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The role of brain MRI in autoimmune encephalitis

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Purpose of review
Brain MRI is central to the diagnostic work-up of autoimmune encephalitis (AE). This review summarizes established patterns on routine MRI sequences across antibody-defined AE types and highlights emerging quantitative and network-based approaches aimed at improving sensitivity, phenotypic stratification, and prognostication.

Recent findings
Conventional MRI reveals syndrome-associated patterns that can support diagnosis in several AE types, including medial temporal involvement in LGI1, CASPR2, and GABA(B) receptor encephalitis; multifocal cortico-subcortical lesions in GABA(A) receptor encephalitis, and radial perivascular enhancement in GFAP astrocytopathy. Longitudinal studies show that regional and global atrophy better reflect cumulative injury and clinical outcome than acute lesions in entities such as NMDA receptor encephalitis or IgLON5 antibody-mediated disease. Basic quantitative measures derived from routine sequences can detect microstructural abnormalities in normal-appearing tissue. Advanced techniques including quantitative MRI, diffusion tensor imaging, and resting-state functional MRI demonstrate widespread structural and functional network disruption beyond visible lesions. Translational animal studies further link imaging changes to antibody-mediated synaptic dysfunction.

Summary
Conventional MRI remains indispensable for differential diagnosis and pattern recognition in AE. Pragmatic approaches such as volumetry and basic quantitative metrics are closest to clinical implementation, whereas advanced techniques require further standardization and validation. Together, these developments position MRI to evolve from a primarily diagnostic tool toward precision phenotyping, prognosis, and longitudinal monitoring in AE.

Keywords
autoimmune diseases of the nervous system, encephalitis, magnetic resonance imaging, review

THE ROLE OF MRI IN AUTOIMMUNE ENCEPHALITIS

Brain MRI is a central component of the diagnostic work-up in autoimmune encephalitis (AE). In routine practice, MRI can facilitate correct diagnosis of AE by revealing characteristic patterns of inflammatory involvement. At the same time, several AE subtypes, most notably NMDAR encephalitis, often present with normal or only nonspecific findings on conventional MRI, even in clinically severe disease. Therefore, understanding the spectrum of expected imaging patterns across AE subtypes is essential.

In recent years, growing clinical and radiological experience and larger, better phenotyped cohorts have sharpened entity-specific pattern recognition and clarified the temporal evolution of imaging abnormalities (Table 1). For multiple AE types, characteristic regional lesion sites are now increasingly recognized, and longitudinal studies link acute lesion burden, residual structural damage, and progressive atrophy to clinically relevant outcomes such as cognitive impairment and long-term disability.

In parallel, MRI methodology has moved beyond qualitative lesion detection. Quantitative structural imaging, advanced diffusion techniques, and structural and functional network analyses can reveal

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KEY POINTS

- Routine MRI provides syndrome-specific diagnostic patterns in several antibody-defined autoimmune encephalitis (AE) entities but can remain normal or nonspecific in clinically severe disease, particularly in NMDAR encephalitis.
- Longitudinal MRI, especially volumetry, captures cumulative injury and outcome-relevant trajectories more reliably than acute lesion burden in multiple AE subtypes.
- Basic quantitative metrics derived from standard sequences, such as the T1w/T2w ratio, can detect microstructural abnormalities in normal-appearing tissue and add clinically scalable information.
- Diffusion connectomics and resting-state fMRI reveal widespread structural and functional network disruption beyond visible lesions, supporting a network-level disease concept in AE.
- Translational animal models reproduce antibody-associated connectivity changes, strengthening the biological validity of emerging MRI markers for monitoring and trials.

microstructural damage and network-level disruption in tissue that appears normal on conventional scans. These techniques extend MRI from identifying focal lesions to quantifying subtle diffuse brain damage and network dysfunction, and they hold promise as imaging biomarkers for prognosis and longitudinal monitoring.

In this review, we summarize recent advances in MRI across antibody-defined AE syndromes, discuss clinical applicability and methodological challenges, and highlight promising techniques for routine implementation and for use as endpoints in clinical trials.

