BALNEO RESEARCH Balneo and PRM Research Journal
English Edition
e ISSN 2734 - 8458 p ISSN 2734 - 844X ROMANIAN ASSOCIATION OF BALNEOLOGY

Research article

Quantitative EEG Indices in Acute Ischemic Stroke Presenting with Epileptic Seizures: A Case Series Analysis

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Citation: Nădejde A.M., Sirbu M.I., Vlad L.E., Roman R., Cuciureanu I.D. - Quantitative EEG Indices in Acute Ischemic Stroke Presenting with Epileptic Seizures: A Case Series Analysis
Balneo and PRM Research Journal ***2026****, 17(1): 972*

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Received: 01.02.2026
Published: 31.03.2026

Reviewers:
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Publisher's Note: Balneo and PRM Research Journal stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

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Abstract: Acute ischemic stroke (AIS) presenting with epileptic seizures represents a diagnostic and prognostic challenge. Quantitative electroencephalography (QEEG) indices, including the Delta/Alpha Ratio (DAR), Delta-Theta/Alpha-Beta Ratio (DTABR), and Brain Symmetry Index (BSI), have emerged as potential biomarkers for outcome prediction in stroke patients, yet their application in seizure-onset stroke remains underexplored. The aim of this article is to characterize QEEG indices and spectral power patterns in patients with acute ischemic stroke presenting with acute symptomatic seizures and compare findings with established literature benchmarks. We present a prospective case series of three patients with acute ischemic stroke presenting with acute symptomatic seizures who underwent continuous video-EEG monitoring (cEEG) within 12 hours of hospital arrival, patients admitted between May 2023 to June 2023 in the Neurology I Clinic of Emergency Clinical Hospital "N. Oblu" Iași. All patients had middle cerebral artery (MCA) territory infarctions with cortical involvement. QEEG indices (DAR, DTABR, BSI) were calculated using standardized processing pipelines (EEGLAB/Brainstorm) and correlated with outcomes, and power spectral density (PSD) analysis was performed to assess frequency band alterations. All three patients demonstrated elevated DAR (range: 4.57-5.37) and DTABR (range: 2.79-5.65) compared to reported normative thresholds. BSI values ranged from 0.09 to 0.29, indicating interhemispheric asymmetry. PSD analysis confirmed increased delta power and decreased alpha power in ischemic regions, with spectrotopographic mapping revealing regional specificity corresponding to vascular territories. All patients experienced acute symptomatic seizures within 5 days of stroke onset. Functional outcomes varied, with discharge mRS scores of 1, 1, and 4, corresponding to SeLECT-EEG prognostic scores of 13%, 77%, and 29%, respectively. QEEG indices in seizure-onset acute ischemic stroke demonstrate patterns consistent with severe cortical dysfunction, with DAR and DTABR values exceeding typical prognostic thresholds. Spectral analysis reveals characteristic slow-wave augmentation in ischemic territories with preserved contralateral rhythms. The combination of quantitative EEG biomarkers and clinical variables may enhance outcome prediction in this high-risk population. Larger prospective studies are needed to validate these findings and establish seizure-specific QEEG thresholds.

Keywords: Acute ischemic stroke, quantitative EEG, acute symptomatic seizures, Delta/Alpha Ratio, Delta-Theta/Alpha-Beta Ratio, Brain Symmetry Index, power spectral density, spectrotopographic mapping, outcome prediction

1. Introduction

One of the primary epileptogenic disorders and the primary cause of seizures in the elderly is acute ischemic stroke (AIS), which has an incidence of 0.07% to 4.2% for post-stroke early seizures (ES) - close temporal association (within seven days)

with stroke (acute symptomatic, provoked, situation-related, or early seizures [ES]) - and 0.10% for post-stroke epilepsy (PSE). Furthermore, these two conditions exhibit distinct long-term prognostic trajectories regarding the decadal risk of seizure recurrence. Specifically, the probability of recurrence is estimated at 20% following a single provoked (acute symptomatic) seizure, whereas it rises significantly to 60% following an initial unprovoked remote symptomatic seizure. Additionally, the occurrence of acute symptomatic seizures serves as a robust clinical predictor, associated with a fourfold increase in the risk of developing post-stroke epilepsy (PSE) [1,2].

The co-occurrence of stroke and seizures complicates clinical management and may indicate more severe cortical involvement, potentially influencing prognosis [3]. Electroencephalography (EEG) has long been recognized as a valuable tool for assessing cerebral function in stroke patients, with quantitative EEG (QEEG) analysis emerging as an objective method for characterizing brain electrical activity and predicting functional outcomes [4,5].

