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Review

Epileptiform Activity in Acute Ischemic Stroke and Neurological Deterioration: A Comprehensive Narrative Review

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Abstract: Epileptiform activity (EA), including interictal discharges, lateralized periodic discharges (LPDs), and non-convulsive status epilepticus (NCSE), is increasingly recognized after acute ischemic stroke (AIS). While overt seizures occur in a minority of patients, continuous EEG (cEEG) studies have shown that EA is detected around 40% of patients [1,2]. Evidence suggests EA is not merely an epiphenomenon but an active contributor to early neurological deterioration (END), disability, and post-stroke epilepsy (PSE). The objective of this review is to synthesize evidence on incidence, risk factors, mechanisms, prognostic significance, and management of EA in AIS, with emphasis on neurological deterioration and functional outcome. A narrative review of academic literature was conducted. Data were drawn from more than 40 peer-reviewed studies published between 1993 and 2025, including observational cohorts, multicenter analyses, translational models, and international guidelines. A thorough search of databases including PubMed/MEDLINE and Google Scholar was performed using keywords such as "epileptiform activity" or "non-convulsive status epilepticus" or "seizures" or "EEG abnormalities" and "acute ischemic stroke" or "cerebral infarction" and "neurological deterioration" or "prognosis" or "outcome" or "progressive stroke". Studies providing quantitative data on neurological deterioration and prognosis were prioritized for review. Results from large cohorts demonstrate that EA independently predicts END, poor functional outcome, and mortality. Risk stratification tools such as the SeLECT and SeLECT-EEG models improve prediction of late seizures and epilepsy. While antiseizure medications (ASMs) are indicated for clinical seizures and NCSE, there is no consensus on treating asymptomatic EA. EA represents a clinically relevant but underdiagnosed biomarker of poor prognosis in AIS. Standardization of EEG biomarkers, multicenter validation of predictive tools, and randomized controlled trials are needed to establish whether early detection and targeted intervention can improve outcomes..

Keywords: acute ischemic stroke (AIS), epileptiform activity, neurological deterioration, continuous electroencephalography (cEEG), post-stroke seizures, ictal-interictal continuum (IIC), prognosis, neurocritical care

1. Introduction

Stroke is a neurological impairment from acute vascular injury to the central nervous system, representing a primary contributor to disability and death globally [3]. Romania sees 61,552 new stroke cases annually, with an anticipated 24% rise between 2015 and 2035 [4].

Epileptic seizures are a common stroke complication, causing 11% of adult epilepsy and half of cases over 60 [5]. However, there is a notable absence of dependable clinical guidelines addressing seizure management after stroke [6].

In ischemic stroke, the damaged brain core is surrounded by a recoverable penumbra, but its survival depends on a fragile metabolic balance. Despite medical advances, early neurological decline affects up to 38% of patients [7,8] and is linked to a fourfold higher risk of death or dependency at 3 months compared to individuals without worsening neurological deficits [8]. Early neurological deterioration (END) operationally defined as any measurable decline in neurological status within 72 hours of stroke onset, including worsening motor deficits (≥1-point NIHSS motor subscale increase), decreased consciousness (≥1-point NIHSS consciousness item increase), or new neurological symptoms affects 10-40% of AIS patients and substantially worsens functional outcomes and mortality [3-5]. In roughly half of these instances, hemorrhagic transformation, swelling, recurring ischemia, or systemic medical issues that compromise blood oxygen levels or elevate neuronal metabolism cause neurological decline [9,10]. In the remaining 50% of cases, the fundamental cause of neurological deterioration remains unclear, hindering the creation of suitable management approaches [11]. A lesser-known cause is epileptiform activity, which triggers a "metabolic crisis" that can damage penumbra neurons and worsen neurological outcomes [2].

The emergence of epileptic events is linked to increased death, hindered recovery, and post-stroke epilepsy development [12]. Epileptiform activity includes ictal activity and interictal activity [13], classified as early (within 7 days) or late events (> 7 days) [14-16] per International League Against Epilepsy criteria [5].

Key terms used throughout this review:

- ✓ Epileptiform activity: Spectrum of abnormal electrical discharges including ictal patterns (electrographic seizures), periodic discharges, rhythmic delta activity, and sporadic epileptiform discharges.
- ✓ Ictal-interictal continuum (IIC): EEG patterns falling between clearly ictal and interictal activity, whose clinical significance remains debated.
- ✓ Periodic discharges: Repetitive waveforms at 0.5-3 Hz, classified as GPDs, LPDs, or BIPDs.
- ✓ Epileptiform burden: Cumulative duration or percentage of recording time with epileptiform discharges (variably defined) [1].
- ✓ Neurological deterioration: As defined above—any decline within 72 hours of stroke onset [10].
- ✓ NCSE: Continuous/near-continuous electrographic seizure activity >30 minutes without motor manifestations [2].

Understanding the relationship between END and epileptiform activity is clinically important because: (1) epileptiform patterns are potentially modifiable through antiseizure medications; (2) detection requires specialized monitoring not universally available; and (3) treatment decisions remain controversial due to limited high-quality evidence.

Continuous EEG helps diagnose and monitor seizures during acute stroke, guiding treatment and improving outcomes. Early detection is crucial, as missed epileptiform activity can worsen neurological decline [5]. A 2017 study found that continuous EEG didn't increase ictal activity detection but doubled interictal findings [13]. Continuous EEG is recommended by the Society of Neurophysiology for acute brain injury with altered or unclear mental status and suspected seizures [17]. Epileptic events after stroke range from 2.2% to 67%, depending on timing [6]. Continuous EEG detected

seizures in 37% of stroke patients, mostly non-convulsive [18]. Additionally, lateralized periodic discharges strongly predicted seizure recurrence, unlike other EEG patterns [19].

Treatment choices should be guided by specific EEG patterns. Periodic discharges may result in lasting neuronal damage; hence, anticonvulsant drugs are often recommended [6]. A 2017 European Stroke Journal publication notes that primary preventive use of drugs for early epileptic seizures during the acute phase is permissible and should be discontinued after this initial phase [20].

Evidence is limited, and the European Stroke Association advises against routine anticonvulsant use for seizure prevention. Antiepileptic drugs are typically delayed unless a second seizure occurs, though stroke-related brain damage makes early treatment a case-by-case decision [6].

Therefore, this narrative review comprehensively summarizes data about incidence, risk factors, mechanisms, prognostic significance, and management of epileptiform activity in acute ischemic stroke, emphasizing neurological deterioration and functional outcome.