ROUTINE CLINICAL IMAGING

NMDAR encephalitis

NMDAR encephalitis typically presents with a viral-like prodrome followed by prominent psychiatric and behavioral symptoms, memory impairment, seizures, movement disorders, autonomic dysfunction, and reduced consciousness, with frequent persistent cognitive and neuropsychiatric sequelae [1]. Routine MRI is normal in more than half of patients at symptom onset. When abnormalities are present, they are usually subtle and nonspecific, most commonly small T2/FLAIR hyperintensities in cortical or subcortical regions, occasionally involving deep gray matter, brainstem, or cerebellum (Fig. 1a). Contrast enhancement is uncommon. Lesion burden correlates poorly with clinical severity, and severe disease can occur despite unremarkable imaging.

A recent study reported a substantial prevalence (~11.6%) of patients with an NMDAR encephalitis – multiple sclerosis (MS) overlap syndrome in the German autoimmune encephalitis registry GENERATE [2▪▪]. Overlap patients fulfilled the diagnostic criteria for both disorders and showed CSF profiles compatible with combined NMDAR encephalitis and MS immunopathology, including amplified intrathecal IgG/IgM synthesis and polyspecific antiviral responses. On MRI, lesion distribution and morphology were MS-typical, with a very high proportion of central vein sign (CVS)-positive lesions, a feature regarded as highly specific for MS. Importantly, in

Table 1. Typical patterns on routine MRI for the most common autoimmune encephalitis types

AE type	Typical MRI pattern
NMDAR encephalitis	MRI normal or small unspecific lesions
LGI1 encephalitis	FBDS stage: MRI normal in most cases, unilateral T2/FLAIR hyperintensity in the basal ganglia reported in up to 30%; Limbic encephalitis: bilateral medial temporal lobe T2/FLAIR hyperintensity
CASPR2 antibody-mediated disease	Limbic encephalitis: bilateral medial temporal lobe T2/FLAIR hyperintensity; Morvan syndrome or neuromyotonia: MRI mostly normal
GAD65 antibody-mediated disease	Limbic encephalitis: bilateral medial temporal lobe T2/FLAIR hyperintensity; Cerebellar phenotype: cerebellar atrophy; Stiff person syndrome: MRI normal
GABA(B)R encephalitis	Bilateral medial temporal lobe T2/FLAIR hyperintensity
GABA(A)R encephalitis	Multifocal, confluent cortico-subcortical T2/FLAIR hyperintensities
GFAP astrocytopathy	Predominantly periventricular T2/FLAIR hyperintensities; in 40-50% of cases linear, radial perivascular contrast enhancement
IgLON5 antibody-mediated disease	Atrophy in brainstem, hypothalamus, basal ganglia, and nucleus accumbens