QEEG indices, particularly the Delta/Alpha Ratio (DAR), Delta/Theta-Alpha-Beta Ratio (DTABR), and Brain Symmetry Index (BSI), have demonstrated prognostic value in acute ischemic stroke populations [6-8]. These indices quantify the shift from normal faster rhythms (alpha, beta) to pathological slow-wave activity (delta, theta) that characterizes ischemic brain tissue. There have been reports of significant associations between 30-day NIHSS (National Institutes of Health Stroke Scale) and QEEG indices at 48 hours after stroke. Delta/alpha power ratio (DAR) showed the strongest (positive) correlation; in fact, it was just as significant as the link between outcome NIHSS scores and 48 hours [4]. Subsequent studies have confirmed that elevated DAR and DTABR correlate with larger infarct volumes, higher NIHSS scores, and poorer functional outcomes at 3-6 months post-stroke [4,7,9].

The Brain Symmetry Index (BSI) quantifies interhemispheric differences in spectral power, with values >0.1 considered abnormal and it correlates with NIHSS [10]. Studies have shown that increased pairwise derived BSI (pdBSI) correlates with motor impairment and functional disability, specifically with the modified Rankin Scale (mRS) score at month 6, though findings in the same study have been less consistent for DAR and DTABR [11]. Power spectral density (PSD) analysis complements these ratio-based indices by providing detailed characterization of frequency band alterations, typically revealing increased delta power and decreased alpha power in ischemic territories [12].

We hypothesize that quantitative EEG indices—specifically the delta/alpha ratio (DAR), (delta+theta)/(alpha+beta) ratio (DTABR), and brain symmetry index (BSI)—will demonstrate greater abnormality in seizure-onset stroke compared to non-seizure stroke populations. Based on established associations between EEG background slowing, asymmetry, and post-stroke epilepsy [9,10,11], we predict that seizure-onset stroke patients will exhibit: (1) elevated DAR and DTABR values reflecting increased slow-wave activity associated with epileptogenic cortical dysfunction, and (2) higher BSI values indicating greater interhemispheric asymmetry, which has been identified as an independent predictor of post-stroke epilepsy in the first year following cerebrovascular events [9].

Despite growing evidence for QEEG in stroke prognosis, most studies have focused on unselected stroke populations or post-reperfusion cohorts following mechanical thrombectomy [13,14]. The specific QEEG characteristics of stroke patients presenting with epileptic seizures—a population potentially at higher risk for adverse outcomes—remain poorly characterized. Furthermore, the relationship between QEEG indices, spectral patterns, and functional outcomes in this subset has not been systematically explored.

Seizure-onset stroke represents a distinct clinical phenotype that warrants separate investigation from general stroke populations, as seizures may occur in close

temporal association with stroke or after a variable interval, with acute symptomatic seizures serving as independent predictors of vital outcome and remote symptomatic seizures predicting functional outcome in the first year after stroke [1,6]. Furthermore, early EEG abnormalities in these patients provide independent prognostic information comparable to age and clinical/imaging infarct severity, reflecting the unique pathophysiologic cascade and electrographic signature that distinguishes this subset from broader stroke cohorts [6,9].

This case series presents three patients with acute ischemic stroke presenting with epileptic seizures, all of whom underwent comprehensive QEEG analysis including DAR, DTABR, BSI calculation, and detailed PSD assessment with spectrotopographic mapping. We compare our findings with established literature benchmarks and discuss the potential role of QEEG in risk stratification and prognostic assessment for this clinically important population.

2. Methods

2.1 Study Design and Setting

This prospective case series was conducted at the Neurology I Clinic of Emergency Clinical Hospital "N. Oblu" Iași between May 2023 and June 2023. The study was approved by the institutional review board, and informed consent was obtained from all patients or their legally authorized representatives prior to EEG monitoring. The study protocol was reviewed and approved by the Ethics Committee of the 'Gr. T. Popa' University of Medicine and Pharmacy Iași (Approval No. 297 / 30.04.2023) and the Ethics Committee of the 'Prof. Dr. N. Oblu' Emergency Hospital Iași (Approval No. 4037 / 23.02.2023). The research was conducted in accordance with the principles of the Declaration of Helsinki, and written informed consent was obtained from all participants prior to their inclusion in the study.