1. Methods

The scope of this narrative review is to synthesize current evidence on epileptiform activity in AIS and its association with END. Unlike systematic reviews or meta-analyses, narrative reviews do not employ exhaustive search strategies or formal quality assessment tools. Consequently, our conclusions reflect expert interpretation of available literature rather than systematic-review-level certainty. Important limitations characterizing this field include:

- ✓ Heterogeneous study designs: Most evidence derives from observational studies with inherent selection bias and confounding.
- ✓ Variable END definitions: Studies employ inconsistent criteria for defining and measuring neurological deterioration.
- ✓ Inconsistent EEG classification: Epileptiform pattern categorization varies across institutions and studies, limiting comparability.
- ✓ Limited randomized controlled trial data: Few RCTs address treatment of epileptiform activity in stroke.
- ✓ Institutional resource variability: EEG monitoring access, duration, interpretation expertise, and triage criteria vary substantially.
- ✓ Small sample sizes: Many studies include limited patient numbers, reducing statistical power and generalizability.

These methodological constraints limit the strength of recommendations and underscore the need for rigorous prospective studies.

Given the educational and synthesizing nature of this review, we employed an adaptive PRISMA methodology to ensure transparency, reproducibility, and completeness in the literature selection and reporting process, while acknowledging the inherent selectivity of a narrative format.

2.1. Literature Search Strategy

The literature search was conducted up to November 2025. The primary objective was to identify pertinent literature covering the core topics: acute ischemic stroke, epileptiform activity (including seizures, non-convulsive status epilepticus, and periodic discharges), and neurological deterioration/prognosis.

2.1.1. Databases and Search Terms

The search spanned major electronic databases, including PubMed/MEDLINE and Google Scholar. Key search terms and their combinations, informed by the review's title and scope, included:

- ✓ "Epileptiform activity" OR "non-convulsive status epilepticus" OR "seizures" OR "EEG abnormalities"

- ✓ AND "Acute Ischemic Stroke" OR "Cerebral infarction"
- ✓ AND "Neurological deterioration" OR "prognosis" OR "outcome" OR "progressive stroke"

2.1.2. Eligibility Criteria (Inclusion/Exclusion)

Since this is a narrative review, the selection was guided by the need to include literature that provides a robust foundation for the narrative's key themes.

Inclusion Criteria:

- ✓ Peer-reviewed articles (e.g., original research, systematic reviews, meta-analyses, and comprehensive guidelines) directly address the relationship between epileptiform activity and acute ischemic stroke outcomes or neurological deterioration.
- ✓ Articles defining key concepts (e.g., stroke, epilepsy, guidelines for management).
- ✓ Studies on pathophysiology, diagnosis (especially continuous EEG), and treatment of post-stroke seizures/epileptiform activity.

Exclusion Criteria:

- ✓ Articles primarily focus on hemorrhagic stroke, chronic stroke complications (unless relevant to prognosis), or non-human studies, unless they provided critical mechanistic insights.

2.1.3. Selection Process and Synthesis

Relevant titles and abstracts were initially screened by the author. Full-text articles were then reviewed to ensure they contained content critical for building the comprehensive narrative.

- ✓ The process involved identifying pivotal studies (e.g., Dhakar et al., 2020; Scoppettuolo et al., 2019) that established the core relationship, followed by incorporating key guidelines and definitions (e.g., Sacco et al., 2013; ILAE, 2014; Jauch et al., 2013), and finally, integrating literature on diagnostic tools (e.g., continuous EEG, Claassen et al., 2004) and prognostic models (e.g., Galovic et al., 2018; Schubert et al., 2024).
- ✓ The synthesis was structured to systematically address the definition of key concepts, the epidemiology and risk factors, the role of continuous EEG, and the impact of epileptiform activity on neurological deterioration and long-term prognosis.

2.2. Data Extraction and Reporting

Information was extracted and synthesized thematically. Due to the narrative nature, the focus of extraction was on key findings, clinical implications, consensus statements, and mechanistic explanations. Specific attention was paid to the literature distinguishing between different forms of acute epileptiform activity (acute symptomatic seizures, non-convulsive status epilepticus, and subclinical epileptiform discharges) and their differential impact on patient outcomes.

Thematic Sections: The narrative review was developed around distinct themes, including:

1. Definitions and Epidemiology of Post-Stroke Seizures.
2. Mechanisms Linking Ischemia to Epileptogenesis.
3. The Role of Continuous EEG Monitoring.
4. Impact on Neurological Deterioration and Prognosis.
5. Management Strategies.

The final set of references (as provided above, n=48) represents the body of literature that informed the final comprehensive narrative. The above search process and strategy are illustrated in the flowchart in Figure 1.

