

Peri-ictal magnetic resonance imaging findings in pediatric seizures: a scoping review on pearls and pitfalls from a heterogeneous population

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Background: Peri-ictal magnetic resonance imaging (MRI) brain abnormalities are increasingly recognized, yet their temporal evolution and imaging features in children remain incompletely characterized. This scoping review aims to map and synthesize two decades of evidence on MRI-based seizure-related changes in pediatric patients, providing an overview of the existing literature, reported imaging findings, diagnostic challenges, and knowledge gaps.

Methods: PubMed and MEDLINE databases were searched (January 2000 to September 2025) for studies evaluating MRI changes in focal seizures (FS), prolonged febrile seizures (PFS), or status epilepticus (SE) in children aged 0-18 years. Studies reporting diffusion weighted images/apparent diffusion coefficient maps (DWI/ADC), T2/fluid attenuated inversion recovery (FLAIR), arterial spin labelling (ASL), functional MRI (fMRI), or diffusion tensor imaging (DTI) changes were considered.

Results: Twenty-seven studies were included, most focusing on children with PFS and SE, whereas focal seizures were underreported, particularly extratemporal ones. Acute MRI findings (<1-week) most commonly showed hippocampal or limbic swelling with T2/FLAIR hyperintensity and diffusion restriction, consistent with edema, particularly following prolonged seizures. ASL reported cases of early focal hyperperfusion concordant with the seizure focus, often persisting longer than diffusion abnormalities. Subacute changes (days 4-10) included evolving edema and post-ictal hypoperfusion, while chronic findings included hippocampal atrophy, mesial temporal sclerosis (MTS), and persistent white matter abnormalities on DTI. Thalamic, basal ganglia, and subcortical white matter involvement have been hypothesized to reflect excitotoxic or inflammatory propagation beyond the seizure onset zone. Early DWI/ADC changes correlated with seizure duration, and when involving the hippocampus predicted MTS and long-term cognitive sequelae in some studies.

Conclusions: Peri-ictal MRI abnormalities in children are most frequently described in PFS and SE, where recurring patterns of acute edema and later structural changes have been reported. However, existing evidence is heterogeneous and weighted toward early-life and acute symptomatic populations, limiting generalizability. Associations between early imaging changes and longer-term outcomes remain inconsistent.

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Advanced imaging techniques, largely underreported, may provide additional insights into seizure-related network alterations, although their prognostic relevance remains uncertain. Overall, larger, seizure-type specific longitudinal studies are needed before definitive conclusions can be made.

Keywords: Epilepsy; seizures; children; acute; magnetic resonance imaging (MRI)

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Introduction

Background

Epilepsy is the fourth most common neurologic disorder across all ages (1), yet the role of seizures in brain changes remains controversial. Magnetic resonance imaging (MRI)

changes during seizures and status epilepticus (SE) show dynamic alterations across T2-weighted/fluid-attenuated inversion recovery (T2/FLAIR), diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC), and perfusion imaging. Understanding the timing and pathophysiology of these changes is crucial to distinguish seizures from other acute neurological conditions and potentially guide prognostication.

Highlight box

Key findings

- In children, the hippocampus emerges as the most frequently involved structure in prolonged seizures and status epilepticus (>15–30 minutes), with possible secondary involvement of thalamus, basal ganglia, white matter, and cortical networks. Magnetic resonance imaging (MRI) findings in focal seizures remain underreported and incompletely characterized.

What is known and what is new?

- Peri-ictal MRI changes in children are inadequately characterized, in part due to limited availability of longitudinal controlled studies. In addition, the pediatric brain shows age-specific vulnerability, limiting extrapolation from adult or animal data.
- Existing evidence, which is largely derived from heterogeneous studies involving limited seizure-population subgroups with prolonged seizures and status epilepticus, suggests an association with temporally evolving MRI abnormalities, from acute edema to later structural remodeling. The hippocampus is the most commonly reported site of involvement, although other regions may also be affected. It remains unknown whether short seizures cause any brain imaging changes in children with pharmacoresistant epilepsy.

What is the implication, and what should change now?

- Early multimodal imaging may potentially provide additional insights into seizure-related brain changes and prognostic associations, particularly in cases of prolonged seizures and status epilepticus. However, given the heterogeneity and potential confounding of the available evidence, the clinical applicability of these findings remains uncertain and requires further validation in well-designed studies.

Rationale and knowledge gap

Most of the current knowledge on the topic comes from animal models: focal seizures seem to induce an early ADC decrease that later normalizes and becomes chronically elevated at the seizure focus (2,3). The restricted diffusion observed after SE has been proposed to reflect a combination of cytotoxic edema, glutamate-mediated excitotoxicity, and increased membrane permeability (4,5). Increased neural excitability causes immediate increase in cerebral blood flow (CBF) that can be detected using arterial spin labeling (ASL) (6). Importantly, ASL-detected hyperperfusion in seizure-involved regions can persist longer than DWI-restricted diffusion, even beyond 24 hours from onset (7). However, preclinical studies on rodents indicate that peri-ictal deficits, which are transient neurological impairments occurring during or shortly after seizures (e.g., Todd's paresis) arise not only from seizure discharges alone but also from COX-2-dependent hypoxia in affected regions, as confirmed by pharmacologic inhibition of COX-2 or L-type calcium channels (8).

Although translating these mechanisms to children remains limited and indirect, human studies using ASL and computed tomography perfusion (CTP) support this stroke-like neurovascular response, detecting post-ictal hypoperfusion in the seizure onset zone (SOZ) associated with transient deficits, typically occurring 30–90 minutes after a seizure and correlating with seizure duration (9,10).

Apparent inconsistencies across studies likely arise from the highly dynamic behavior of peri-ictal perfusion changes, together with the lack of standardized imaging timepoints.

Microstructural changes may occur in the peri-ictal or post-ictal stages, involving the white matter (WM) and the blood-brain barrier (BBB). WM disruption, including demyelination, impaired axonal integrity, and maladaptive activity-dependent myelination, can alter network connectivity and synchrony, which can further reinforce epileptic network dysfunction (11). BBB damage is hypothesized to allow serum proteins and cytokines to enter, disrupt ionic homeostasis, and enhance neuroinflammation, lowering seizure thresholds (12–14). These can cause T1-weighted contrast enhancement following SE in rodents (15), or even mimic pseudoprogession in patients with brain tumors (16).

Objective

Since most of these insights come from animal models or studies involving mixed age groups, the unique pediatric brain changes remain largely unexplored. While prior works have already attempted to summarize acute seizure-related imaging changes in mixed-aged cohorts (17,18), an updated pediatric-focused overview is lacking. To address this gap, this scoping review attempts to map the temporal evolution of seizure-related neuroimaging findings on MRI in the pediatric population alone, focusing on changes detected on structural, diffusion, perfusion and advanced imaging techniques, such as diffusion tensor imaging (DTI) and functional MRI (fMRI), from the pre-ictal to chronic stages. We present this article in accordance with the PRISMA-ScR reporting checklist (available at <https://tp.amegroups.com/article/view/10.21037/tp-2026-0293/rc>) (19).

Methods

We systematically searched PubMed and MEDLINE from January 2000 to September 2025 to identify published clinical studies on imaging biomarkers in seizure-related abnormalities, with a focus on T2/FLAIR, DWI/ADC, and perfusion changes, and their temporal evolution across ictal and post-ictal phases. Keywords were searched in titles and abstracts and combined with Medical Subject Headings (MeSH) terms, where available. The search strategy included terms related to seizures and epilepsy (“Seizures“, “Epilepsy“, “status epilepticus“), neuroimaging modalities (“MRI“, “DWI“, “ADC“, “T2“, “FLAIR“,

“perfusion imaging“, “ASL“, “DSC“, “SPECT“), and temporal descriptors (“ictal“, “peri-ictal“, “postictal“). Boolean operators (“AND“, “OR“) were applied to ensure comprehensive coverage of the literature. The detailed search string is outlined as follows: (“Seizures”[MeSH] OR “Epilepsy”[MeSH] OR seizure*[tiab] OR epilep*[tiab] OR “status epilepticus”[tiab]) AND (“Magnetic Resonance Imaging”[MeSH] OR “Diffusion Magnetic Resonance Imaging”[MeSH] OR “Perfusion Imaging”[MeSH] OR “Tomography, Emission-Computed, Single-Photon”[MeSH] OR MRI[tiab] OR DWI[tiab] OR ADC[tiab] OR T2[tiab] OR FLAIR[tiab] OR perfus*[tiab] OR ASL[tiab] OR DSC[tiab] OR SPECT[tiab]) AND (“Time Factors”[MeSH] OR ictal[tiab] OR periictal[tiab] OR postictal[tiab]).