2.2 Data Collection

Clinical data were systematically extracted from electronic medical records and included: demographic information (age, sex), stroke characteristics (vascular territory, cortical involvement, ASPECTS score, hemorrhagic transformation), clinical severity scores (NIHSS), treatment details (IV thrombolysis, endovascular therapy, antiseizure medications), EEG findings (background activity, epileptiform discharges, seizures), QEEG indices (DAR, DTABR, BSI), and functional outcomes at discharge (discharge NIHSS, modified Rankin Scale [mRS]).

2.3 Patient Selection

Patients were eligible for enrollment if they met all the following criteria: 1. Confirmed acute ischemic stroke by neuroimaging (computed tomography or magnetic resonance imaging); 2. Symptom onset ≤72 hours at the time of EEG monitoring initiation; 3. Age ≥18 years; 4. Clinical indication for EEG monitoring, defined as one or more of the following: a) Clinically observed seizures: Convulsive events directly witnessed and documented by specialized medical personnel; b) Neurological deterioration: an increase in the NIHSS score by >= 2 points, or the emergence of new clinical symptoms corresponding to the stroke topography that cannot be attributed to findings from concurrent paraclinical investigations; c) Declining level of consciousness: a reduction in the GCS (Glasgow Coma Scale) score by >= 1 point compared to baseline at admission, in the absence of explanatory paraclinical findings; d) Fluctuating or unexplained mental status: rapidly changing or idiopathic alterations in cognitive and behavioral states.

Patients were excluded if they had any of the following: 1. Primary intracerebral hemorrhage (hemorrhagic transformation of ischemic stroke was permitted); 2.

Known history of epilepsy or chronic anticonvulsant medication use prior to the index stroke; 3. Inability to obtain informed consent from patient or legally authorized representative; 4. Life expectancy <48 hours based on clinical assessment; 5. Previous stroke with significant pre-existing disability (modified Rankin Scale [mRS] score >3).

2.4 Neuroimaging Protocol

All patients underwent non-contrast computed tomography (CT) of the head at presentation to exclude hemorrhage and if necessary, in case of neurological deterioration. The Alberta Stroke Program Early CT Score (ASPECTS) was calculated based on neuroimaging.

2.5 EEG Monitoring Protocol

2.5.1 Equipment and Technical Specifications

All EEG recordings were performed using a standardized digital EEG system with the following specifications: **System**: 35-channel digital EEG recording system, including GND (ground) and REF (reference) electrodes; **Sampling rate**: 512 Hz; **Electrode placement:** International 10-20 system with additional electrodes as needed; **Montage**: Standard bipolar and referential montages; **Impedance:** Maintained at <5 kΩ for all electrodes throughout recording; **Electrode type**: Standard scalp electrodes (EasyCap system with 35 plastic electrode mounts).

2.5.2 Monitoring Schedule and Duration

EEG monitoring was initiated according to the following protocol: **Timing**: Within 12 hours of clinical indication (clinically observed seizures, neurological deterioration, declining level of consciousness, fluctuating or unexplained mental status – as detailed above); **Duration**: Minimum 3 hours of continuous monitoring for all patients; **Monitoring mode**: Continuous video-EEG monitoring with real-time review capability.

2.5.3 EEG Interpretation Standards

All EEG recordings were interpreted by trained neurologists with expertise in clinical neurophysiology using standardized terminology from the American Clinical Neurophysiology Society (ACNS). The following EEG features were systematically assessed: background activity (symmetry, dominant posterior rhythm frequency, degree of background slowing) and epileptiform activity (presence, type, location, frequency).

Seizure Definitions:

✓ **Electrographic seizure:** Abnormal paroxysmal event distinct from background, with evolving pattern in morphology, frequency, or spatial distribution, lasting ≥10 seconds.

✓ **Nonconvulsive status epilepticus (NCSE):** Continuous electrographic seizure activity ≥5 minutes without clinical convulsive manifestations.

✓ **Status epilepticus:** Continuous seizure activity (clinical or electrographic) ≥5 minutes or recurrent seizures without recovery of baseline neurological function.

2.6 EEG Data Processing Pipeline

All EEG recordings underwent comprehensive processing and analysis using validated computational methods. The processing pipeline consisted of the following stages described below.