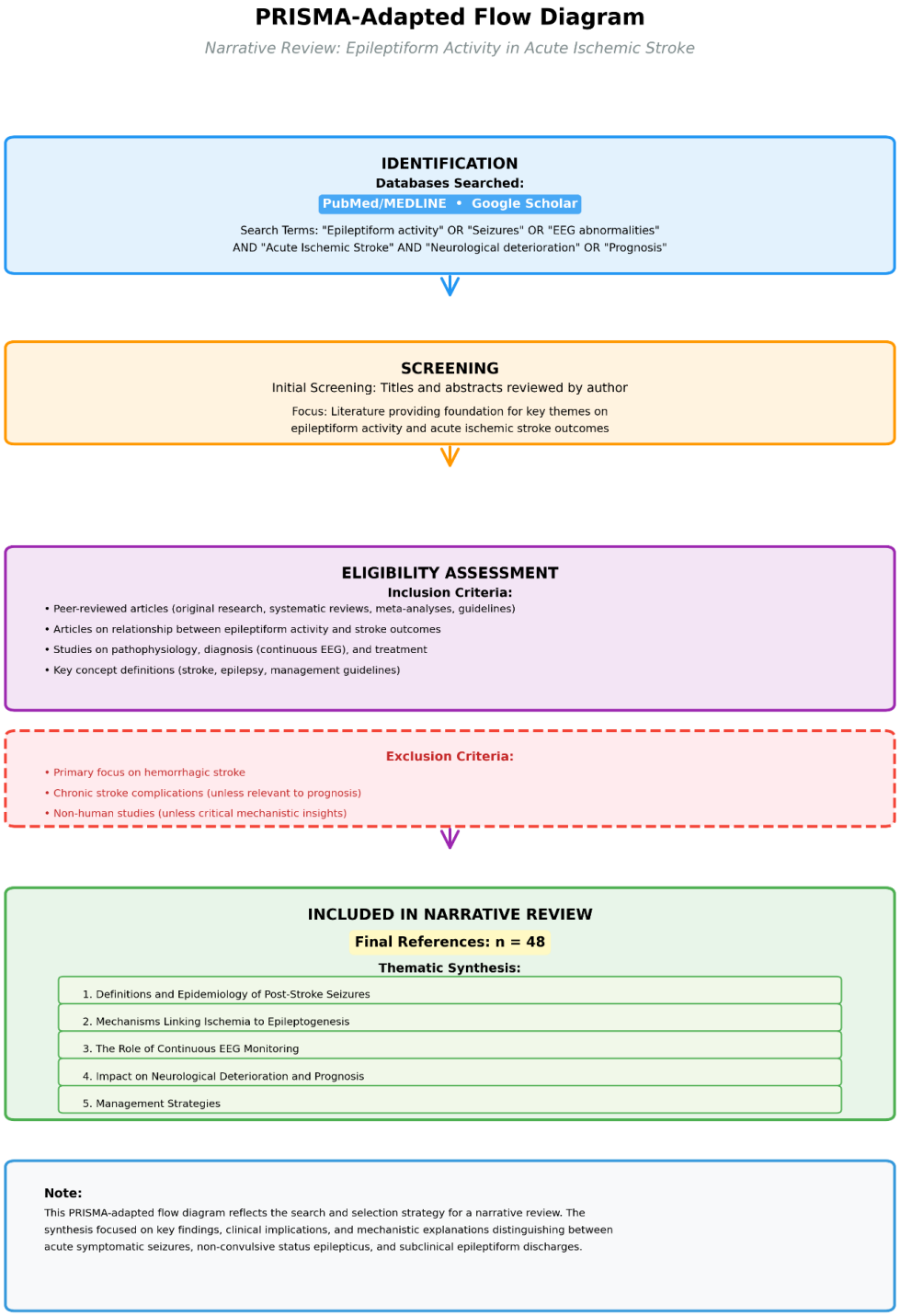

Figure 1. PRISMA-Adapted flow diagram for this narrative review

2. Results

3.1. Epidemiology of seizures and epileptiform discharges after stroke

3.1.1. Early seizures: incidence, risk factors.

The International League Against Epilepsy (ILAE) specifies that post-stroke epileptic seizures (ES) are those that take place within 7 days following an acute stroke event.

Experiencing early ES is linked to poorer stroke results, longer durations of hospital stay, and a greater chance of developing late epileptic seizures. Early ES complicate stroke care and occur in about 7% of cases. Risk is higher with hemorrhagic strokes, severe strokes, cortical involvement, and hemorrhagic transformation [21].

A study found early ES in 6.6% of stroke patients, with no differences by age, sex, or common risk factors, except higher risk in those without hypertension. Stroke location, severity, and treatment type had no impact [22].

A prospective study of 638 first-time stroke patients demonstrated that 4.8% developed early ES, with increased incidence in cases involving cortical lesions, severe strokes, and hemorrhagic transformation of ischemic infarcts. Early ES rates were comparable between ischemic and hemorrhagic stroke subtypes and showed no association with increased mortality or disability at discharge. Multivariate analysis identified hemorrhagic transformation as an independent predictor of early ES. These findings indicate that approximately 5% of acute stroke patients experience early seizures, which, while not affecting short-term outcomes, correlate with specific stroke characteristics [23].

Table 1 summarizes the risk factors mentioned above because it is of interest that early ES be followed in patients at high risk of developing them.

3.1.2. Late seizures and post-stroke epilepsy: incidence, risk factors.

The current definition provided by the ILAE stipulates that experiencing just one late ES is enough to classify the condition as structural epilepsy. This is because the likelihood of another seizure happening within the subsequent decade is substantial, exceeding 60% [24]. Studies show varying figures regarding how often post-stroke seizures occur, with incidence rates ranging from 2% to 20% [3] and the occurrence of PSE also varies, reported between 6% and 15% [21].

Late ES can occur weeks to years after a stroke, driven by gradual epileptogenic changes. The SeLECT score predicts late ES risk using five stroke-related factors, with higher scores indicating greater risk. The highest score (9 points) indicated a 63% chance of late ES occurring within 1 year and over an 80% chance of developing epilepsy within 5 years post-stroke. This tool may help identify ischemic stroke patients who could benefit most from future epilepsy prevention therapies [25].

Risk factors identified for late post-stroke seizures (LPSs) included total anterior circulation infarct, partial anterior circulation infarct, a history of acute symptomatic seizures, using antidepressants when the LPS occurred, hemorrhagic infarct, being male, and having hyperglycemia [26].

Furthermore, patients who have experienced ischemic strokes a while ago (more than 2 weeks prior), are taking multiple anti-epileptic drugs (three or more) during follow-up visits and exhibit sharp waves or spikes on follow-up EEG are at a higher risk of experiencing another seizure if they initially had Acute Symptomatic Seizures (ASyS) when first examined [27]. Moreover, cortical strokes, especially in the anterior circulation and with lesions ≥70 ml, significantly increase seizure risk [28]. Table 1 emphasizes the need for more careful patient assessment for the documented use of cEEG.

Taking these aspects into account, the development of post-stroke epilepsy is clinically important as careful monitoring of patients who are susceptible to developing it is desired.

Table 1. Key risk factors for post-stroke seizures and epilepsy

Timing	Clinical factors	Imaging/laboratory	Treatment-related
Early seizures	Severe stroke [21,23] No hypertension [22]	Cortical involvement Hemorrhagic transformation [21,23]	-
Late seizures	Male gender [26] Prior stroke (>2 weeks) [27] History of ASyS [26,27]	Anterior circulation infarct Hemorrhagic transformation Hyperglycemia [26] Large lesion (≥70 ml) [28] Epileptiform EEG activity [27]	Antidepressant use [26] ≥3 AEDs during follow-up [27]
Post-stroke epilepsy (pse)	Male gender [26] Severe stroke [21,23] History of ASyS [26,27] High SeLECT score [25]	Cortical involvement [21,23,28] Anterior circulation territory [26,28] Hemorrhagic transformation [23] Hyperglycemia [26] Large lesion (≥70 ml) [28]	-

3.1.3. Incidence of subclinical epileptiform discharges (data from continuous EEG studies).

EEG is the key tool for detecting epileptic activity and often shows general slowing in acute ischemic stroke. Extended EEG use has revealed recurring patterns like spikes and periodic discharges linked to higher seizure risk. The clinical meaning of these patterns remains unclear, and early ES rates vary due to study differences. In a study of AIS patients treated within 72 hours, 45.3% showed early abnormal electrical activity (EA), mostly as inter-epileptiform discharges (95.8%) and periodic patterns (16.9%). EA was linked to higher NIHSS scores, female sex, and hypertension. At three months, worse outcomes were independently associated with EA and elevated NIHSS scores [29].

In contrast, a study of 69 stroke patients without seizures found EEG abnormalities in 58%, mainly localized deceleration (43.5%), linked to older age ($p = 0.021$). Irregular EEGs, especially widespread deceleration, were associated with higher NIHSS scores and hemorrhagic transformation. Widespread, but not localized, deceleration correlated with clinical decline ($p = 0.003$). No EEG abnormalities were seen in patients with lacunar strokes [30].