Databases were searched using filters to include only clinical or observational research publication types. Specifically, the search was limited to: adaptive clinical trials, case reports, clinical studies, clinical trials (including veterinary), clinical trial protocols, comparative studies, controlled clinical trials, equivalence trials, evaluation studies, meta-analyses, multicenter studies, observational studies, pragmatic clinical trials, randomized controlled trials, and validation studies. Non-clinical publications such as animal-only preclinical studies, *in vitro* research, reviews, systematic reviews, editorials, letters, comments, conference proceedings, guidelines, consensus statements, historical articles, and other non-original research materials were excluded from the analysis. We restricted the search to English-language studies involving human participants aged 0–18 years, as we aimed to specifically focus on the available evidence regarding seizure-related effects in the pediatric population. Duplicate records were removed, and the remaining articles were screened based on their titles and abstracts. All full texts were obtained for those meeting the initial screening criteria and were then evaluated individually for final inclusion in the summary. Given the limited number of large-cohort studies, we also incorporated pertinent case series (n≥3), and small-sample studies that involved imaging assessments. Single or two-case reports were excluded, as well as nuclear medicine studies. To identify additional relevant studies, a thorough snowball search of the references of all selected articles was performed. The entire process adhered to the most recent PRISMA-ScR guidelines. No automated tools were utilized for literature screening, selection, and analysis. The initial screening was conducted by the primary reviewer (L.F.) and confirmed by a senior reviewer (F.D.), who

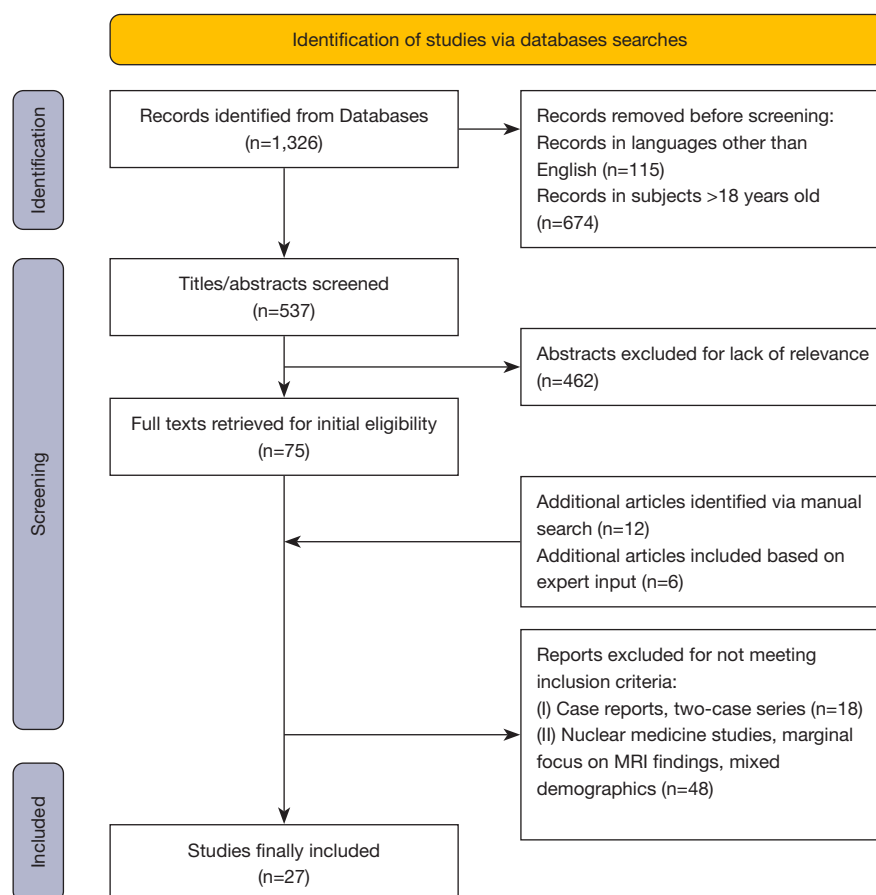


Figure 1 PRISMA flow diagram.

approved the final dataset and suggested additional relevant studies. Available imaging data were jointly assessed by two neuroradiologists (L.F., F.D.). Disagreements were resolved by consensus. Clinical-radiological associations were evaluated with primary emphasis on the timing of MRI acquisition relative to seizure onset, while also considering, to a lesser extent, seizure duration and burden, with the goal to delineate the dynamic trajectory of MRI changes rather than defining specific underlying etiologies. To support the inclusion of a broader range of literature, MRI techniques and heterogeneous cohorts, no formal quality assessment of the included studies was conducted, in line with scoping review methodology.

Results

Literature screening

The complete literature selection framework is summarized

in *Figure 1*.

Summary of core findings

Twenty-seven studies met the inclusion criteria, primarily involving infants and children with prolonged febrile seizures (PFS) and febrile status epilepticus (FSE). Cases of focal seizures in the absence of concurrent genetic or metabolic disorders were underrepresented. Acute MRI findings (<1-week post-onset) included hippocampal or limbic swelling, T2/FLAIR hyperintensity, and variable restricted diffusion. ASL studies reported perfusion abnormalities, from acute-phase increased CBF to later hypoperfusion in affected regions. Subacute to chronic follow-ups showed hippocampal atrophy, mesial temporal sclerosis (MTS), and persistent WM disruption and reorganization, with some studies reporting correlations between early imaging changes and seizure burden, duration, and risk of long-term sequelae. Studies

including advanced imaging techniques described pre-ictal functional activation in BOLD-fMRI, as well as post-ictal microstructural WM abnormalities on DTI [$\downarrow$ fractional anisotropy (FA), $\uparrow$ mean diffusivity (MD)] in bilateral tracts, comprising the hippocampus, thalamus, and corpus callosum.

Results of individual studies

A complete overview of all included studies and retrieved data is displayed in *Table 1* (in alphabetical order by first author). The full detailed summary of individual studies can be found in the [Appendix 1](#).

Discussion

Key findings

To our knowledge, this is the first review study to ever include two decades of MRI-based evidence examining the structural and microstructural alterations of prolonged seizures in the pediatric population alone. Across 27 widely heterogeneous studies, covering modalities from conventional MRI to ASL, DTI, and fMRI, available data outline possible associations between seizures and evolving patterns of brain injury, mainly affecting the hippocampus, thalamus, basal ganglia, cortex and WM tracts, especially in children with PFS and SE. Acute phases are commonly described as showing both cytotoxic/excitotoxic edema with diffusion restriction and vasogenic edema, as well as increased blood flow on ASL (*Figure 2*) followed by subacute normalization or pseudonormalization. Nonetheless, diffusion and perfusion abnormalities often extend beyond the primary seizure focus and can persist for several days post-ictally (*Figures 3,4*), suggesting possible widespread network involvement. In this phase, SE-related DWI/ADC peri-ictal abnormalities do not usually follow a vascular territory and are less marked than in stroke. At later stages, chronic atrophy consistent with gliosis and sclerosis of the mesial temporal lobe has been reported in some cohorts (*Figure 5*). Across studies, imaging severity was hypothesized to correlate with clinical seizure burden, with early abnormalities possibly predicting progression to chronic structural changes. An at-a-glance graphical summary of the retrieved MRI data and the proposed pathophysiological patterns is presented in *Figure 6* and *Table 2*.

Hippocampal involvement

The hippocampus is the most frequently reported site of involvement during and after PFS and FSE, likely for its proposed central role in the generation and propagation of seizures. In animal models, neonatal and early-life seizures trigger structural and functional remodeling of hippocampal circuits, including impaired neurogenesis in the dentate gyrus, disrupted synaptic plasticity, and long-term cognitive deficits, with effects dependent on developmental stage (46,47). In immature rats, neuronal loss after kainate-induced seizures evolves slowly and selectively in hippocampal CA1–CA3 regions, unlike the rapid necrosis observed in adults (48). Overall, in preclinical studies, the immature hippocampus displays unique vulnerability to acute excitotoxic and inflammatory injury, with relative resistance to immediate massive cell death, likely leading to impaired neurogenesis and cognitive comorbidities at later stages. In children, it is also hypothesized that individuals who develop PFS may have an underlying hippocampal vulnerability, predisposing them to seizure initiation and subsequent structural injury when exposed to febrile stress (39,42,45).

Acute hippocampal edema, characterized by decreased ADC and increased T2/FLAIR signal within 48–72 hours from seizure onset, has been repeatedly observed among children with PFS and SE (14,22,24,35,40,42,45), possibly reflecting excitotoxic swelling and febrile metabolic stress, particularly in the context of prolonged seizure burden (>15–30 minutes). In these cases, increasing seizure duration was associated with a greater incidence and severity of acute hippocampal changes overall. One study (39) found elevated instead of decreased ADC values within the first 48 hours, suggesting the development of transient vasogenic edema due to BBB disruption. Additional recurrent findings described in the acute stage are hippocampal volumetric increase and prolongation of T2 relaxation time between 2 and 5 days after the onset of seizures consistent with acute edema (24,37), which was also confirmed in one study where refractory FSE (≥ 60 minutes) was associated with significantly increased hippocampal volumes compared to controls (33).