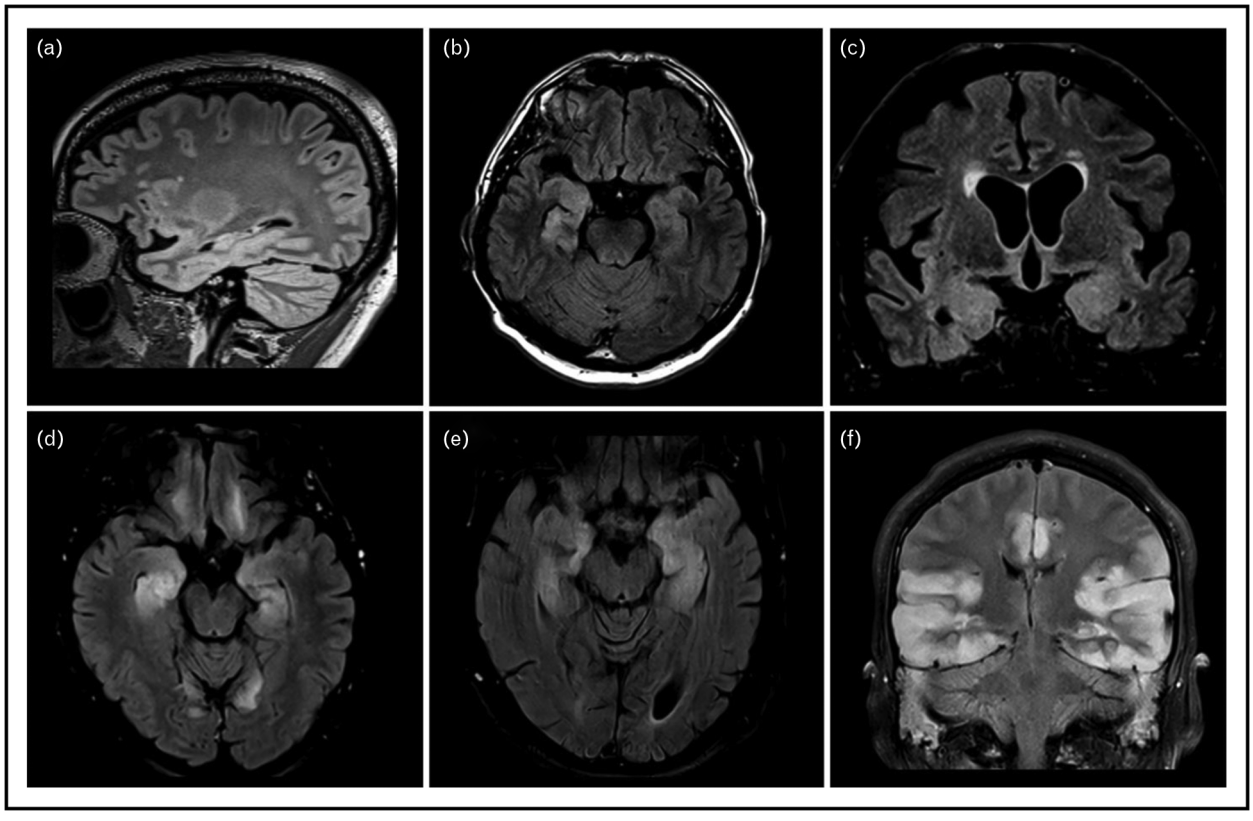

FIGURE 1. (a) A patient with NMDA receptor encephalitis, with small and nonspecific T2-FLAIR hyperintense lesions in subcortical regions. (b) A patient with LGI1 encephalitis in the limbic encephalitis stage and bilateral medial temporal hyperintensity in T2-FLAIR. (c) A patient with CASPR2 encephalitis and bilateral T2-FLAIR medial temporal hyperintensity and swelling. (d) GAD65 encephalitis patient with T2-FLAIR hyperintense lesions in the medial temporal lobe bilaterally. (e) Patient with GABA(B) receptor encephalitis and bilateral medial temporal hyperintense T2-FLAIR lesions. (f) A patient with GABA(A) receptor encephalitis showing multifocal cortical and subcortical T2-FLAIR hyperintense lesions involving the frontal and temporal lobes, with associated cortical swelling (image courtesy of Prof. Markus Kraemer, Department of Neurology, Alfried Krupp Hospital, Essen, Germany).

comparison to a control group of RRMS patients, overlap patients less frequently received long-term maintenance immunotherapy and exhibited faster T2 lesion accumulation, highlighting the need for adequate maintenance immunotherapy and MRI monitoring in these patients. Another recent study reported MS-like lesions in 5.5% of NMDAR encephalitis patients and extensive FLAIR lesions in 1.9% of patients, and MS-like lesions were associated with poorer long-term outcomes [3▪▪]. Beyond NMDAR encephalitis, overlap constellations between demyelinating disease and autoimmune encephalitis have also been described, including a recent case with sequential transitions from infectious encephalitis to seronegative autoimmune encephalitis and subsequently to MS [4]

Pediatric disease shows a similar frequency of MRI abnormalities [5,6]. Volumetric analyses have demonstrated reduced whole brain volumes as well as lower hippocampal and basal ganglia volumes, and longitudinal data indicate a failure of age-expected brain growth in children with NMDAR encephalitis, suggesting a particularly detrimental effect of the disease on the developing brain [5]. Furthermore, an abnormal brain MRI (e.g., T2/FLAIR hyperintense lesions or focal atrophy) has been associated with poorer long-term outcomes in two independent studies [5,6].

Longitudinal analyses identified some patients with reversible global atrophy [7]. This finding may, however, reflect critical illness and intensive care unit related factors such as fluid balance shifts, medication effects, prolonged immobilization, and critical illness-associated encephalopathy, rather than NMDAR encephalitis-specific neurobiology [8]. In contrast, cerebellar atrophy has been shown to be irreversible or even progressive and has been associated with poor functional outcome [9].