Also, a 2020 study of 143 ischemic stroke patients found epileptiform discharges in 46.9%, with 86.7% showing significant disability. These discharges were linked to poorer outcomes at hospital discharge. While current guidelines for managing stroke offer a tentative endorsement for EEG monitoring in ischemic stroke patients [17], there exists a subset of individuals for whom cEEG monitoring should be more routinely considered [31].

To summarize, irregular EEG readings in general, and widespread deceleration specifically, are related to a worsening of clinical condition after an acute ischemic stroke. The usefulness of routine EEG lies in its role as a straightforward diagnostic tool for evaluating stroke patients, especially concerning predictions of short-term outcomes.

3.2. Pathophysiological Mechanisms

Seizures after stroke arise from poorly understood mechanisms. Early seizures reflect acute biochemical disruptions like glutamate release, ion imbalance, and oxidative stress while late seizures result from long-term structural brain changes [32].

3.2.1. The pathogenic mechanisms of early-onset epileptic seizures in AIS

Because patients who have early-onset seizure don't develop a consistent pattern of epileptic activity and the brain has the ability to heal itself, their condition is classified as seizures rather than epilepsy.

3.2.1.1. Ion channel dysfunction

The lack of blood flow and oxygen to the brain caused by a stroke can make nerve cell membranes unstable and disrupt the metabolism of neurons. This acute brain injury can then lead to the failure of the sodium pump, which causes more sodium ions to enter the cell and depolarize the membrane potential. When the membrane's potential reaches a certain level of depolarization, calcium channels are activated, and calcium ions quickly flow into the cell, increasing the concentration of calcium inside the cell, mechanisms also highlighted in Figure 2. This causes neurons to become overexcited, leading to excitatory toxicity and the gradual loss of function of suppressor cells in the area. When these local suppressor cells are no longer able to stop the spread of rhythmic, hypersynchronous discharges, seizures can happen.

3.2.1.2. Neurotransmitter imbalance

Early-onset seizures can also be triggered by an imbalance of neurotransmitters. The lack of blood flow and oxygen to brain tissue after a stroke causes a large amount of glutamate to be released. This over-activates the glutamate receptors on the postsynaptic neuron, making neurons more excitable and putting the brain in a state of irritability. This makes patients more likely to have seizures.

Stroke reduces GABA receptor function through decreased expression of the $\alpha 1$ subunit and impaired membrane trafficking, weakening inhibitory signaling and lowering the seizure threshold.

Additionally, early ischemic stroke decreases GABA levels and neuronal activity, triggering increased nitric oxide (NO) production via NMDAR activation. Elevated NO subsequently blocks M-type K+ channels, which normally stabilize neuronal resting potential and regulate excitability. This blockade enhances central nervous system excitability, promoting seizure onset, the links between these mechanisms being illustrated for better understanding also in Figure 2.

3.2.1.3. Elevated serum cortisol levels

AIS activates the hypothalamic–pituitary–adrenal (HPA) axis, causing elevated cortisol levels, illustrated as cortisol pathway in Figure 2. This cortisol exacerbates hypoxic injury to neurons and astrocytes, impairs cerebral glucose metabolism, and increases neuronal excitability by upregulating postsynaptic glutamate receptor density. Additionally, elevated post-stroke cortisol reduces neuronal populations in the hippocampal CA3 region and disrupts adult neurogenesis in the dentate gyrus, altering temporal lobe structure and function. These cortisol-mediated changes collectively lower the seizure threshold and promote epileptogenesis following stroke [33].

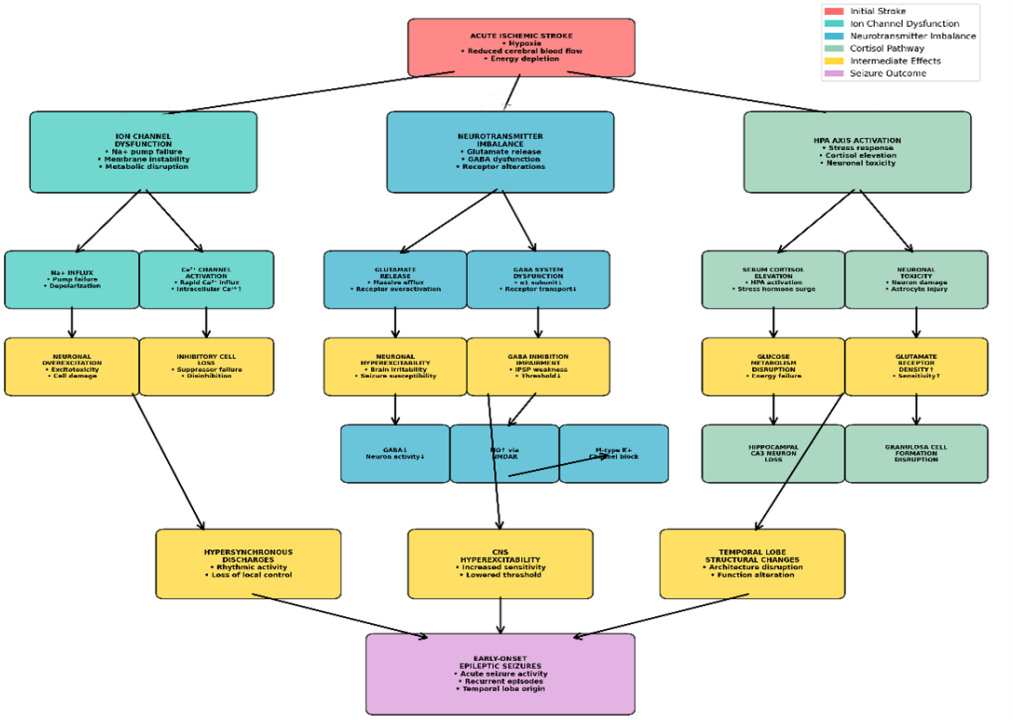

Figure 2. Pathogenic mechanisms early-onset epileptic seizure following ischemic stroke

3.2.2. The pathogenic mechanisms of late-onset epileptic seizures

Post-stroke epileptogenesis involves molecular and cellular remodelling that increases neuronal excitability and generates recurrent seizures. Key neuronal changes include apoptosis, membrane property alterations, mitochondrial and receptor dysfunction (particularly GABA receptor downregulation), synaptic pruning, and aberrant axonal sprouting. Blood-brain barrier disruption permits albumin extravasation, activating astrocytes and microglia, which elevates extracellular glutamate, releases inflammatory mediators, and further compromises barrier integrity. Thrombin and its protease-activated receptor 1 (PAR1) contribute to maladaptive plasticity, producing persistent structural changes that alter neuronal signaling and circuit function, pathways highlighted in Figure 3. Post-stroke gene expression changes additionally promote epileptogenesis by impairing neuroprotection, dysregulating synaptic plasticity, enhancing excitability, and increasing glial scarring. These interconnected pathological processes collectively remodel neuronal network structure and function, culminating in spontaneous recurrent seizures [34].

