MRI in Diagnosis of Autoimmune Encephalitis

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See the slide presentation here.

The pathophysiology of autoimmune encephalitis (AE) revolves around the production of specific autoantibodies against central nervous system targets. This immune response triggers inflammation, leading to damage of the targeted structure. In this sense, the types of AE can be divided into those with antibodies against intracellular antigens (group I) and those with antibodies against extracellular antigens (group II). This classification has clinical significance, as it influences treatment responses and exhibits associations with malignancy based on the encephalitis group, thereby providing valuable prognostic insights. Another classification method involves using an anatomic approach, with AE categorized on the basis of the affected central nervous system region. This includes limbic, cortical and subcortical, striatal, diencephalic, brainstem, cerebellar, encephalomyelitis, meningoencephalitis, and combined manifestations. Furthermore, AE can be classified etiologically as idiopathic, paraneoplastic, or postinfectious.

With AE, there is significant variability and overlap in imaging and clinical manifestations, but the clinical and imaging findings are usually determined on the basis of the location of the central nervous system structure targeted by the autoantibody. Clinical features may include alteration in behavior, psychosis, seizures, memory impairment, cognitive deficits, atypical movements, dysautonomia, and reduced consciousness. In the evaluation of AE, neuroimaging emerges as a pivotal player, contributing to both the diagnosis and the differential diagnosis of AE.

MRI stands out as the imaging modality of choice when AE is suspected. A comprehensive MRI protocol encompasses T1- and T2-weighted, fluid-attenuated inversion-recovery (FLAIR), and diffusion-weighted imaging sequences. Depending on the clinical scenario, postcontrast T1-weighted as well as perfusion-weighted sequences may be added. These imaging sequences enable the identification of abnormalities in the limbic structures, striatum, diencephalon, and romboencephalon. An important benefit is that they aid in distinguishing AE mimics such as herpes-related encephalitis, posterior reversible encephalopathy syndrome, various toxic and metabolic disorders, and tumors, among others, based on distinctive imaging patterns.

Despite recent advances in autoantibody detection and MRI technology, diagnosis of AE remains a challenge. There is a potential risk of false-positive autoantibody results among symptomatic patients, contributing to the risk of misdiagnosis. To enhance sensitivity and specificity, a broad differential diagnosis is imperative. The differential diagnosis should encompass considerations for toxic or metabolic encephalopathies; functional neurologic disorders; primary psychiatric diseases; neurodegenerative disorders; neoplasms; non–immune-mediated seizure disorders; cognitive syndromes linked to fibromyalgia, chronic fatigue, sleep disorders, and medication-related issues; and notably, infectious disorders.

The accompanying slide presentation is intended to serve as a comprehensive and practical reference for guiding the diagnosis of AE. It addresses essential aspects related to AE, including pathophysiology; classification; clinical presentations; radiologic findings, such as those seen in Figures 1 and 2; and differential diagnosis based on symptoms and imaging patterns. The cases presented reinforce key concepts.

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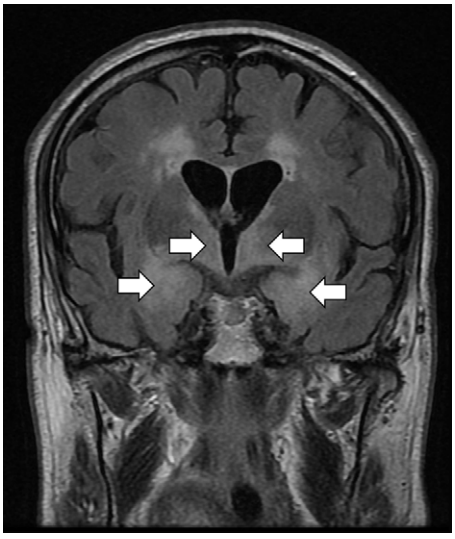

Figure 1. Known anti-Ma1 encephalitis in an 81-year-old patient with high-grade undifferentiated bladder carcinoma. Axial T2-weighted FLAIR brain MR image shows bilateral increased signal intensity with a tumefactive appearance and involving both hippocampi and thalami (arrows).

Slide Presentation

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Abbreviations: AE = autoimmune encephalitis, FLAIR = fluid-attenuated inversion recovery

TEACHING POINTS

- The pathophysiology of autoimmune encephalitis (AE) revolves around the production of specific autoantibodies against central nervous system targets. This immune response triggers inflammation, leading to damage of the targeted structure.
- In the evaluation of AE, neuroimaging emerges as a pivotal player, contributing to both the diagnosis and the differential diagnosis of AE.
- MRI stands out as the imaging modality of choice when AE is suspected. A comprehensive MRI protocol encompasses T1- and T2-weighted, fluid-attenuated inversion-recovery, and diffusion-weighted imaging sequences. Depending on the clinical scenario, postcontrast T1-weighted as well as perfusion-weighted sequences may be added. These imaging sequences enable the identification of abnormalities in limbic structures, the striatum, the diencephalon, and the romboencephalon.

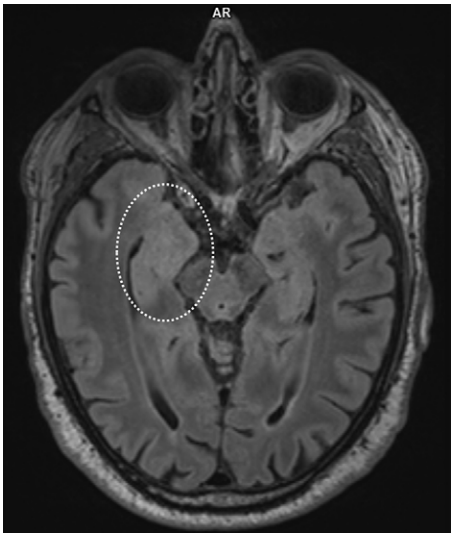

Figure 2. Known tumefactive active leucine-rich glioma-inactivated-1 AE in a 68-year-old man. Axial T2-weighted FLAIR MR image shows a very subtle increase in T2 FLAIR signal intensity and thickening of the right amygdala and hippocampal head (oval outline).

Suggested Readings

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