The prognostic value of conventional MRI currently remains uncertain. While abnormal MRI findings are associated with poorer long-term outcome in children [5,6], evidence in adults is mixed. Abnormal

baseline MRI contributed to the composite score NEOS and was associated with worse one-year outcome [10], whereas MRI was not retained as an independent predictor for outcome in the updated NEOS2 model derived from a larger multinational cohort ($n=702$) [11]. However, dichotomizing MRI as normal versus abnormal likely obscures clinically meaningful information, and pattern-based interpretation that considers lesion topography, limbic versus extralimbic involvement, and longitudinal evolution is expected to be more informative for risk stratification.

LGI1 encephalitis

Autoimmune encephalitides (LGI1) encephalitis typically presents with faciobrachial dystonic seizures (FBDS), often followed by limbic encephalitis with memory impairment, seizures, and behavioral abnormalities; early immunotherapy during the FBDS stage can prevent progression [12]. Hyponatremia, sleep disturbance, and persistent cognitive deficits, particularly affecting memory and executive function, are common long-term sequelae [13–15].

MRI findings are stage dependent. During isolated FBDS, routine MRI is frequently normal, although contralateral T1 and/or T2 hyperintensities in the basal ganglia have been reported in some patients [16,17]. With established limbic encephalitis, approximately 50–70% show unilateral or bilateral medial temporal lobe (MTL) T2/FLAIR hyperintensities (Fig. 1b). Abnormalities are typically restricted to the hippocampus and amygdala and show no diffusion restriction or contrast enhancement [18▪▪]. Longitudinally, most patients develop hippocampal atrophy regardless of initial MRI findings, and the extent of MTL damage correlates with memory outcome and epilepsy risk [14,19]. Relapses occur in approximately 10–15% of patients. A recent study reported MRI abnormalities in ~60% of patients with relapses, most commonly persistent MTL T2/FLAIR hyperintensities, while ~10% showed newly developed MTL changes [20▪].

CASPR2 encephalitis

CASPR2 antibody-mediated disease encompasses heterogeneous clinical phenotypes including limbic encephalitis, Morvan syndrome, and neuromyotonia [21]. Limbic encephalitis typically presents with memory impairment, confusion, behavioral changes, and temporal lobe seizures, resembling other forms of autoimmune limbic encephalitis. In contrast, Morvan syndrome is characterized by severe autonomic dysfunction, including hyperhidrosis and hemodynamic instability, together with profound insomnia, peripheral nerve hyperexcitability (often neuromyotonia), and a prominent behavioral syndrome. Psychiatric manifestations such as hallucinations, agitation, and delusions are common. Although these phenotypes can be clinically distinct, overlapping central and peripheral nervous system involvement is frequently observed in CASPR2 antibody-associated disease [22].

MRI abnormalities correlate with clinical phenotype. Patients with isolated neuromyotonia or Morvan syndrome often have normal brain MRI, whereas those with limbic encephalitis can show unilateral or bilateral mesial temporal T2/FLAIR hyperintensities that may evolve into hippocampal atrophy on follow-up (Fig. 1c) (Bien *et al.*, 2017) [23]. This clinicoradiological association is supported by systematic analyses demonstrating that structural abnormalities largely segregate with limbic clinical presentations [24]. Similarly to LGI1 encephalitis, T2/FLAIR hyperintensities are restricted to the MTL and show no diffusion restriction or contrast enhancement [18▪▪]. Overall, MRI in CASPR2 disease primarily supports the identification of a limbic phenotype and exclusion of mimics, while normal imaging remains common in nonlimbic presentations.

GAD65 antibody-mediated disease

GAD65 antibody-mediated disease comprises partially overlapping phenotypes including limbic encephalitis, stiff-person syndrome, and cerebellar ataxia [25]. MRI findings vary by phenotype. Limbic presentations can show MTL T2/FLAIR hyperintensity with swelling, particularly involving the hippocampus and amygdala (Fig. 1d) [26–28]. Swelling often regresses over time but frequently evolves into hippocampal sclerosis and persistent MTL atrophy, paralleling chronic epilepsy and cognitive impairment. Longitudinal imaging-pathology correlations further support this evolution: an early inflammatory stage characterized by medial temporal edema progresses to established sclerosis, with histology demonstrating $CD8^+$ T-cell-mediated neuronal injury, supporting MRI as a marker of active immune damage preceding irreversible atrophy [29]. In a recent Chinese cohort, about 70% of patients had limbic T2/FLAIR hyperintensities, while 10% showed combined limbic and extra-limbic abnormalities [30]. In contrast, cerebellar phenotypes can show isolated cerebellar atrophy, while no consistent brain MRI signature has been described for stiff-person syndrome [31–33].