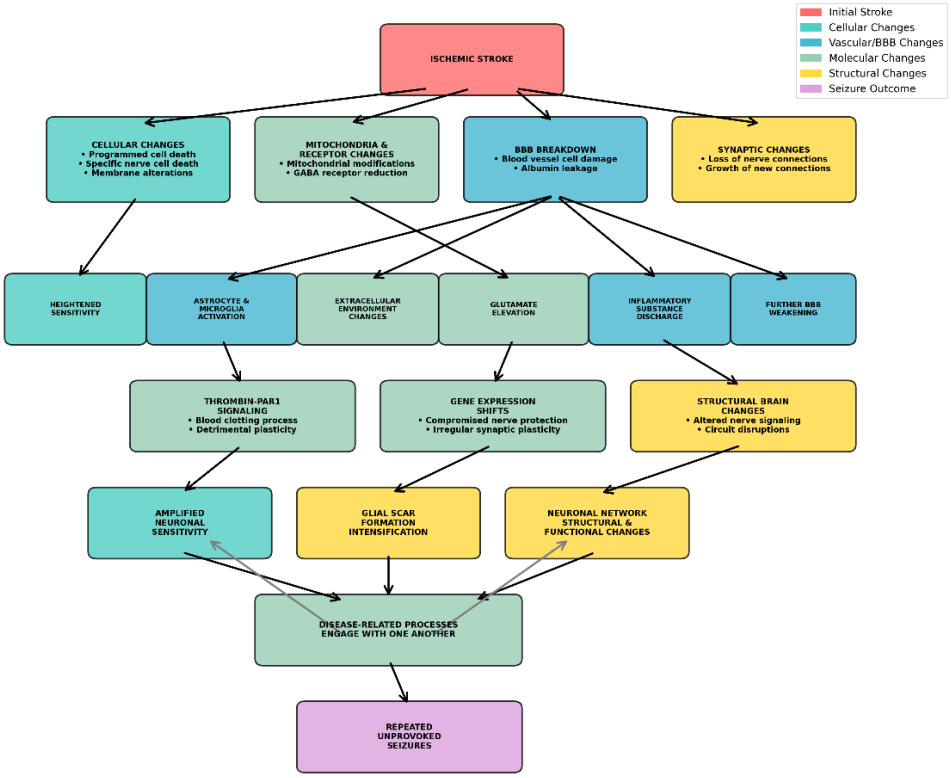

Figure 3. Pathogenic mechanisms late-onset epileptic seizure following ischemic stroke

3.3. Epileptiform discharges and early neurological deterioration (END)

Continuous EEG and quantitative EEG detect frequent subclinical epileptiform activity after AIS, correlating with END, worse short-term outcomes, and increased post-stroke epilepsy risk; routine AED prophylaxis remains unsupported.

END represents a critical endpoint following large vessel and other ischemic strokes, potentially reflecting infarct progression, hemorrhagic transformation, metabolic derangements, or ictal activity. While EEG changes correlate with END detection and outcomes, standardized operational definitions remain lacking across studies and devices.

Continuous and quantitative EEG modalities can detect END or impending decline earlier than bedside exams in selected populations. A recent study of a quantitative EEG (qEEG) system applied after endovascular treatment for large vessel occlusion showed high diagnostic accuracy for detecting END and highlights qEEG as a monitoring adjunct for early decline after reperfusion therapy [35]. Reviews of cEEG in acute ischemic stroke emphasize its role in detecting nonconvulsive seizures, monitoring cerebral physiology, and informing management in patients with neurological decline [36].

3.3.1. Definition and relevance of END

END definitions vary substantially across studies, complicating synthesis:

- ✓ ≥2-point NIHSS increase within 24-72 hours (most common).
- ✓ ≥4-point NIHSS increase (more stringent threshold).
- ✓ Worsening in specific domains (motor, consciousness).
- ✓ Clinical deterioration requiring escalation of care.
- ✓ Modified Rankin Scale worsening at discharge.

For this review, we adopt the operational definition from Section 1 (any measurable decline within 72 hours), noting specific thresholds when citing individual studies.

END is associated with worse short-term outcomes and is a common trigger to initiate extended EEG monitoring or urgent imaging [35,36].

Quantitative EEG can improve sensitivity for detecting deterioration after thrombectomy/large vessel oclusion and may provide early physiologic warning when clinical exam is limited [35].

However, methodological limitations constrain causal inference:

- ✓ Observational designs cannot establish causality (epileptiform activity may be consequence rather than cause of deterioration).
- ✓ Confounding by stroke severity (larger strokes cause both epileptiform activity and END).
- ✓ Variable control for competing END mechanisms (edema, hemorrhagic transformation).
- ✓ Retrospective ascertainment of END timing relative to epileptiform activity onset.

Despite these limitations, consistent associations across multiple studies suggest epileptiform activity may contribute to END in at least a subset of patients. The strongest evidence exists for NCSE, with weaker and more controversial evidence for isolated IIC patterns.

3.3.2. Continuous EEG monitoring

Continuous EEG identifies electrographic seizures and a spectrum of epileptiform abnormalities that are frequently underdetected by clinical exam alone. Multiple cohort and review studies show that cEEG is the reference for identifying nonconvulsive seizures, periodic/rhythmic patterns, and evolving ictal burden after acute ischemic stroke [36,37].

Major reviews recommend cEEG when seizures are suspected, with unexplained neurologic decline, coma, or impaired consciousness after stroke, and for detection/treatment of NCSE [36]. In unselected acute stroke cohorts undergoing cEEG, epileptic activity occurs more often than clinically appreciated (electrical epileptic activity recorded in ~17% in one series), with stroke severity, cortical involvement, and reperfusion treatments among predictors [37]. Real world administrative data show increasing cEEG utilization for cerebrovascular disease and an association between cEEG use and lower in hospital mortality after adjustment, supporting potential outcome benefit from EEG guided care pathways [38].

In cohorts undergoing cEEG, EEG findings prompted anti-seizure medications (ASMs) changes in a substantial minority (≈20%) of patients, indicating direct clinical influence of monitoring [1].

cEEG monitoring practices vary widely across institutions, influenced by resource availability and clinical philosophy:

- ✓ Liberal approach: cEEG for all severe strokes (e.g., NIHSS >10) or decreased consciousness. Maximizes detection but requires substantial resources.
- ✓ Selective approach: cEEG reserved for unexplained deterioration, suspected seizures, or persistent altered consciousness. Resource-efficient but may miss subclinical events.
- ✓ Hybrid approach: Brief screening EEG for high-risk patients, with extended monitoring if abnormalities detected.

