motor initiation. This may explain the slower neurological recovery compared to pediatric cases [14, 15].

Therapeutic Strategies and Neuroprotection

Management in the ICU focuses on strict control of secondary brain injury (SBIs). According to SFAR guidelines (2024), seizure control using titrated protocols (valproate, levetiracetam) is essential to limit neuronal damage [7, 8].

Regarding immunomodulation, although high-level evidence is lacking, early administration of intravenous immunoglobulins (IVIG) or high-dose corticosteroids is often recommended in ADEM-like presentations to reduce inflammatory cerebral edema [11-15].

4. CONCLUSION

Post-measles encephalitis in young adults is a devastating condition whose diagnosis may be obscured by deceptively normal CSF findings. This case underscores the importance of early MRI and multidisciplinary management in neurocritical care.

In the context of global measles resurgence, vaccination remains the only effective strategy to prevent these severe neurological complications.

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