GABA(B)R encephalitis

GABA(B)R encephalitis most commonly manifests as limbic encephalitis with prominent seizures, memory impairment, and behavioral change, while cerebellar

or brainstem involvement is less frequent [34]. MRI typically shows a classic limbic pattern, with unilateral or bilateral mesial temporal T2/FLAIR hyperintensities in the majority of patients (Fig. 1e). In a recent cohort, medial temporal MRI abnormalities were observed in approximately two-thirds of cases, whereas one-third had unremarkable imaging [35]. Less commonly, cerebellar or brainstem signal changes may accompany ataxic or brainstem phenotypes. Imaging abnormalities often improve with treatment but may evolve into residual medial temporal atrophy [36].

GABA(A)R encephalitis

GABA(A)R encephalitis is characterized by an acute encephalopathy with cognitive dysfunction, disorientation, and memory impairment, frequently accompanied by refractory seizures or status epilepticus, and additional psychiatric symptoms such as psychosis, depression, or mutism [37,38].

MRI is abnormal in most patients and often highly characteristic. The typical pattern consists of multifocal, often confluent T2/FLAIR hyperintensities involving cortical gray matter and adjacent subcortical white matter, resulting in widespread cortico-subcortical lesions (Fig. 1f). Lesions are frequently bilateral and dynamic, commonly with absent or only limited diffusion restriction and contrast enhancement, and may migrate or fluctuate over time [37,38]. Early serologic cohorts demonstrated that low-titer or nonsubunit-specific antibodies may be associated with normal or nonspecific imaging, whereas high-titer IgG antibodies correlate with the inflammatory phenotype [39]. With treatment, lesions may partially resolve, although regional or generalized atrophy can develop in previously affected areas.

More recently, systematic lesion mapping showed a predominantly supratentorial distribution with limbic, frontal, and temporal predominance and consistent bilateral cingulate involvement, and indicated that lesion burden stratifies disease severity, with extensive confluent patterns associated with poorer outcomes [40]. In addition, an early diffusion-based "split ADC sign", defined as cortical diffusion restriction with facilitated subcortical diffusion, has been described as a potential marker of acute inflammatory cortical injury and was observed in GABA(A)R encephalitis, albeit not specific to this disease [41]. Overall, GABA(A)R encephalitis is among the most consistently MRI-positive AE subtypes, with a distinctive and clinically informative imaging signature.

GFAP astrocytopathy

GFAP astrocytopathy is associated with a broad clinical spectrum including encephalopathy, myelitis, optic neuritis, ataxia and psychiatric symptoms, frequently accompanied by systemic features such as fever and headache and, in severe cases, impaired consciousness or seizures [42]. On brain MRI, patients often show extensive, poorly demarcated T2/FLAIR hyperintensities with a predominately periventricular distribution, frequently extending to the brainstem, diencephalon, cerebellum, corpus callosum and optic nerves [43]. A characteristic imaging hallmark is linear, radial perivascular contrast enhancement, reported in approximately half of cases and often accompanied by leptomeningeal enhancement. These enhancement patterns are highly suggestive and can help distinguish GFAP astrocytopathy from other inflammatory or demyelinating disorders [43]. Spinal MRI is abnormal in 50–70% of patients and commonly demonstrates longitudinally extensive transverse myelitis, often involving long segments of the thoracic cord or, in some cases, the entire spinal cord [44]. Consistently, a recent large multicenter cohort reported abnormal brain MRI in about two-thirds of patients, most often with multifocal T2/FLAIR lesions and frequent leptomeningeal or radial perivascular enhancement, while spinal cord involvement and longitudinally extensive myelitis were also common, with most abnormalities improving on follow-up [45▪]. However, imaging findings can be heterogeneous. Individual reports describe marked hippocampal and global subcortical atrophy together with longitudinal spinal cord involvement despite the absence of typical cerebral T2 lesions, indicating that atypical or predominantly degenerative-appearing patterns may also occur [46].