In the subacute stage, typically 1–3 weeks post-ictal, normalization of ADC values has been described (24), while T2/FLAIR hyperintensity persisted and early atrophy started to show, potentially predicting MTS and poor neurological outcomes in some cases (24,42,45). In fact,

Table 1 Overview of all included studies and retrieved data (in alphabetical order by first author)

Author, year	PMID	Country	Study type	Sample size (seizure duration cut-off)	Cases age	Magnet strength	Technique: sequences (software used)	MRI timing	Relevant MRI findings	Clinical implication
Amarreh (20), 2013	24148888	USA	Cross-sectional case-control	n=9 active idiopathic epilepsy with normal structural MRI + n=11 remissions + n=29 normal controls	Mean 18.61±3.57 y (range ≈11–26 y)	1.5-T	MRI-DTI (FMRIB TBBS)	5 y form diagnosis	Chronic (>5 y): ↓FA (FOF, R cingulum/fornix), ↑MD, ↑AD (FOF, L CR), ↑RD (L CR) in active vs. controls (P<0.05); ↓FA (IC, R cingulum, R CR, splenium) ↑MD (CR, L FOF), ↑AD (L IC), ↑RD (R IC CR, splenium) in active vs. remitted (P<0.05)	Seizure-activity can determine long-term (>5 y), partially reversible WM microstructural abnormalities detectable via DTI
Basti (21), 2020	33160202	Italy	Prospective observational cohort	n=24 HIE with electrographic seizures (>15 min/h or ≥30 min continuously)	5 m–30 m	3-T	Structural MRI: DWI/ADC, T1, T2, FLAIR	N/D	Acute/subacute: ↑seizure burden in 26% (n=6 frequent seizures + n=2 SE); moderate/severe T1-T2 injury in 26% (n=8), including BG/thalamus (n=4), WM (n=4), PLIC (n=2), cortex (n=1); more MRI abnormalities in ↑seizure burden group vs. sporadic seizure (P=0.0009)	High seizure burden (>15 min) can exacerbate secondary HIE injury on MRI despite hypothermia and predict poor outcome
Chevret (22), 2008	18204804	France	Prospective case series	n=4 RFSE with unknown etiology (≥20 d)	5 y–10 y	1.5-T	Structural MRI: DWI/ADC, T2, FLAIR	T ₁ (all): <5 d T ₂ (n=3): 10 d T ₃ (all): 7 m from onset	Acute/subacute (<5 d): ↓ADC (n=2), ↑T2/FLAIR (n=4) hippocampus Subacute (10 d): persistent ↑T2/FLAIR hippocampus (n=3) Chronic (7 m): atrophic hippocampus (n=4)	Early (<5 d) hippocampal injury on T2 may predict chronic atrophy and poor neurological outcome in RFSE
Federico (23), 2005	15975948	Italy	Prospective case series	n=3 partial seizures	11, 15, 22 y	3-T	Continuous BOLD-fMRI: GRE-EPI	Starting 30 min before onset	Pre-ictal (20-11 min): BOLD changes (>FL), ipsilateral (pt 1) or contralateral (pt 2, 3) to seizure focus	Seizure generation involves prolonged pre-ictal network imbalance (excitability + inhibition) detectable via BOLD-fMRI up to 20 min before seizure onset
Farina (24), 2004	14991256	Italy	Retrospective case series	n=5 complex focal seizures with viral prodrome, including n=2 FSE (≥15 min)	17 m–7 y	1.5-T	Structural MRI: serial DWI/ADC, T1, T2, FLAIR	T ₁ (n=4): <72 h T ₂ (n=1): 15 d T ₃ (n=4): 2-18 m from onset	Acute/subacute (<72 h): enlarged hippocampus, ↑T2/FLAIR, ↓ADC (mean 14% head, P=0.04, 15% body, P=0.006) (n=4), Subacute (~15 days): ↑T2/FLAIR, normal ADC hippocampus (n=1) Chronic (2-18 m): atrophic hippocampus, ↑ADC (mean 19% head, 17% body) (n=5)	Early (<72 h) ↓ADC reflects trainset cytotoxic edema and may represent a sensitive predictive marker for MTS
Gonçalves (25), 2020	32381541	USA	Retrospective case series	n=13 POLG+ with seizures	7 m–16 y	N/D	Standard MRI: DWI/ADC, T1, T2, T2*/SWI + MRS + MRI-ASL (only at T ₂)	T ₁ : -1 to 22 d from EEG diagnosis T ₂ : w to m after onset	Acute/subacute: abnormal signal changes (n=12), including 54% unilateral/ bilateral perirolandic (n=5/n=2), 77% thalamic (n=5/n=5), perirolandic + thalamic (n=6), 42% other regions (n=5); ↑lactate on MRS (n=3); DWI most sensitive (n=10) vs. T2 (n=9) or FLAIR (n=7) Subacute/chronic: volume loss in 77% (n=10), occipital lobe/thalamus most affected (61%); late onset perirolandic sign (n=3); ↑ASL perirolandic, thalamus, occipital/parietal lobes (n=N/D)	Acute perirolandic ± thalamic involvement on DWI may be a POLG biomarker in children with seizures
Govindan (26), 2008	18436432	USA	Cross-sectional case-control	n=13 LTLE + n=12 normal controls	11 m–19 y	3-T	MRI-DTI (DTI-Studio v2.40)	N/D	Chronic: ↓FA bilateral WM (UF, ARF, ILF, CST), altered tensor shape metrics with ↑Cs, ↓CI/Cp TL and CST (L > R, ⊥ to axons); reversal ARF asymmetry (R > L); group differences (P<0.05)	LTLE determines long-term bilateral WM tract reorganization secondary to axonal swelling/ degeneration detectable via DTI
Kimiwada (27), 2006	16417545	USA	Cross-sectional case-control	n=14 TLE, including n=7 with secondarily generalization + n=7 partial seizures + n=14 normal controls (≥1 weekly attack, ≥1 weekly to multiple partial seizures/d)	1.6 y–16 y	1.5-T	MRI-DTI (DTI-Studio)	N/D	Chronic: ↓FA bilateral hippocampus (P<0.001 ipsilateral, P=0.002 contralateral); ↑ADC ipsilateral hippocampus (P=0.003); ↑ADC ipsilateral thalamus in secondarily generalized seizures (n=7, P=0.09); no group differences in the lentiform nuclei	TLE produces long-term bilateral hippocampal/ thalamic microstructural injury detectable via DTI
Lee (28), 2019	30718217	South Korea	Retrospective observational cohort	n=43 new seizure cases with normal structural MRI, including n=36 focal + n=7 generalized	Mean 6.3± 3.3 y (range 0.9–16.2 y)	3-T	Structural MRI + MRI-ASL	2 h–90 d	Abnormal ASL in 58.1% (n=25); among focal seizures cases, ASL abnormal in n=24, concordant with clinical focus in 52.8% (n=19) Overall moderate ASL-clinical concordance (κ =.542) Acute (≤1 d): ↑CBF (n=3), significantly associated to concordance (P=0.014) Subacute/chronic (2 d–90 d): ↓CBF (n=22)	Early post-ictal ASL improves seizure focus localization in MRI-negative epilepsy, with diagnostic concordance primarily dependent on early post-seizure acquisition (≤1 d)
Lewis (29), 2014	24318290	USA	Prospective longitudinal case-control (FEBSTAT cohort)	n=226 FSE + n=38 SFS controls (≥30 min)	1 m–6 y	1.5-T	Structural DWI/ADC, T1 (volumetry), T2 (SnAP:IRIS15)	T ₁ : <7 d T ₂ (n=130): 1 m–2.5 y (median 1.07 y) from onset	Acute/subacute (<7 d): ↓ADC/↑T2 hippocampus (CA1) in 9.7% (n=22) Chronic (≈ 1 y): MTS in 71% with initial ↑T2 (n=10 out of 14 with available FUP); ↑ADC vs. SFS (P=0.02)	Acute (<7 d) hippocampal cytotoxic injury after FSE drives later sclerosis in FSE vs. SFS, especially in predisposed children

Table 1 (continued)

Table 1 (continued)

