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LETTER TO THE EDITOR

# When can antiseizure medications be safely withdrawn after encephalitis?

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Dear Editor,

Post-encephalitic epilepsy (PEE) remains one of the most important long-term neurological complications after encephalitis and may significantly affect long-term functional outcome and quality of life. Although the reported incidence varies according to etiology and follow-up duration, the long-term risk of epilepsy after encephalitis is consistently higher than in the general population [1-3]. Earlier population-based studies showed that the long-term risk of epilepsy after viral encephalitis was markedly higher in patients with early seizures during the acute phase [2]. More recent studies have linked later epilepsy risk to acute symptomatic seizures (ASyS), epileptiform electroencephalography (EEG) abnormalities, structural brain injury, and autoimmune etiologies, although the strength of these associations varies across cohorts [3,4].

Although several risk factors for PEE have been identified, their implications for antiseizure medication (ASM) management remain less clear. The central clinical question is no longer whether encephalitis increases epilepsy risk, but how clinicians should decide when to start, continue, and discontinue ASM treatment. In practice, ASM initiation after ASyS is common, particularly in patients with cortical injury, recurrent seizures, or epileptiform EEG findings. Status epilepticus and recurrent ASyS are generally accepted indications for treatment because both are associated with greater seizure burden and higher risk of subsequent epilepsy [3,4]. Uncertainty persists, however, in patients with isolated or brief ASyS who show apparent clinical recovery.

This uncertainty partly reflects evolving concepts of epileptogenesis. ASyS were traditionally regarded as transient events occurring during acute brain injury. Current models instead suggest that epilepsy develops through a process involving neuroinflammation, neuronal injury, synaptic reorganization, and progressive network hyperexcitability [5]. From this perspective, ASyS may represent an early manifestation of epileptogenesis rather than purely transient acute seizures. Still, the presence of ASyS does not necessarily mean that all patients require prolonged ASM treatment. The main difficulty is identifying patients with ongoing seizure risk while avoiding unnecessary long-term treatment in lower-risk individuals.

Although no standardized duration has been established for ASM treatment after encephalitis, several recent studies support shorter treatment durations in selected lower-risk patients. In the prospective PROSE study, median ASM duration was approximately 3 months, and longer treatment was not associated with lower relapse risk despite frequent prolonged ASM use in clinical practice [6]. Similarly, Dhawan et al. reported comparable seizure recurrence rates between 4-week and 12-week ASM regimens in children with acute encephalitis syndrome [7]. Routine prolonged ASM treatment after encephalitis may therefore be unnecessary in many patients, although evidence specifically addressing ASM withdrawal after

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encephalitis remains limited. Prolonged ASM exposure may also contribute to unnecessary adverse effects and treatment burden in lower-risk patients.

Still, these observations should not be generalized to all patients. Persistent epileptiform EEG abnormalities, temporal lobe injury, autoimmune encephalitis, recurrent ASyS, and status epilepticus may argue against early ASM withdrawal and support closer follow-up in selected higher-risk individuals. Rather than relying on arbitrary treatment durations alone, ASM withdrawal after encephalitis may depend more on follow-up EEG findings, structural imaging, and overall clinical course (Table 1).

Acute epileptiform abnormalities during encephalitis do not necessarily predict persistent seizure risk, and follow-up EEG may therefore be more informative for ASM withdrawal decisions. Sivaraju et al. demonstrated that persistent epileptiform abnormalities on repeat EEG were strongly associated with later epilepsy development, whereas resolution of EEG abnormalities was associated with lower long-term seizure risk [8]. Routine acute-phase EEG recordings may not fully reflect later seizure risk or evolving cortical irritability.

Neuroimaging findings may further refine this decision-making process. Structural brain injury, particularly when involving cortical or temporal regions, is generally considered a higher-risk feature after encephalitis, although reported predictors vary across studies [3,6]. Ongoing cognitive symptoms, fluctuating behavioral changes, or episodic neurological complaints may require closer EEG follow-up before ASM discontinuation.

Autoimmune encephalitis deserves particular consideration because seizure evolution may differ substantially from that observed in infectious encephalitis. In a recent long-term study, Jia et al. reported that nearly one-third of autoimmune encephalitis patients with acute seizures developed treatment-resistant PEE during follow-up [9]. Epileptiform discharges during the subacute phase were among the strongest predictors of later epilepsy development. Not all autoimmune encephalitis subtypes appear to behave similarly. Patients with leucine-rich glioma-inactivated 1 (LGI1) or N-methyl-D-aspartate receptor (NMDAR) encephalitis may become seizure-free after immunotherapy and may not always require chronic ASM treatment, whereas delayed immunotherapy, ongoing inflammatory activity or structural temporal injury may support prolonged treatment and closer follow-up in selected patients [4,9].

A uniform ASM strategy after encephalitis may be difficult to justify. Patients with isolated brief ASyS, normal follow-up EEG findings, absence of cortical lesions, and rapid neurological recovery may be candidates for earlier ASM withdrawal after reassessment, often within the first few months. In contrast, recurrent seizures, status epilepticus, persistent epileptiform EEG abnormalities, autoimmune encephalitis, and temporal or cortical injury may argue against early ASM withdrawal. In practice, ASM withdrawal decisions may be better guided by follow-up EEG findings, structural imaging, and clinical course rather than by predetermined treatment durations alone.

**Table 1.** A practical approach to ASM withdrawal decisions after encephalitis.

| Clinical domain | Lower-risk features | Higher-risk features | Possible implication for ASM withdrawal |
|---|---|---|---|
| Acute symptomatic seizures (ASyS) | Single brief seizure | Recurrent seizures or status epilepticus | Recurrent seizures may argue against early withdrawal |
| Follow-up EEG | Resolution of epileptiform abnormalities | Persistent epileptiform abnormalities | Persistent abnormalities may support prolonged treatment or closer follow-up |
| Neuroimaging | No cortical or temporal injury | Cortical, limbic, or mesiotemporal lesions | Structural injury may favor delayed withdrawal |
| Etiology | Self-limited infectious encephalitis | Autoimmune encephalitis or ongoing inflammatory activity | Withdrawal decisions may require individualized reassessment |
| Clinical course | Stable neurological recovery | Persistent cognitive/ behavioral symptoms | Repeat EEG evaluation may be helpful before withdrawal |

Abbreviations: ASM: antiseizure medication; ASyS: acute symptomatic seizures; EEG, electroencephalography.

After encephalitis, the key clinical question is often not whether ASM should be started, but when it can be safely discontinued. Follow-up EEG, structural imaging, seizure characteristics, and etiology may all help guide these decisions. A more individualized approach may help reduce unnecessary long-term ASM use while supporting closer monitoring in higher-risk patients. Prospective studies specifically addressing ASM withdrawal after encephalitis are still needed.

## Author contributions

Conception and design: O.S.; Data acquisition: O.S.; Data analysis: O.S.; Data interpretation: O.S.; Drafting of the manuscript: O.S.; Critical revision of the manuscript: O.S. The author reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

## Conflict of interest

The author declares that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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## Generative AI statement

The author declares that during the preparation of this study, the following AI-assisted technology was used: ChatGPT (OpenAI) on May 2026. Extent of Use: ChatGPT (OpenAI) was used in May 2026 solely for language editing and improving the clarity of the manuscript. The scientific content, interpretation, and conclusions were developed by the author. The author confirms that he/she has critically reviewed and edited any AI-generated content and takes full responsibility for the integrity, accuracy, and originality of the publication. The author certifies that the original human contribution is maintained and that AI-assisted tools are not listed or cited as authors.

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