IgLON5 antibody-mediated disease

IgLON5 antibody-mediated disease presents with a broad and heterogeneous clinical spectrum including sleep disturbance, bulbar and respiratory dysfunction, movement disorders, neuromuscular involvement, cognitive impairment, and ocular motor abnormalities [47–49], and shows a strong immunogenetic association with HLA-DQ haplotypes, particularly HLA-DQB1*05 [50].

Conventional MRI is frequently normal in cohort studies [47,51]. When abnormalities are present, findings are heterogeneous and mostly described in single case reports, including leptomeningeal inflammation, focal inflammatory lesions with brainstem or hippocampal atrophy, hypothalamic or hippocampal signal changes, and reversible cerebellar or brainstem T2/FLAIR hyperintensities [52–58]. Additional patterns include bilateral trigeminal nerve enlargement with symmetric brainstem signal abnormalities in bulbar-onset motor neuron

disease-like phenotypes and occasional diffusion restriction in brainstem and cerebellum [52,59]. A recent tau PET study in four patients reported increased tracer binding in the pons, medulla and cerebellum [60]. Early studies also noted regional atrophy of the midbrain, brainstem, hippocampus, or cerebellum, but were limited to small series [47,51].

More recently, a systematic multicenter analysis of 127 patients demonstrated a consistent atrophy pattern with preferential involvement of the brainstem, hypothalamus, basal ganglia, and nucleus accumbens, accompanied by ventricular enlargement and progressive brainstem volume loss over time; regional atrophy correlated with clinical phenotypes, linking basal ganglia involvement to movement disorders and hippocampal or thalamic atrophy to cognitive impairment [61▪▪]. Together, these findings establish volumetric MRI as a sensitive marker of disease burden and anatomical predilection sites, whereas routine structural imaging frequently remains unrevealing.

EMERGING MRI METHODS AND IMAGING MARKERS

Microstructural imaging

Basic quantitative MRI markers can detect microstructural abnormalities despite normal-appearing conventional imaging. The T1w/T2w ratio, which can be derived from routine structural sequences, shows widespread reductions in normal-appearing white matter and deep gray matter in NMDAR encephalitis, including the hippocampus, amygdala, and thalamus, and these changes correlate with verbal memory impairment [62].

Multiparameter mapping (MPM) provides more comprehensive whole-brain measures sensitive to myelin, iron, and water content (PD, MTsat, R1, R2*). Modern accelerated protocols enable acquisition within minutes and have demonstrated good stability and cross-site reproducibility, with increasing use in clinical research of neuroimmunological diseases such as MS [63–65]. Although not yet systematically studied in autoimmune encephalitis, these properties make MPM a promising candidate for detecting subtle inflammatory or degenerative alterations and for longitudinal monitoring.

Diffusion tractography and graph-theoretical analyses further indicate that structural damage in autoimmune encephalitis extends beyond focal lesions. In NMDAR encephalitis, diffusion-based structural connectome analyses in the postacute stage indicate relatively preserved global network topology but focal medial temporal network alterations consistent with hippocampal hub failure, with reduced hippocampal node strength correlating with verbal long-term memory impairment and paralleling hippocampal volume loss [66]. In contrast, LGI1 encephalitis is associated with reduced whole-brain connectivity and widespread hypoconnectivity that relates to persistent deficits in memory, attention, and executive function, suggesting that cognitive impairment reflects distributed network disconnection rather than isolated hippocampal injury [67▪].

Resting-state functional MRI

Resting-state fMRI has provided early systems-level evidence of network dysfunction in NMDAR encephalitis. Even with largely normal structural MRI, patients show reduced hippocampal connectivity to medial prefrontal and default-mode regions, correlating with memory deficits and disease severity [68,69]. More recently, time-resolved and dynamic functional connectivity approaches have extended this framework by capturing fluctuations of network organization on the scale of seconds, revealing unstable and more frequent transitions between functional brain states, indicating reduced network stability that relates to clinical severity [70,71].

Complementary voxel-wise dynamic eigenvector centrality mapping has shown increased temporal variability of nodal importance across frontotemporal and default-mode regions, with greater instability associated with worse memory performance [72]. Notably, these effects were not apparent with conventional static connectivity metrics. Finally, animal models replicate hippocampal network disconnection after exposure to NMDAR antibodies, linking functional MRI changes to antibody-mediated synaptic pathology and supporting the biological validity of these imaging markers [73]. In a CASPR2 monoclonal antibody infusion mouse model, resting-state fMRI revealed markedly reduced functional connectivity across multiple regions, including bilateral hippocampus as well as caudate, putamen, hypothalamus, and neocortical areas, despite the absence of structural MRI changes [74].