Author, year	PMID	Country	Study type	Sample size (seizure duration cut-off)	Cases age	Magnet strength	Technique: sequences (software used)	MRI timing	Relevant MRI findings	Clinical implication
Lewis (30), 2024	38606600	USA	Prospective longitudinal case-control (FEBSTAT cohort)	n=222 FSE + n=416 controls, including n=109 SFS + n=344 normal subjects (≥30 min)	1 m–5 y	1.5-T	Structural MRI: high resolution T1 (volumetry), T2 (SnAP:IRIS15)	T ₁ : 1 y T ₂ : 5 y T ₃ : 10 y Note: full 10-y imaging FUP in 14/22 ↑T2 subjects + 37/159 normal T2 subjects	Acute/subacute (mean 4.4 d): ↑T2 hippocampus (n=22) Chronic (1-10 y): atrophic/sclerotic hippocampus in acute ↑T2 group (P<0.0001), with definite MTS in 70% (n=10 vs. n=1 controls); atrophy R > L (P<0.0001), with persistent ↑T2 R > L (P=0.0003); ↑asymmetry (P<0.0001) and ↑epilepsy (n=7; MTLE n=2, both with definite MTS) only in ↑T2 group	Hippocampal T2 hyperintensity in the post-FSE acute phase can predict long-term risk for epilepsy and MTS
Mabray (31), 2018	29600511	USA	Retrospective case series	n=3 subjects with underlying conditions, including n=2 SE	0–7 d	3-T	MRI-ASL	2–3 h	Acute (<3 h): focal ↑CBF corresponding to seizure focus (n=3) Subacute (<6 d): persistent ↑CBF (n=1)	ASL-MRI can show focal hyperperfusion within 2-3 h from seizure activity in neonates, persisting up to several days post-ictal
Meng (32), 2010	20656430	USA	Retrospective case-control	n=8 TLE with focal seizures/aura + n=8 normal controls	5.5–21 y	3-T	MRI-DTI (DTIStudio v2.4)	1–12 y from diagnosis	Chronic (>1 y): bilateral ↓FA SCC + capsular (P<0.05), ↑MD capsular (P<0.05), ↑λ⊥ capsular (P<0.001), ↑λ SCC (P<0.05), correlated with age and duration	TLE can be associated with long-term WM disruption secondary to impaired myelination/axonal loss detectable via DTI
Natsume (33), 2007	17784534	Japan	Prospective case-control	n=12 PFS + n=13 normal controls (≥30 min)	10 m–5 y	1.5-T	Structural MRI: DWI/ADC, T1 (volumetry), T2, FLAIR (DISPLAY)	<5 d from onset	Acute/subacute (<5 d): bilateral enlarged hippocampus (P=0.02 R, P=0.04 L) with positive correlation to PFS duration (P=0.03 R, P=0.001 L); in PFS ≥60 min group (n=7), ↑DWI unilateral hippocampus (n=3), ipsilateral thalamus (n=2) and anterior cingulate (n=1)	PFS ≥60 min induce early (<5 d) hippocampal/limbic edema visible on DWI
Nguyen (34), 2022	36471320	USA	Retrospective observational cohort	n=123 HIE, including n=39 with seizures (various types)	4–7 d	N/D	N/D	T ₁ : <24 h after rewarming, within 10 d of life T ₂ (n=36) 12–18 m T ₃ (n=24): 18 m	Acute/subacute: ↓ADC splenium in 21.1% (n=26, of which 69.2% with changes in other ≥2 regions); ↓ADC splenium SEN=54% SP =94%, PPV =81%, higher compared to other brain regions Chronic (12-18 m): developmental delay significantly associated with seizure burden (P<0.05) and ↓ADC splenium (P<0.01)	Splenial restricted diffusion has high PPV for recent seizures in neonates with HIE undergoing therapeutic cooling and can predict poor outcome
Okamoto (14), 2006	16541366	Japan	Retrospective case series	n=4/42 FSE with neurological sequelae (≥30 min)	12–19 m	1.5-T/3-T	Structural MRI: DWI/ADC, T2, FLAIR	T ₁ : 1–3 d T ₂ : 4–10 d T ₃ : 11–30 d T ₄ : >30 d from onset	Hyperacute/acute (1–3 d): normal acute/subacute (4–10 d): ↓ADC diffuse/subcortical WM (n=4); ↑CK TNF-α, IL-6 (n=3) subacute/chronic (>11 d): persistent ↑T2/FLAIR (n=3), atrophy (n=4) Longer seizure duration positively associated with neurological sequelae (P=0.002)	FSE may cause delayed inflammatory-mediated WM cytotoxic injury in some patients
Provenzale (35), 2008	18356445	USA	Prospective case-control	n=11 FSE, including TLE and complex partial seizures cases + n=30 normal controls (≥30 min)	11–45 m	1.5-T	Structural MRI: axial T1 (volumetry), axial/coronal T2 (Analyze, Biomedical Imaging Resource, Mayo Clinic)	T ₁ : <72 h T ₂ : 3–23 m from onset	Acute (<72 h): ↑T2 hippocampus (n=7, moderate/marked in n=4) chronic (3–23 m): atrophic hippocampus/MTS in patients with acute ↑T2 (n=5) statistically significant correlation between ↑T2 and V change (P <0.01)	Conventional T2 within 72 h can provide early prognostic information on hippocampal injury and seizure risk
Shah (36), 2014	24443407	UK	Prospective observational cohort	n=85 HIE, n=44 with seizures (>15 min/h or >30 min continuously)	4–35 d	1.5-T	Structural MRI: T1, T2	N/D, likely <1 w from onset in most cases	Acute/subacute: severe MRI injury in 36% (n=31); diffuse ↑T2 WM (n=13), BG/thalami (n=26), PLIC (n=14), cortex (n=38); minor SAH (n=21); ↑MRI changes in 2/3 of children with high seizure burden (P=0.01)	EEG seizure burden can predict MRI severity of hypoxic-ischemic damage in newborns with HIE undergoing hypothermia
Scott (37), 2002	12183341	USA	Cross-sectional case-control	n=21 FSE + n=14 afebrile SE + n=not given controls (≥30 min)	12–23 m (febrile) 17–60 m (afebrile)	1.5-T	Structural MRI: 3D T1 MPRAGE/FLASH (volumetry), T2 (relaxometry) (MEDx v3.3)	<5 d	Acute: early (≤48 h) transient ↑T2 relaxation time (P=0.048) + enlarged hippocampus (P=0.004) only in FSE group; MRI abnormalities in 5/21 FSE (asymmetry n=3, TL abnormality n=1, incidental finding n=1); in afebrile SE no enlargement hippocampus (P =1.0) but ↑T2 relaxation time (P=0.02) Subacute (3–5 d): no difference in hippocampal T2 relaxation time vs. controls (P =0.90)	Febrile-driven inflammation/dysmetabolism can trigger early (≤48 h) transient edema manifested as increased T2 relaxation time and hippocampal volume in FSE

Table 1 (continued)

Table 1 (continued)										
Author, year	PMID	Country	Study type	Sample size (seizure duration cut-off)	Cases age	Magnet strength	Technique: sequences (software used)	MRI timing	Relevant MRI findings	Clinical implication
Scott (38), 2003	12937081	USA	Prospective longitudinal case-control	n=14 FUP cases from previous cohort + n=29 normal controls (≥30 min)	14–31 m	1.5-T	Structural MRI: 3D T1 FLASH (volumetry), axial/coronal T2 (relaxometry) (MEDx v3.3)	4–8 m	Chronic 4–8 m: ↓V/T ₂ relaxation time (P=0.002/P=0.018) and ↑asymmetry hippocampus (P<0.05); no persistent T2 abnormality	FSE induces acute (≤48 h) transient hippocampal edema that can precede MTS and may reflect: 1. seizure-related neural loss (++), or 2. pre-existing vulnerability (+)
Scott (39), 2006	16981865	USA	Prospective longitudinal case-control	n=14 PFS (of 23 scanned) + n=24 normal controls (≥30 min)	14–31 m	1.5-T	Serial MRI DWI/ADC	T ₁ : <5 d T ₂ : 4–8 m from onset	Acute (≤48 h): ↑ADC hippocampus with reduction over time (P=0.048) Subacute (3–5 d): no significant change in ADC vs. controls (P =0.90) Chronic (4–8 m): absent age-related hippocampal ADC decline (P=0.029)	PFS are associated with acute (≤48 h) transient hippocampal vasogenic edema and may unmask underlying hippocampal vulnerability in the long-term
Shinnar (40), 2012	22843278	USA	Prospective longitudinal case-control (FEBSTAT cohort)	n=191 FSE + n=96 SFS controls (≥30 min)	1 m–5 y	1.5-T	Structural MRI: DWI/ADC, 3D T1, T2,	<72 h (67.5%) or 72 h–1 w (18.9%)	Acute/subacute (<1 w): ↑abnormalities hippocampus (P=0.001) and adjacent TL, amygdala, insula (P=0.015) in FSE vs. SFS: ↑T2 hippocampus only in FSE cases (n=22, definite in n=17, of which n=2 with MTS), R > L (n=13), extended to TL (n=6); developmental abnormalities (n=20, vs. n=2 controls, P=0.0097)	FSE can produce MRI evidence of acute hippocampal injury in approximately 11% of cases
Suzuki (41), 2020	32473849	Japan	Retrospective observational cohort	n=51 PFS (≥15 min)	8–70 m	1.5-T	Structural MRI: Standard DWI/ADC, T1, T2, FLAIR	T ₁ : ≤6 h from PFS cessation (82% <2 h) T ₂ : 13–72 h T ₃ : 4–71 m (median 36 m)	Hyperacute: ↓ADC cortex within 100 min in 18% (n=9, all <3 y, TL/FL more affected), associated with male sex (P=0.034), asymmetrical seizures (P=0.001), shorter seizure-to-MRI interval (P=0.02), but not seizure duration (P=0.601); cortex + thalamus in n=6; delayed ↓ADC hippocampus in n=1; Acute (6–72 h): ↓ADC cortex completely resolved Note: median (range) PFS duration 45 min (15–180 min)	PFS can induce hyperacute, transient cortical cytotoxic edema in some children, sometimes preceding hippocampal injury, with no evidence of unfavorable neuroimaging/epileptic sequelae in the short-term
Tanabe (42), 2011	21463269	Japan	Prospective longitudinal cohort	n=59 PFS (≥15 min)	8 m–5 y	1.5-T	Structural MRI: T1, T2, FLAIR with coronal sections ⊥ hippocampal long axis	T ₁ : ≈ 72 h from cessation T ₂ : 46 d T ₃ : 1 y	Acute: ↑T2/FLAIR hippocampus (n=1, 35 min seizure duration); no similar changes in other PFS cases Chronic (46 d–1 y): progressive MTS in subject with acute ↑T2/FLAIR	FSE determines T2/FLAIR changes in the hippocampus around 72 h from seizure cessation, reflecting possible genetic vulnerability that may predict MTS
Takanashi (43), 2006	16682659	Japan	Retrospective case-control	n=17 PFS with encephalopathy + n=3 PFS controls (>30 min)	10 m–4 y	1.5-T	Structural MRI: DWI/ADC, T2, FLAIR + MRS	T ₁ : <2 d, T ₂ : 3–9 d T ₃ : 10–25 d from onset	Acute (<2 d): no abnormality Acute/subacute (3–9 d): ↓ADC subcortical WM (n=17) + linear ↑T2/FLAIR along subcortical U-fibers (n=13); frontal/frontoparietal WM most affected, with perirolandic sparing Chronic (9–25 d): DWI normalization; atrophy after 2 w (n=16); transient ↑Glu/Gln, ↓NAA on MRS (n=1) Note: seizure mostly >1 hour (n=12); infective etiology common (n=10)	PFS can induce transient glutamate-mediated excitotoxic injury in the subcortical WM, which can be detected on early MRS around day 3
Yoong (44), 2013	24304434	UK	Prospective longitudinal case-control	n=33 PFS + n=47 non-PFS + n=31 normal controls (>30 minutes)	0.18–15.5 y	1.5-T	Structural MRI: 3D-FLASH T1 (volumetry) (MRlcroN, BET-FSL)	T ₁ : 1 m T ₂ : 6 m T ₃ : 12 m from diagnosis	Chronic (m to y): ↓V hippocampus in 25% of those with ≥2 MRI scans (n=15/60, including n=5/26 PFS and 10/34 non-PFS, unilateral >bilateral); older children more likely to show volume loss (P=0.003); significant negative association between ↑PFS-hippocampal growth (P<0.001)	SE-induced acute hippocampal injury can lead to progressive hippocampal volume loss, regardless of whether the SE is febrile or not
Yokoi (45), 2019	31166617	Japan	Prospective longitudinal case-control	n=22 FSE + n=13 normal controls (≥30 min)	6 m–5 y	1.5-T	Structural MRI: DWI/ADC, T1, T2	T ₁ : <5 d T ₂ : 9–13 y from onset	Acute (<5 d): ↑DWI hippocampus ± thalamus in 27% (n=6, seizure duration ≥45 min), usually ipsilateral (L > R) Chronic (9–13 y): atrophic hippocampus/MTS in all children with acute ↑DWI, and higher MTLE incidence (n=5/5 vs. n=1/10 without, P=0.002)	FSE induces early (<5 d) cytotoxic edema detected by DWI/ADC that may predict MTLE evolution in some cases