Monitoring duration also varies (6 hours to >5 days). Longer monitoring increases detection but with diminishing returns after 48-72 hours in most patients [35,36]. Optimal duration likely depends on individual patient factors and initial EEG findings.

Critical consideration: Published detection rates reflect the populations and monitoring protocols of tertiary referral centers. Applicability to community hospitals with limited EEG access remains uncertain. Implementation requires consideration of local resources, expertise, and patient population.

3.3.3. Critical EEG patterns

Specific EEG patterns carry distinct prognostic and management implications after ischemic stroke. The literature separates outright electrographic seizures from periodic/rhythmic patterns and interictal epileptiform discharges, each associated variably with seizure risk and outcomes [1,31]. As seen in the comparative Table 2 regarding EEG patterns and the reported outcomes, regardless of the frequency with which they occur in acute ischemic strokes, they are accompanied by short-term neurological deterioration and sometimes by a poor functional prognosis.

Table 2. Comparative table of key EEG patterns and reported association[1]

Pattern	Typical description	Reported frequency in AIS cohorts	Clinical association
Electrographic seizures	Evolving rhythmic discharges meeting seizure criteria	2–8% in unselected cEEG series; higher (up to ~18%) in selected deteriorating/ICU cohorts [37]	Directly linked to worse neurological outcome; treatable target [31,37]
Lateralized periodic discharges (LPDs)/PDs	Repetitive focal periodic complexes over stroke region	Reported as part of epileptiform abnormalities in ~12–25% depending on cohort [1,39]	Strongly predictive of subsequent seizures and associated with need for ASM escalation [1,31]
Rhythmic delta / LRDA	Focal rhythmic slow activity often adjacent to lesion	Common among abnormal EEGs (focal delta and generalized slowing frequent) [39]	Correlates with cortical dysfunction and poorer functional outcomes [39]
Interictal epileptiform discharges (IEDs)	Sporadic spikes/sharp waves	Detected in 17–44% across studies depending on timing and selection [37,39]	Associated with higher long term epilepsy risk and worse discharge/3-month outcomes [40,41]

[1]Statistics and pattern frequencies are drawn from continuous/standard EEG cohorts; variation reflects selection criteria and monitoring duration)

3.3.4 Clinical correlations

Across observational cohorts, both ictal and interictal EEG abnormalities correlate with short term neurological deterioration and longer-term seizure outcomes, but causal effects of treating subclinical patterns on stroke recovery remain unproven. Evidence links EEG burden and specific patterns to discharge outcomes, mortality, and later epilepsy [31,39].

Greater electrographic epileptiform activity burden (daily % of epoch with EA) associates dose dependently with worse discharge neurologic outcomes after severe AIS, independent of clinical covariates [31].

In ICU and neurocritical cohorts, NCSE is common among patients with impaired consciousness after stroke and independently associated with in hospital mortality and poor 3 month outcomes, arguing for active detection and treatment [42,43].

Abnormal EEG findings in the acute phase (regional slowing, sporadic discharges) predict worse functional status at discharge and at 90 days in prospective series [39].

Presence of early EA on cEEG markedly increases the risk of developing post stroke epilepsy during follow up cohorts and is being integrated into prognostic models [40,41].

3.4. Clinical implications

EEG findings after ischemic stroke affect diagnostic clarity, immediate management, and prognostication, but high-quality randomized data to guide prophylaxis or routine treatment of subclinical patterns are lacking. Practical guidance from the literature supports targeted EEG use in high risk or clinically deteriorating patients and cautions against routine prophylactic AEDs absent seizures [36,44].

3.4.1. EEG monitoring who and when

Selecting patients for EEG requires balancing seizure risk, stroke severity, and resource availability. Reviews and cohort studies recommend EEG (routine or continuous) when clinical exam is unreliable, when cortical involvement or high NIHSS raise seizure risk, or when unexplained deterioration occurs [36,44].

Consider cEEG for: cortical strokes, high NIHSS, hemorrhagic transformation, new or fluctuating deficits, stupor/coma, or after reperfusion when neurological trajectory is uncertain [36,37,43,44].

Early routine EEG (first 72 h) can predict later epilepsy and identify acute symptomatic seizures missed clinically; studies performing serial EEGs in the first week detected exclusive electrographic seizures in >20% of patients with early seizures [42,44].

Point of care and qEEG provide pragmatic bedside options for stroke code evaluations and post endovascular treatment monitoring when rapid answers are needed [35,45].

3.4.2. Antiepileptic prophylaxis

Randomized trial evidence does not support routine prophylactic AED use after stroke, and major reviews and guidelines advise against universal prophylaxis because benefits have not been demonstrated, and harms are possible. The Cochrane update found insufficient evidence to recommend AEDs for primary or secondary seizure prevention after stroke [46], and expert reviews echo that routine prophylaxis is not established [36].

Two randomized trials (valproate, short course diazepam) showed no robust reduction in post stroke seizures overall; subgroup signals were inconsistent and overall certainty was low to moderate—routine prophylaxis is not supported by current randomized controlled trials evidence [46].

A study of 3792 ischemic stroke patients found 124 (3.3%) developed epilepsy—48 early and 76 late cases. Early seizure recurrence was 43% without treatment vs. 34% with anticonvulsants. Late seizures recurred in 100% of untreated patients vs. 43% of treated ones, showing long-term benefits of targeted anticonvulsant use [47].

Reviews caution against routine prophylaxis and recommend individualized decisions based on cortical location, hemorrhage, or high seizure burden when present [32,36]. Further randomized control trials (RCTs) are required to identify subgroups that might benefit [46].

3.4.3. Treatment of subclinical epileptiform discharges

Management of subclinical EEG patterns on the "ictal–interictal continuum" (IIC) is controversial; the literature demonstrates that these patterns predict seizures and worse outcomes but lacks definitive trials proving that aggressive treatment of non seizure IIC patterns improves neurological recovery. Expert reviews and cohort analyses therefore leave room for clinician judgment [31,36].

IIC patterns (PDs, rhythmic delta) increase subsequent seizure risk and are associated with poor outcomes in observational cohorts, making them potential therapeutic targets [31,37].

No randomized controlled trial in the reviewed literature demonstrates that treating isolated IIC patterns alters infarct growth or functional recovery; authors call for comparative studies to test whether EEG guided treatment improves outcomes [31,37].

Many centers escalate ASMs when high risk patterns or electrographic seizures are detected; such escalation changed management in ≈20% of a cEEG AIS cohort [1] and predicts longer term ASM continuation [48].