DISCUSSION AND CRITICAL APPRAISAL

The evolving role of MRI in autoimmune encephalitis

Brain MRI remains the cornerstone imaging modality in autoimmune encephalitis. At disease onset, it identifies overt inflammatory changes and excludes important mimics. In several antibody-defined syndromes, characteristic topographic patterns already

provide positive diagnostic clues, making MRI a pattern-recognition tool rather than a purely exclusionary test. However, many patients, particularly with NMDAR encephalitis, have normal or only subtle MRI abnormalities despite severe symptoms. This clinico-radiological dissociation is consistent with AE often causing distributed synaptic dysfunction and network-level disturbance or diffuse injury rather than focal macroscopic lesions, limiting the sensitivity of conventional imaging.

Longitudinal imaging indicates that atrophy often reflects cumulative disease burden more reliably than acute lesions. Hippocampal and MTL atrophy in limbic syndromes, cerebellar volume loss in selected phenotypes, and more diffuse brain atrophy in NMDAR encephalitis consistently correlate with cognitive outcome, epilepsy risk, and functional disability. Accordingly, volumetry is emerging as a pragmatic marker of cumulative injury and recovery.

Emerging techniques and future directions

Quantitative metrics derived from routine sequences such as the T1w/T2w ratio can detect microstructural abnormalities in normal-appearing tissue and complement visual assessment without requiring specialized acquisition protocols. Similarly, structural complexity analysis of anatomical MRI data has emerged as a sensitive new tool for brain morphometry that can be applied to standard T1-weighted images [75,76]. This technique was recently applied in AE and has shown high sensitivity in capturing structural brain changes and neuropsychiatric outcomes of NMDAR encephalitis [77▪▪]. Further methods extend MRI from lesion detection toward network-level characterization. Diffusion MRI with tractography and connectomics demonstrates widespread structural disconnection beyond visible lesions, whereas resting-state fMRI reveals disrupted large-scale functional organization and altered temporal dynamics. Translational animal models showing comparable connectivity changes strengthen the biological validity of these markers. Nevertheless, most advanced techniques remain primarily research tools, given requirements for longer acquisition times, specialized processing pipelines, and standardization. At present, automated volumetry and basic metrics such as the T1w/T2w ratio are closest to routine implementation, whereas diffusion and functional MRI will require further protocol simplification and cross-site reproducibility. Key priorities include multicenter harmonization, prospective longitudinal studies to assess treatment responsiveness and relapse, and validation of imaging markers as prognostic tools or trial endpoints, ideally integrated with clinical and immunological data.

CONCLUSION

Conventional MRI remains central in AE and, when interpreted in an entity-specific and longitudinal manner, provides meaningful diagnostic and prognostic information. At the same time, AE is increasingly understood as a network disorder, explaining the frequent mismatch between symptoms and visible lesions. Quantitative microstructural and connectivity-based approaches provide important complementary information and may enable precision phenotyping, monitoring, and outcome prediction with ongoing standardization and clinical validation.

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Conflicts of interest

There are no conflicts of interest.

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Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

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Nationwide data from the GENERATE network identified an NMDARE-MS overlap in 11.6% (22/189) of definite NMDAR encephalitis patients, characterized by an MS-typical MRI lesion pattern and markedly accelerated T2 lesion accumulation over five years, while 7T MRI demonstrated MS-specific lesion morphology with a high proportion of central vein sign-positive lesions in overlap patients comparable to RRMS.

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LGI1 and CASPR2 antibody encephalitis are characterized by T2/FLAIR hyperintensities largely confined to the medial temporal lobes, with absent diffusion restriction and only rare contrast enhancement. This MRI pattern helps distinguish LGI1 and CASPR2 encephalitis from mimics such as viral encephalitis and Creutzfeldt–Jakob disease.

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Brain MRI is abnormal in about 60% of relapse episodes in LGI1 encephalitis, mostly showing persistent MTL abnormalities, but also novel MTL changes in about 10%.

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