AD, axial diffusivity; ADC, apparent diffusion coefficient; ARF, arcuate fasciculus; ASL, arterial spin labelling; BG, basal ganglia; BOLD, blood oxygen level dependent; CBF, cerebral blood flow; CK, cytokines; CR, corona radiata; CST, corticospinal tract; DTI, diffusion tensor imaging; DWI, diffusion weighted imaging; EEG, electroencephalogram; FA, fractional anisotropy; FL, frontal lobe; FOF, fronto-occipital fasciculus; FSE, febrile status epilepticus; FUP, follow-up; h, hour(s); HIE, hypoxic ischemic encephalopathy; IC, internal capsule; IL-6, interleukin-6; ILF, inferior longitudinal fasciculus; L, left; LTLE, left temporal lobe epilepsy; M, months; MD, mean diffusivity; min, minutes; MR, magnetic resonance; MRI, magnetic resonance imaging; MRS, magnetic resonance spectroscopy; MTS, mesial temporal sclerosis; MTLE, mesial temporal lobe epilepsy; PFS, prolonged febrile seizures; pt, patient; PLIC, posterior limb internal capsule; POLG, DNA polymerase gamma; PPV, positive predictive value; R, right; RD, radial diffusivity; RFSE, refractory febrile status epilepticus; SCC, splenium corpus callosum; SE, status epilepticus; SEN, sensitivity; SFS, simple febrile seizures; SP, specificity; T, timepoint; TBBS, tract-based spatial statistics; TL, temporal lobe; TLE, temporal lobe epilepsy; TNF- α , tumor necrosis factor- α ; UF, uncinate fasciculus; V, volume; WM, white matter; y, years.

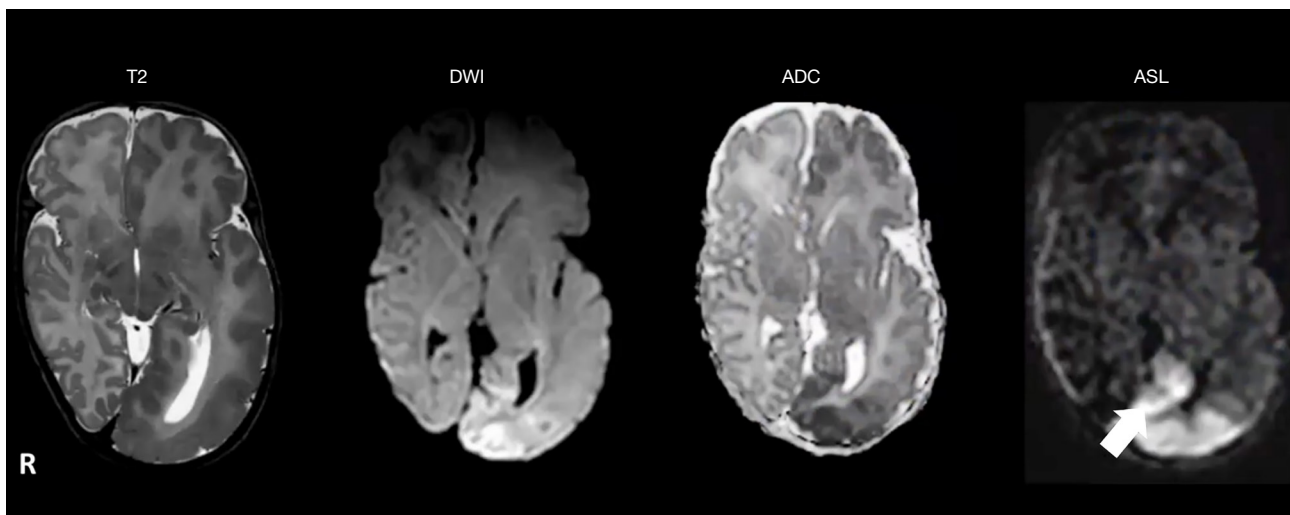


Figure 2 Acute MRI changes involving the left occipital lobe of a 1-month-old female with left-sided hemimegalencephaly. Note is made of increased perfusion on ASL (white arrow). ADC, apparent diffusion coefficient; ASL, arterial spin labeling; DWI, diffusion weighted imaging; MRI, magnetic resonance imaging; R, right.

longitudinal follow-ups months to years after seizure onset often confirmed progressive hippocampal volume loss and asymmetry (29,30,38,42,44). Hippocampal disrupted growth patterns have been described even when acute MRI appears normal (29), which may suggest an underlying structural vulnerability. Interestingly, Yoong *et al.* (44) reported progressive hippocampal volume loss in 20–30% of children after both febrile and non-febrile SE, implying that hippocampal susceptibility may not be limited to febrile cases alone.

Taken together, these studies propose a temporal and pathophysiologic trajectory: acute hippocampal excitotoxic/cytotoxic/vasogenic edema detectable on DWI/ADC maps within 72 hours from seizure onset, its resolution or progression to volume loss, and an associated increased risk of subsequent MTS and mesial temporal lobe epilepsy (MTLE) that can depend on superimposed vulnerability. Specifically, according to the latest FABSTAT longitudinal outcomes (30), acute hippocampal T2 hyperintensity after FSE increases 10-year MTLE risk to around 22–39% and the risk of developing definite MTS to 70%. Early DWI and T2 changes after PFS and FSE have therefore been theorized to serve as potential non-invasive prognostic markers for MTS. Nonetheless, interpretation of the existing literature is limited by the general lack of direct evidence for pre-existing hippocampal abnormalities, as children typically do not undergo MRI before their first seizure episode, and by substantial interstudy variability in

the definition of PFS. In addition, the FEBSTAT cohort included a relatively high proportion of children with pre-existing neurological and MRI abnormalities, raising the possibility that some hippocampal abnormalities may have preceded FSE. By contrast, the reported risk appears lower in UK population-based studies, which excluded children with prior neurological abnormalities from PFS cohorts.

Thalamic and basal ganglia abnormalities

In our dataset, thalamic and, more variably, basal ganglia involvement appeared in a subset of patients with severe or prolonged seizures, particularly in neonates and very young children. Some studies documented diffusion restriction and T2/FLAIR hyperintensity in the thalamus or basal ganglia within 5 days from seizure onset, often ipsilateral to co-occurring hippocampal lesions (33,45). In neonates with hypoxic ischemic injury (HIE), high seizure burden can exacerbate deep gray matter injury and T2/FLAIR changes despite therapeutic hypothermia (21,36), providing only limited evidence of a seizure-dependent effect, as HIE- and seizure-related changes are often difficult to distinguish. From a mechanistic perspective, thalamic vulnerability may reflect both direct excitotoxic propagation via cortico-thalamic circuit recruitment and secondary metabolic distress. In line with this, DTI studies in patients with TLE also showed altered anisotropy in thalamic and subcortical pathways (27), consistent with the possibility