3.4.4. Choice of AEDs

There is limited stroke specific evidence guiding AED selection; decisions commonly follow general epilepsy principles while accounting for stroke comorbidity, drug interactions, and hemodynamic effects. The Cochrane dataset includes older agents (diazepam, valproic acid) in trial contexts but provides no modern comparative evidence favoring specific new generation agents for prophylaxis or treatment of subclinical patterns [46].

No high quality RCTs compare specific AED agents for primary prevention after ischemic stroke in the reviewed studies; diazepam and valproate were studied historically without clear benefit [46].

Observational work shows chronic ASM use is common after electrographic ASyS and is associated with downstream long-term treatment, underscoring the need for careful initiation and planned re assessment [48]

Choose agents considering cardiovascular profile, drug–drug interactions (antiplatelets, anticoagulants), renal/hepatic function, and cognitive side effect risk; the reviewed studies endorses individualized regimens rather than a single universal first line AED [32,48].

3.4.5. Management and Guidelines

Existing literature suggests potential benefits of targeted cEEG use and treating electrographic seizures/NCSE, though guideline-level recommendations on prophylaxis or management of IIC patterns remain limited. Several authors and systematic reviews explicitly call for trials to define optimal EEG-guided treatment algorithms [32,36].

cEEG may be considered when nonconvulsive seizures/NCSE are suspected or when unexplained neurological decline occurs. While observational data suggests that detected NCSE should be treated, this recommendation lacks support from randomized controlled trials [36,38].

Management of PDs/IIC patterns and the role of prophylactic AEDs remain areas of significant uncertainty due to absence of randomized trial evidence. Major reviews recommend against routine prophylaxis and highlight the critical need for comparative effectiveness studies and randomized trials [36,46].

The national increase in cEEG use contrasts with large practice variation. Authors have proposed but not validated through controlled trials standardized triage protocols, access expansion, and EEG-guided care pathways [36,38].

Key limitations: No randomized controlled trials exist to guide treatment of isolated IIC patterns, optimal antiseizure medication selection in stroke populations, or the efficacy of prophylactic approaches. Current recommendations reflect expert opinion and observational data rather than high-quality evidence.

3.4.5.1 Practical Barriers and Cost-Effectiveness

Real-world implementation of cEEG monitoring faces substantial barriers:

- ✓ Equipment costs: cEEG systems require significant capital investment.
- ✓ Staffing: 24/7 neurophysiology interpretation and EEG technologist coverage.
- ✓ ICU bed availability: Monitoring often requires ICU-level care.
- ✓ Expertise: Interpretation of complex patterns requires specialized training.

Cost-effective analyses are sparse. Preliminary data suggest targeted monitoring in high-risk patients may be economically justifiable if treatment improves outcomes [41,42]. However, comprehensive health economics research incorporating equipment, staffing, and opportunity costs is needed to guide resource allocation decisions.

3.4.5.2 Practical Clinical Algorithm

The following algorithm from Figure 4 synthesizes evidence reviewed above into a practical clinical pathway. Each step aligns with discussed data, though clinicians must adapt recommendations based on local resources and individual patient factors.

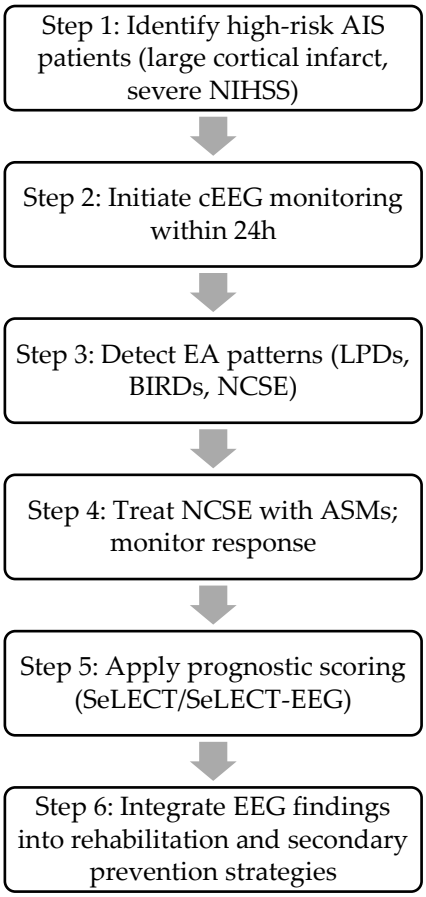

Figure 4. Proposed algorithm for detection and management of EA after AIS

3.5. Future research directions

Advances in monitoring technology and prognostic tools create clear research priorities: validate EEG biomarkers as trial enrichment tools, integrate bedside devices into stroke workflows, and test whether EEG guided intervention alters brain injury and recovery.

3.5.1. AI algorithms and automatic detection of epileptiform patterns

Automated detection and AI assisted interpretation can accelerate recognition of epileptiform events, but there are no primary studies validating AI algorithms for post stroke EEG detection. Therefore, specific claims about AI performance in this setting cannot be supported from these papers. Future work should focus on algorithm validation against human cEEG reads and outcome linkage.

3.5.2. Portable EEG and bedside monitoring

Portable/point of care EEG and simplified qEEG systems show promise for rapid bedside detection of nonconvulsive seizures and for monitoring END. Recent studies demonstrate feasibility and clinically actionable detection of seizures in stroke evaluations [35,45].

A real-world series using a rapid response EEG device in stroke codes detected seizures/highly epileptiform patterns in a meaningful fraction of patients (including

stroke and stroke mimic populations), demonstrating utility for acute differential diagnosis [45].

Quantitative EEG applied after endovascular treatment (EVT) demonstrated high diagnostic accuracy for detecting END, suggesting a role in post reperfusion monitoring and early triage [35].

Prospective trials comparing point of care/qEEG strategies to standard care, and studies establishing optimal electrode montages and alert thresholds, are required [35,45].

3.5.3. Ongoing randomized studies

Randomized trials are essential to test whether EEG guided detection and treatment of subclinical activity change outcomes, but the reviewed literature contains no completed randomized trials specifically addressing aggressive treatment of IIC patterns or prophylactic AEDs for ischemic stroke beyond older small trials summarized in systematic review. The Cochrane review stresses the need for larger contemporary RCTs [46].

No contemporary RCTs in the supplied reviewed studies address routine prophylaxis or randomized EEG guided treatment of subclinical epileptiform activity; authors call for well designed trials with seizure and functional endpoints [31,46].

3.5.4. Multimodal integration EEG perfusion CT MRI serum biomarkers

Integrating EEG with perfusion imaging and biomarkers could improve pathophysiologic phenotyping and trial enrichment, and several reviews recommend correlational research to exploit EEG blood flow coupling. The reviewed articles include calls for such multimodal studies but limited empirical integrative data [36,29].