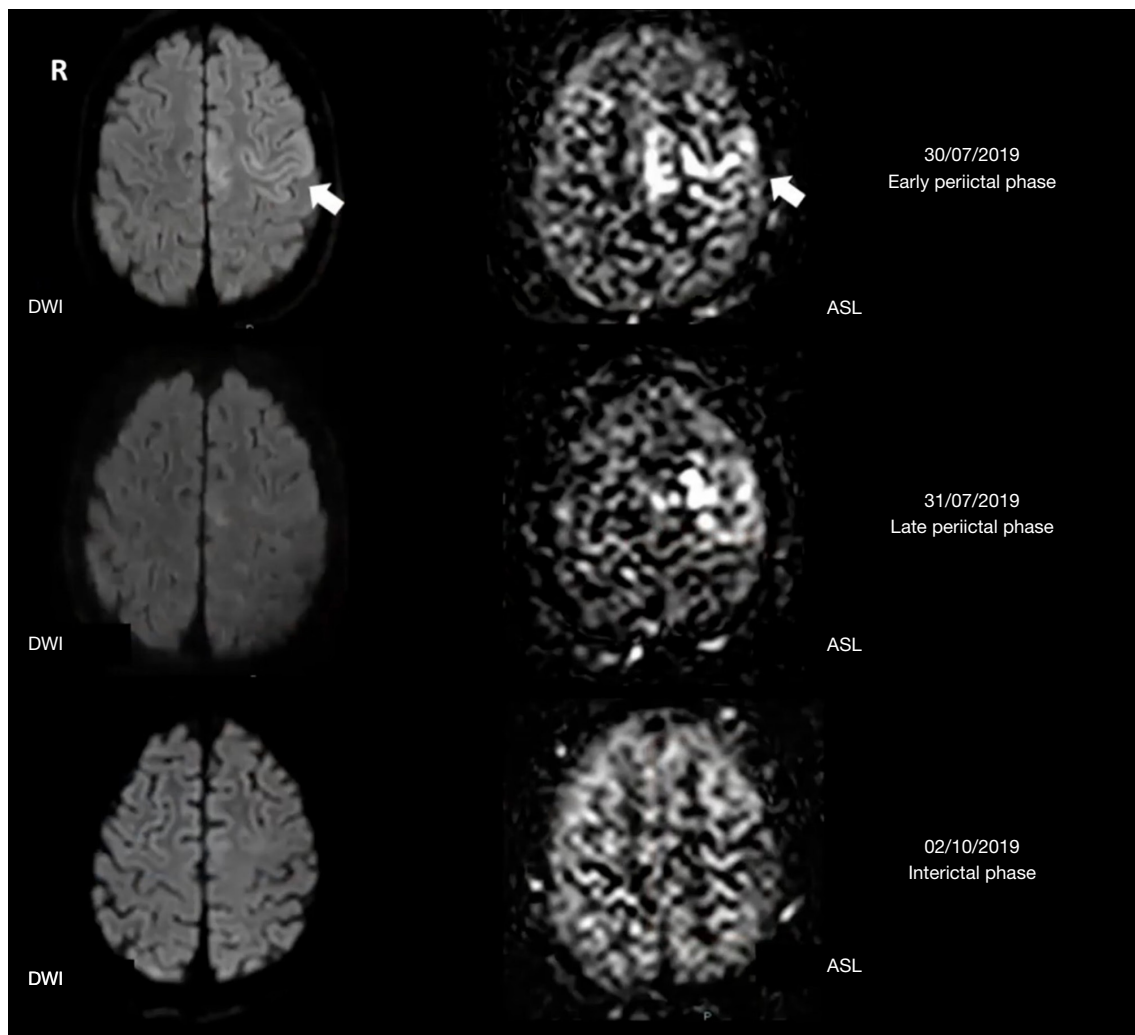


Figure 3 Acute (white arrows) to subacute diffusion and perfusion MRI changes in a 14-year-old female with left focal epilepsy triggered by tick-borne encephalitis. Diffusion abnormalities fade after the early peri-ictal phase, while increased perfusion persists in the late peri-ictal phase (second row). After a 2-month interval period (third row), no residual signal changes are detected. ASL, arterial spin labeling; DWI, diffusion weighted imaging; MRI, magnetic resonance imaging; R, right.

that the thalamus participates in seizure generalization while undergoing permanent changes through network remodeling. In fact, autopsy studies of patients presented with SE who died have demonstrated neuronal loss in the thalamus (in addition to other brain regions), ipsilateral to the seizure focus (49). Acute thalamic signal abnormalities have similarly been described in certain neurogenetic and rare pediatric epileptic syndromes, such as polymerase gamma (POLG)-related epileptic encephalopathy and febrile infection-related epilepsy syndrome (FIRES) (25,50), where disease-specific susceptibilities likely interact with intense and prolonged seizures to produce deep gray lesions.

In FIRES and New-Onset Refractory Status Epilepticus (NORSE), MRI can also detect bilateral basal ganglia signal changes, including the claustrum sign, which are associated with presumed cytokine-mediated injury (51,52). Although in children these findings can mimic mitochondrial or metabolic disorders, they are mainly dynamic in nature and related to prolonged refractory seizure activity, emphasizing the need for clinical correlation and serial imaging for correct interpretation. Moreover, thalamic (particularly pulvinar) and basal ganglia signal abnormalities, often bilateral, are not uncommon in older patients as well, reflecting that deep gray matter involvement can occur

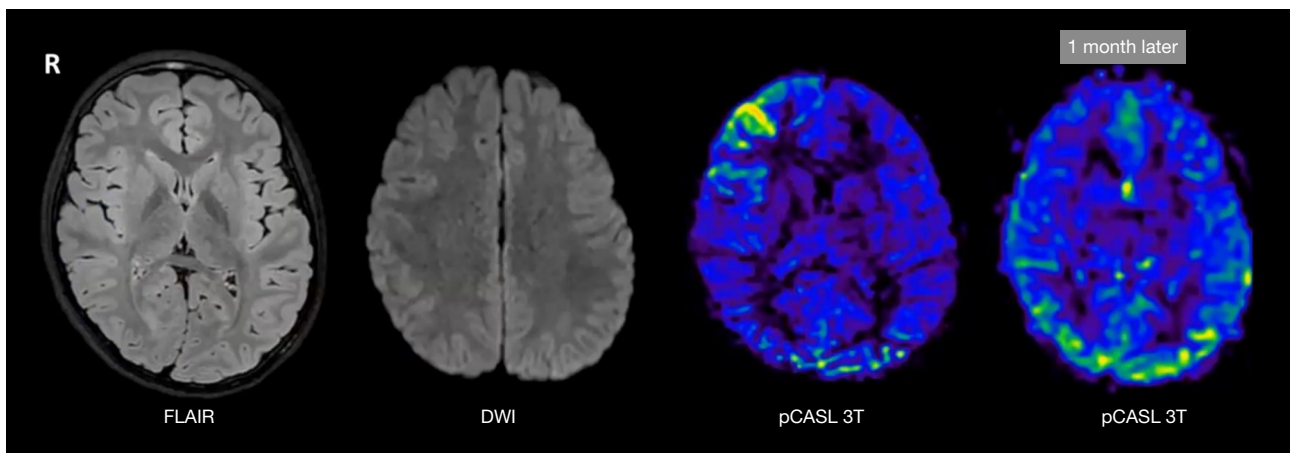


Figure 4 Acute to chronic MRI changes after first seizure presentation in a 11-year-old female with positive right frontal EEG abnormalities. In the acute phase, conventional MRI sequences (DWI, FLAIR) show no visible signal changes, whereas ASL reveals increased perfusion in the seizure focus, concordant with EEG findings. After 1 month from seizure onset, ASL-hyperperfusion is no longer detectable. 3T, 3-tesla; ASL, arterial spin labelling; DWI, diffusion weighted imaging; EEG, electroencephalogram; FLAIR, fluid attenuated inversion recovery; MRI, magnetic resonance imaging; pCASL, pseudo-continuous arterial spin labelling; R, right.

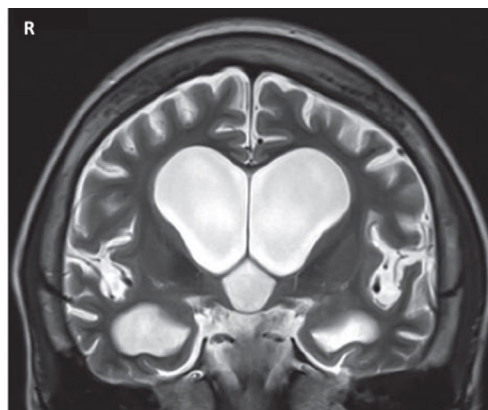


Figure 5 Widespread cerebral atrophy on T2-weighted imaging in a 10-year-old patient with FIRES, reflecting neuronal loss and chronic structural changes. FIRES, febrile infection-related epilepsy syndrome.

across the entire age spectrum in refractory SE (50,53).

Overall, available data support the view that the thalamus is a key network node that is both actively recruited during high seizure activity and vulnerable to seizure-associated injury, with the development of early imaging changes that may evolve into persistent structural and connectivity alterations in some patients. Nonetheless, distinguishing seizure-related injury from an underlying hypoxic-ischemic or systemic pathology in these populations remains particularly challenging, and causal attribution is often not possible.

Subcortical white matter lesions and network disruption

Subcortical white matter abnormalities are observed across pediatric SE and TLE, particularly in severe or refractory cases, although evidence comes largely from small cohorts. Diffusion and T2/FLAIR abnormalities, appearing between 3 and 10 days after seizures, have been suggested to relate to delayed cytotoxic edema and inflammatory demyelination. One study (14) reported delayed WM ADC reductions and increased cytokines following SE in a small subset of patients with neurological sequelae, proposing a possible glial-mediated secondary WM injury in vulnerable

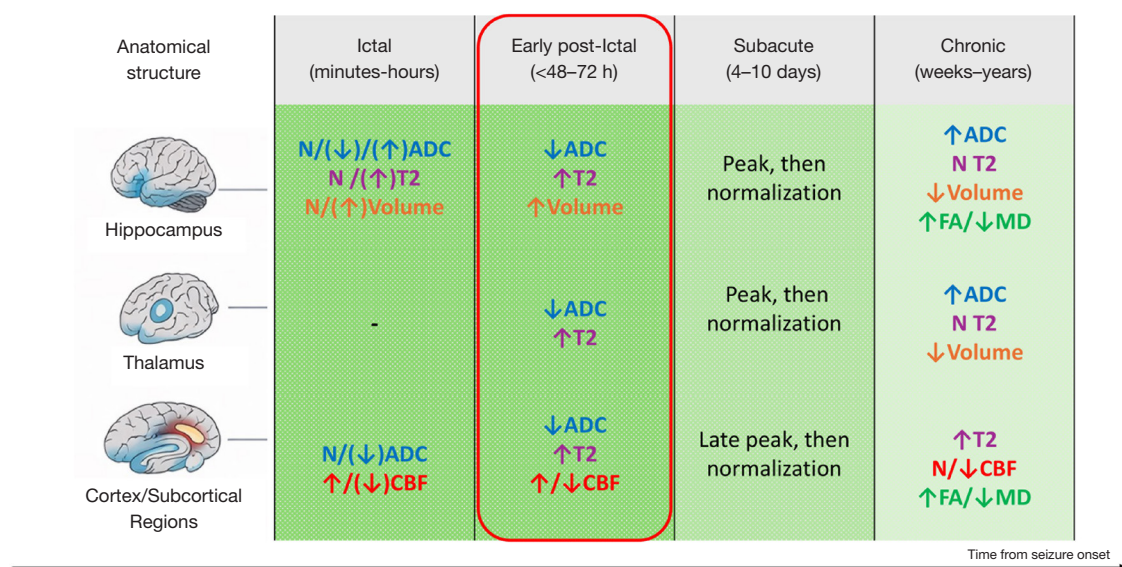


Figure 6 Temporal spectrum of peri-ictal MRI changes in PFS and SE, shown by brain region. Changes in parentheses () indicate patterns that are less commonly observed or less pronounced. Images courtesy of Dr. Domenico Tortora (co-author), from Istituto Giannina Gaslini, Genoa, Italy. ADC, apparent diffusion coefficient; CBF, cerebral blood flow; FA, fractional anisotropy; MD, mean diffusivity; MRI, magnetic resonance imaging; N, normal; PFS, prolonged febrile seizure; SE, status epilepticus.

patients, and showing an imaging pattern very similar to acute leukoencephalopathy with restricted diffusion (ALERD) (54). Similarly, in a small cohort of patients with infection-triggered encephalopathy (ITES), Takanashi *et al.* (43) observed symmetric frontal and fronto-parietal subcortical lesions with transient diffusion restriction and linear T2/FLAIR hyperintensity along U fibers and perirolandic WM, with concurrent glutamate/glutamine elevation and decreased NAA on day 3 in one patient. One study suggested glutamate excitotoxicity as a potential mechanism underlying cytotoxic edema and secondary atrophy in a child with PFS-associated encephalopathy, consistent with excitotoxic myelin injury rather than purely hypoxic-ischemic damage. Normalization of the glutamate/glutamine concentration performed on day 16 could explain the transience of the reduced diffusion, assuming that astrocytic swelling and edema are reversible. Progressively decreased NAA on days 3 and 9, on the other hand, suggests persistent neuronal damage, which may have contributed to the development of cerebral atrophy described at later stages. While these observations are of interest, the derivation of biomarker data from a single patient severely limits generalizability. Two studies (21,36) observed that high seizure burden was associated with higher WM injury in neonates with HIE, particularly involving the internal

capsules. Although the primary pathology is hypoxic-ischemic, these associations raise the possibility that prolonged or recurrent seizures can amplify WM damage in an already compromised hypoxic brain. Subcortical WM appears highly sensitive to both metabolic and excitotoxic stress during prolonged or repeated seizures, easily resulting in inflammatory processes that may evolve into gliotic scarring and impaired myelination (11,32). In line with this, studies using DTI revealed that seizures are not confined to focal regions but involve a large-scale network of WM. Reductions in FA, reflecting disruption of myelin sheaths and axonal membranes, and sustained increases in MD, reflecting reduced cellular density or persistent extracellular expansion, were observed years after seizure onset across multiple brain regions, including the temporal lobes, the corpus callosum, internal capsule, cingulum, fornix, corona radiata, and cortico-spinal tracts (20,26,32). Interestingly, Govindan and colleagues (26) examined patients with left temporal lobe epilepsy (LTLE) and reported compensatory changes to the contralateral side; for instance, the arcuate fasciculus showed functional reorganization toward the right hemisphere, implying reversed asymmetry of language networks. This chronic process of axonal loss and structural reorganization has been observed in patients with ongoing epilepsy and appears to be reversible in those achieving

Table 2 Hypothesized temporal trajectory of seizure-related brain injury on MRI in children

Phase (timeframe)	DWI/ADC	T2	T1 (volumetry, cMRI)	ASL/BOLD-fMRI	MRS	DTI	Pathophysiology
Pre-ictal (20–4 min)	N/A	N/A	N/A	BOLD hyperactivation ipsilateral + contralateral to seizure focus (23)	N/A	N/A	Early hyperexcitability of seizure-generating networks + contralateral inhibition
Ictal (within minutes-few hours)	N (14); possible early ↓ADC hippocampus in severe seizures (24) or ↑ADC if pre-existing vulnerability (39) Possible transient ↓ADC cortex in PFS ≥ 15 min (41)	Possible subtle hippocampal ↑T2 in severe seizures (24,35,37)	Possible early ↑hippocampal volume/swelling in severe seizures (24,33,37)	Marked ↑CBF often corresponding to seizure focus (28,31); possible early transition to ↓CBF in severe seizures (10)	N/A	N/A	Intense neuronal firing increases glucose/oxygen demand and excitotoxicity leading to cytotoxic edema; early vasogenic edema can be detected in vulnerable brains
Early post-ictal (<48–72 h post)	↓ADC hippocampus/thalamus (22,24,29,33,45) Possible early ↓ADC in perirolandic/thalamus in POLG+ (25) and CC splenium in HIE (34) Resolution of transient cortical ↓ADC in PFS ≥15 min (41)	Subtle/mild ↑T2 hippocampus (22,24,35,37,40) Possible early ↑T2 perirolandic/thalamus in POLG+ (25) and CC splenium in HIE (34)	↑Hippocampal volume/swelling (24,33,37)	Persistent ↑CBF (7,28,31) or shift to ↓CBF in severe seizures (10,28)	Transient ↑Glu/Gln, ↓NAA (43)	N/A	Metabolism-perfusion uncoupling: hyperperfusion is no longer sufficient to supply the hyperactive cortex and reverses toward hypoperfusion; glutamate accumulation and cellular damage
Subacute (4–10 days post)	↓ADC/↑DWI hippocampus peak (22,24,29,45) and then begins to normalize (24); possible spread to subcortical WM (14,43) and cortex, including CC splenium in HIE (34) and perirolandic region in POLG+ (25)	Prominent ↑T2 in hippocampus and/or subcortical/cortical region (22,29,36,40,43), including CC splenium in HIE (34) and perirolandic region in POLG+ (25)	N/A Possible post-contrast enhancement seen in animal models/ adult population (15,16)	CBF decrease over days (28,31)	Ongoing Glu/ Gln normalization, progressively ↓NAA (43)	N/A	Gradual transition from cytotoxic to vasogenic edema, reflecting partial tissue recovery and reactive inflammatory response; persistent ↑T2/FLAIR and ↑NAA indicate ongoing inflammation or transition to gliosis; prominent hypoperfusion
Chronic (weeks-years)	Normalization; progressive ↑ADC hippocampus/thalamus (24,27,29,39)	Progressive hippocampal normalization (38) Possible ↑T2 persistence in affected cortex (14)	↓Hippocampal volumes, MTS and asymmetry (24,29,30,35,38,42,44,45); thalamic/ occipital atrophy in POLG+ (25)	N or ↓CBF (28,31)	N/A	↓FA/↑MD in bilateral WM (CC, IC, CST, corona radiata, cingulum, fornix, FOF), hippocampus and temporal lobes in patients with active epilepsy (20,26,27,32)	Tissue remodeling and structural sequelae (e.g. hippocampal sclerosis, cortical atrophy) reflecting permanent neural loss or scarring/gliosis with network reorganization

ADC, apparent diffusion coefficient; ASL, arterial spin labelling; BOLD, blood oxygen level dependent; CBF, cerebral blood flow; CC, corpus callosum; cMRI, contrast-enhanced MRI; CST, cortico-spinal tract; DWI, diffusion weighted imaging; FA, fractional anisotropy; FLAIR, fluid-attenuated inversion recovery; fMRI, functional MRI; FOF, fronto-occipital fasciculus; HIE, hypoxic ischemic encephalopathy; IC, internal capsule; MD, mean diffusivity; MRI, magnetic resonance imaging; MRS, MRI spectroscopy; MTS, mesial temporal sclerosis; N, normal; N/A, not applicable; NAA, N-acetylaspartic acid; PFS, prolonged febrile seizures; POLG, DNA polymerase gamma; WM, white matter.

seizure remission (20), supporting the hypothesis that prompt seizure control may prevent long-term structural and functional deficits. Collectively, these studies are of interest and have been included in our final selection as they propose DTI as a potentially sensitive tool for detecting both reversible and irreversible WM injury in seizure disorders, although evidence remains scarce and mainly derived from very small heterogeneous cohorts lacking standardized acquisition protocols and longitudinal validation.