EEG frequency/amplitude relate to cerebral blood flow and may provide bedside physiologic readouts complementary to perfusion CT/MRI [36].

Early electrographic biomarkers improve epilepsy risk prediction beyond clinical models (SeLECT EEG development), illustrating the value of integrating EEG metrics into prognostic models [41].

Reviews highlight candidate tissue and imaging markers (e.g., cortical superficial siderosis) and urge combining these with EEG signatures to stratify epileptogenesis risk and to enrich prevention trials.

4. Integrated discussion

Across prospective cohorts, retrospective series, and reviews, EEG especially continuous or prolonged monitoring reveals a higher-than expected prevalence of electrographic epileptiform activity after acute ischemic stroke and provides prognostic information not captured by clinical or imaging data alone. However, high quality interventional evidence that EEG guided escalation or prophylaxis improves functional recovery is absent, leaving key clinical questions unresolved. Multiple studies demonstrate that a substantial portion of post stroke EA is electrographic and clinically occult; point of care, routine, and cEEG identify seizures/IIC patterns that change management in a notable minority of patients [1,44,45].

Higher electrical epileptiform burden and specific patterns (LPDs, frequent IEDs, regional slowing) are independently associated with worse early and 3-month outcomes as well as increased risk of later epilepsy; these associations persist after adjusting for classical clinical covariates such as NIHSS and infarct location [31,39,41]. Nonconvulsive status epilepticus in stroke patients associates with higher hospital mortality and unfavorable outcomes; consensus is to detect and treat NCSE, for which cEEG is the diagnostic standard [36,42,43]. Observational signals (treatment changes, reduced mortality associated with centers that use cEEG) suggest potential clinical benefit from EEG guided care, but confounding and heterogeneity preclude definitive causal conclusions; RCTs are lacking and prioritized by reviewers [31,38,46].

Electrographic acute symptomatic seizures frequently prompt inpatient ASM initiation that is often continued long term even when subsequent unprovoked seizures do not occur highlighting the need for protocols to reassess long term ASM indications and for trials to define optimal durations [48]. Portable and quantitative EEG solutions improve the feasibility of early EEG in stroke workflows and show potential for real time END detection and triage, making future implementation studies important [35,45].

4.1. Limitations and heterogeneity across studies

Many studies are single center or enriched for severe strokes/ICU patients, raising incidence variability. Differences in montage, monitoring duration, and classification of patterns (IIC criteria) complicate cross study comparisons [36]. Studies report multiple endpoints (in hospital mortality, discharge mRS, 3-month outcome, development of epilepsy), and few evaluate functional recovery as a primary randomized endpoint of EEG guided therapy. Prospective randomized trials testing whether targeted treatment of electrographic burden or IIC patterns alters infarct growth and functional outcomes. Standardization of EEG monitoring protocols and reporting in stroke studies to enable meta-analysis and guideline development. Trials of protocolized use of portable/qEEG devices for END detection and post EVT surveillance with pre specified clinical actions and outcomes.

5. Conclusions

Readers should differentiate findings by certainty level:

HIGH-CERTAINTY FINDINGS (supported by consistent observational data or clinical consensus):

- ✓ NCSE requires treatment: Multiple studies and expert consensus support treating electrographic status epilepticus.
- ✓ Epileptiform activity is associated with worse outcomes: Consistent observational evidence across multiple cohorts.
- ✓ cEEG detects non-convulsive seizures: Well-established diagnostic utility in critically ill stroke patients.
- ✓ Severe strokes have higher epileptiform activity rates: Robust association across studies.

LOW-CERTAINTY AREAS (requiring further research):

- ✓ Causal relationship between epileptiform activity and END: Observational associations do not prove causation.
- ✓ Treatment benefit of isolated IIC patterns: No RCT data; optimal management speculative.
- ✓ Specific ASM choice in stroke: Limited comparative effectiveness data.
- ✓ Optimal monitoring duration and triggers: Wide institutional variation without definitive guidance.
- ✓ Cost-effectiveness of routine vs. selective monitoring: Insufficient health economics data.

In summary, epileptiform activity is common in severe AIS and associated with worse outcomes. cEEG monitoring enables detection of non-convulsive seizures that would otherwise be missed. However, the field is constrained by observational study designs, variable definitions, and absence of randomized treatment trials. Future research priorities include: (1) RCTs of IIC pattern treatment; (2) standardized END and epileptiform activity definitions; (3) cost-effectiveness analyses; (4) prospective studies with uniform EEG classification; and (5) biomarkers to identify patients most likely to benefit from monitoring and treatment.

Key takeaways with literature support

- ✓ **Detection**: cEEG/qEEG and point of care EEG increase detection of seizures and high risk epileptiform patterns that are frequently missed on clinical exam alone [35,37,45].
- ✓ **Prognosis:** Burden and specific EEG patterns independently predict poor short-term outcomes and higher risk of post stroke epilepsy, and have been integrated into evolving prognostic models (e.g., SeLECT EEG work) [31,41].
- ✓ **Treatment policy:** NCSE should be treated; treatment of other subclinical IIC patterns remains an area of clinical equipoise and requires randomized evaluation [31,36,46].
- ✓ **Prophylaxis:** Routine primary prophylactic AED use after stroke is not supported by current randomized trial evidence and systematic review [46].
- ✓ **Research and implementation**: Priority areas include randomized EEG guided therapeutic trials, validation of automated detection algorithms, pragmatic trials of bedside EEG workflows, and multimodal biomarker integration to enable targeted prevention and treatment strategies [35,41].
- ✓ **Final clinical recommendation from current evidence**: Use EEG (routine or continuous) selectively in patients with cortical infarction, high NIHSS, impaired consciousness, unexplained deterioration, or after EVT; treat electrographic seizures and NCSE; avoid universal prophylactic AEDs; and enroll patients into trials when available to resolve management uncertainties [35,36,46]. cEEG is not just for detecting seizures, but for monitoring brain physiology and predicting decline.

Author Contributions: For research articles with several authors, a short paragraph specifying their individual contributions must be provided. The following statements should be used "Conceptualization, N,A.M. and C.I.D.; methodology, R.C.; software, C.E.B; validation A.C.; formal analysis, N.A.M.; investigation, R.C. S.M.I..; resources, C.E.B.,S.M.I; data curation, C.I.D.; writing—original draft preparation N.A.M.; writing—review and editing, A.C.; visualization, R.C.; supervision, C.I.D.; project administration, A.C.; funding acquisition, R.C. All authors have read and agreed to the published version of the manuscript." Please turn to the CRediT taxonomy for the term explanation. Authorship must be limited to those who have contributed substantially to the work reported.

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