Cortical signal changes and hemodynamic impairment

Cortical abnormalities provide additional insights into the metabolic and hemodynamic implications of seizures, as they may reflect both local excitotoxicity and network-level effects. However, it remains uncertain whether these changes are truly consequences of seizures or pre-existing substrates that increase seizure susceptibility. Immediately post-ictal cortical diffusion restriction has been described in pediatric patients within the first 1–2 hours after PFS, mostly affecting the frontal and temporal lobes, and resolving within 72 hours without structural sequelae or early epilepsy (41). The observed persistent cortical T2/FLAIR hyperintensity during the subacute phase of FSE, on the other hand, is thought to represent the combined result of cytotoxic edema and transient BBB disruption (14,36). ASL and BOLD-fMRI studies also revealed peri-ictal patterns that usually accompany seizure activity. In neonates, focal ASL increase in CBF appeared as early as 2–3 hours after seizure onset and persisted for several days, reflecting sustained hypermetabolism (31). In their case series, Federico and colleagues (23) observed pre-ictal BOLD activation in cortical regions up to 20 minutes before clinical seizure onset, suggesting that functional network instability may precede EEG-detectable changes and potentially contribute to seizure generation rather than merely reflecting ictal propagation. Although more commonly seen in adults, vascular abnormalities like these are typically transient and evolve from an initial phase of hyperperfusion to subsequent hypoperfusion, mimicking a stroke-like pattern that follows the period of cortical hyperexcitability. In epileptic children with negative structural MRI, ASL has shown a high rate of peri-ictal cortical perfusion abnormalities, with early post-ictal hyperperfusion within the first 24 hours followed by hypoperfusion patterns, emphasizing the importance of appropriate acquisition timing (28). Interestingly, post-

ictal hypoperfusion corresponding to the SOZ, as detected by ASL or CTP, may explain transient neurological deficits such as Todd's paresis. In adult cohorts, Li *et al.* (9) performed CTP within 80 minutes of habitual seizures and identified significant regional hypoperfusion in most cases, showing strong concordance with the clinically defined SOZ, while Gaxiola-Valdez *et al.* (10) reported ASL-detected post-ictal hypoperfusion in a vast majority of patients with drug-resistant focal epilepsy; moreover, the degree of perfusion decline correlated with seizure duration, and ASL localized the SOZ with accuracy comparable to or exceeding that of ictal SPECT and interictal PET. Some authors have been interpreting these findings as transient alterations in neurovascular coupling, where excessive neuronal activity initially drives hyperperfusion and hypermetabolism, followed by a phase of vascular and metabolic exhaustion manifesting as post-ictal hypoperfusion. Existing discrepancies across studies regarding peri-ictal hyperperfusion versus hypoperfusion are likely due to the dynamic and time-dependent evolution of cerebral blood flow after seizures, with outcomes strongly influenced by diverse imaging timing, seizure duration, and methodological heterogeneity across studies (10).

Seizure burden effects in hypoxic-ischemic neonates

Prospective studies have hypothesized the synergistic role of seizure duration and hypoxic-ischemic background injury in determining MRI severity. For instance, in epileptic neonates with HIE undergoing cooling therapy, higher and longer electrographic seizure burden correlated with more extensive T2 hyperintensity and diffusion restriction in the WM (in particular the corpus callosum), basal ganglia, thalamus, and cortex within the first week from onset (21,36). In a retrospective analysis by Nguyen and colleagues (34), the corpus callosum was again found as a highly specific MRI marker of recent neonatal seizures in HIE. These data support the theory that seizure-associated metabolic stress can further exacerbate ongoing hypoxic-ischemic cascades in the immature brain, regardless of therapeutic hypothermia. However, definite conclusions cannot be drawn, as there is considerable overlap between primary HIE-related injury and acute seizure-associated changes on MRI, limiting confident interpretation.

Risk stratification according to age, etiology and seizure type

It is important to highlight that pediatric seizure-related

MRI changes vary substantially depending on age and etiology.

In neonates (0–1 month), studies focus primarily on cases of HIE presenting with SE. Imaging in the acute phase can show reduced ADC in the splenium, highly predictive of recent seizures in these patients (34), in addition to less specific signal changes involving the WM and basal ganglia (21,36), positively correlating with seizure burden and poor neurodevelopmental outcome in some cases (21,34). Only in a small case series of newborns with severe underlying neurological conditions other than HIE, persistent increased CBF has also been described (31).

In infants and very young children (1 month to 3–5 years), seizure type is largely represented by PFS and FSE. Acute MRI often demonstrates hippocampal edema with DWI and T2 hyperintensity (29,33,35,37,39,40,42,45), early cortical/subcortical diffusion restriction (41,43), and occasionally non-specific subcortical WM changes with increased inflammatory biomarkers in neurologically impaired patients (14). Chronically, a subset has been described to develop hippocampal atrophy with asymmetry and MTS (29,30,42,45,35,38), suggesting that a genetic or structural predisposition may amplify the risk of injury. Of note, almost the totality of the included literature about newborns and very young children focuses on changes observed after PFS and FSE (≥ 30 minutes, or ≥ 15 minutes in a few cases); short episodes (< 15 minutes) are virtually unstudied, precluding generalization to this seizure subgroup; moreover, in the context of SE, peri-ictal seizure-induced abnormalities can be largely heterogeneous and often difficult to differentiate from the underlying epileptogenic pathology (18).

In cohorts including older children (> 3 –5 years), on the other hand, there are studies which also include cases of idiopathic epilepsy, shorter or partial seizures and a different variety of etiologic conditions, with more consistent use of advanced imaging techniques. Pre-ictal BOLD-fMRI changes showed prolonged network imbalance in three cases of partial seizures, detectable up to 20 minutes before seizure onset (23). ASL abnormalities were observed in a cohort of MRI-negative subjects with both focal and generalized seizures, showing hyperperfusion within the first 24 hours with moderate seizure onset zone concordance (28). Genetic or metabolic conditions such as POLG can present acutely with transient perirolandic and thalamic injury, followed by long-term volume loss (25).

Persistent hippocampal injury was described in refractory SE following T2 hyperintensity (22), in PFS (44), but also in very few cases of complex seizures with viral prodrome without SE (24). Chronic DTI alterations such as bilateral $\downarrow$ FA and $\uparrow$ MD in main WM tracts are common in TLE (20,26,32), and one study also reported long-term hippocampal microstructural changes (27).

Strengths and limitations

Limitations across included studies

Important gaps remain in understanding seizure-related brain changes in the pediatric population. First, as most research has largely focused on prolonged seizures, evidence regarding the effects of focal short seizures (< 15 minutes) is very scarce, limiting generalizability to this subgroup. Secondly, most studies were limited by predominantly small, heterogeneous cohorts, variable MRI protocols and magnet strengths (1.5- or 3-T), with uneven timing of imaging and frequent lack of longitudinal follow-ups beyond one year; in addition, many studies relied on qualitative or semiquantitative visual assessments rather than quantitative metrics, and advanced imaging modalities were implemented in a limited number of cases. Importantly, some of the included studies involved neonates or children with underlying systemic, metabolic, or genetic conditions, which may independently influence MRI findings and confound attribution to seizures alone. Moreover, seizure etiologies were not always consistently described, and baseline pre-seizure imaging and genetic testing were often unavailable, making it difficult to discern preexisting abnormalities from seizure-induced changes; additionally, integration with molecular biomarkers of excitotoxicity or inflammation was rare. Furthermore, seizure timing frequently relied on witness reports rather than continuous video-EEG, creating uncertainty in differentiating acute versus subacute findings. Lastly, associations with cognitive outcomes were predominantly indirect and remain insufficiently characterized, as standardized neuropsychological evaluations were not always administered across the included records. Further studies employing standardized, multimodal MRI protocols with precisely timed baseline under continuous EEG monitoring, combined with systematic analyses of the cause of the seizures and follow-ups, are required to formulate evidence-based practical guidelines.

Review limitations and potential sources of bias

Several methodological limitations should be acknowledged in the present review. First, the exclusion of the term “convulsion(s)” from the keyword search strategy and the temporal limit of 20 years may have introduced selection bias by omitting a substantial body of more outdated, though relevant literature; moreover, this review was restricted to the pediatric population, although including mixed demographics might have provided additional valuable insights. While this approach aligns with our intent to maintain higher comparability across studies, it should be interpreted as a deliberate restrictive methodological choice. Due to considerable heterogeneity in populations, assessment tools, and follow-up intervals, specific seizure etiologies and cognitive outcomes were not systematically targeted as primary variables of interest. Finally, the absence of a formal quality assessment of the included studies limits the strength of inferences that can be made. Therefore, our proposed outline should be regarded as informative rather than conclusive, serving to summarize the current state of literature and to offer pathophysiological insights that can be derived through multimodal imaging, while also acknowledging the substantial uncertainties that remain.

Conclusions

This review highlights the limited availability of large, well-designed longitudinal controlled studies investigating the spectrum of peri-ictal MRI abnormalities in the pediatric population. While some evidence exists from adult and animal models, particularly rodents, the pediatric brain exhibits unique structural and functional properties that limit extrapolation. Addressing this gap in future research is critical, as understanding the precise temporal dynamics of peri-ictal changes in children may help define therapeutic windows, improving diagnostic accuracy and prognostic assessment. The retrieved literature, primarily based on evaluations following PFS and FSE, reports that early-life prolonged seizures can be associated with imaging changes consistent with cytotoxic edema, inflammation, and structural remodeling. The hippocampus often emerges as the primary site of injury, though basal ganglia, thalamic nuclei, white matter, and cortical networks may also show vulnerability in some cases. Nevertheless, these findings arise from heterogeneous and often highly confounded populations, making it difficult to draw definitive conclusions or determine whether most of the observed changes are a cause or a consequence

of the underlying pathology. As such, current evidence should only be accounted for hypothesis-generation, as it remains insufficient to establish causality or to support routine clinical application. Future works should prioritize methodologically robust studies integrating advanced neuroimaging and electrophysiological monitoring to elucidate these unique developmental patterns, as early detection may be pivotal for mitigating long-term neurologic sequelae.

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Footnote

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