2.6.1 EEGLAB Processing (MATLAB R2025b)

Raw EEG data in European Data Format (EDF) were imported into EEGLAB v2025.1.0 running on MATLAB R2025b with licensed software. The following preprocessing steps were applied: **Channel location import:** Electrode positions were imported according to EasyCap manufacturer specifications for the 35-electrode system; **Re-referencing:** Data were re-referenced to the average reference to minimize reference-related artifacts; **Independent Component Analysis (ICA):** Extended Infomax ICA algorithm (Runica) was applied to identify and separate independent sources of electrical activity; **Artifact rejection:** Artifact neutralization was performed using Independent Component Analysis (ICA) via the Infomax algorithm within the Brainstorm toolbox; components were identified for removal based on a dual-criterion approach: (1) a Pearson correlation coefficient > 0.7 with vertical and horizontal EOG channels, and (2) visual inspection of the spatial topography for characteristic non-neural signatures (e.g., frontal dipoles for blinks or high-frequency lateralized bursts for myogenic activity); on average, 2 +/- 1 components were removed per participant to preserve signal integrity; **Manual artifact cleaning:** Remaining artifacts were manually identified and removed.

2.6.2 Brainstorm Processing

Pre-processed datasets from EEGLAB were imported into Brainstorm software for additional processing: **DC offset correction:** Removal of DC bias from all channels; Ba**nd-pass filtering:** Butterworth filter with passband 1-40 Hz to focus on clinically relevant frequency ranges and remove high-frequency noise; **Power Spectral Density (PSD) computation:** PSD was calculated using Welch's method with appropriate window size and overlap to characterize frequency content; frequency definition was according to MATLAB's FFT (Fast Fourier Transform) defaults; **Spectrotopographic mapping:** Topographic distribution of spectral power was computed at representative frequencies (2.0, 6.0, 10.0 and 22.0 Hz) to visualize regional patterns of delta, theta, alpha, and beta activity.

2.6.3 Quantitative EEG Analysis

The following quantitative EEG metrics were computed from fully processed data using EEGLAB and Brainstorm, and the data exported in Excel format were processed in the Jupyter Lab interface (launched via Anaconda Navigator) with a script written in Python:

✓ **Brain Symmetry Index (BSI):** Quantifies interhemispheric asymmetry in spectral power across frequency bands. BSI values range from 0 (perfect symmetry) to 1 (complete asymmetry). Values >0.1 are considered abnormal [10].

Formula: $BSI = \Sigma|P_{left} - P_{right}| / \Sigma(P_{left} + P_{right})$

where P represents spectral power in corresponding electrode pairs.

✓ **Delta/Alpha Ratio (DAR):** Ratio of delta band power (1-4 Hz) to alpha band power (8-13 Hz). Elevated DAR indicates increased slow-wave activity and is associated with cortical dysfunction [8].

Formula: DAR = Power_delta (1-4 Hz) / Power_alpha (8-13 Hz)

✓ **Delta/Theta-Alpha-Beta Ratio (DTABR):** Ratio of slow-wave power (delta + theta, 1-8 Hz) to fast-wave power (alpha + beta, 8-30 Hz). Higher values indicate greater background slowing [8].

Formula: DTABR = [Power_delta (1-4 Hz) + Power_theta (4-8 Hz)] / [Power_alpha (8-13 Hz) + Power_beta (13-30 Hz)]

2.7 Clinical Management

All patients received standard acute stroke care according to institutional protocols and current guidelines. Seizure management followed established protocols, with levetiracetam as the first-line antiseizure medication (ASM) for acute symptomatic seizures. ASM dosing, duration, and adjustments were determined by the treating neurologist based on seizure control, EEG findings, and clinical status.

2.8 Outcome Assessment

Functional outcomes were assessed using the modified Rankin Scale (mRS) at admission and at hospital discharge. The mRS is a 7-point scale ranging from 0 (no symptoms) to 6 (death), with scores of 0-2 indicating functional independence. Neurological deficit severity was quantified using the National Institutes of Health Stroke Scale (NIHSS) at admission and discharge.

2.9 Prognostic Scoring

The SeLECT-EEG score was calculated for prognostic assessment when applicable. This validated score incorporates clinical and electrographic variables to predict outcomes in critically ill patients undergoing EEG monitoring [15].

2.10 Statistical Analysis

Given the small sample size (n=3), descriptive statistics were used to summarize patient characteristics, QEEG indices, and clinical outcomes. Comparisons with published literature thresholds were performed qualitatively. No inferential statistical testing was performed due to the case series design.

3. Results

3.1 Patient Characteristics

Three patients with acute ischemic stroke presenting with acute symptomatic seizures were identified during the study period, defined below as P001, P002 or P003. The median age was 64 years (range: 58-65), with two males and one female. All patients had cardiovascular risk factors, with hypertension and atrial fibrillation present in all three cases. All patients had middle cerebral artery (MCA) territory infarctions with cortical involvement and no haemorrhagic transformation. Due to their presentation time, there was no thrombolysis or endovascular treatment performed. The median admission NIHSS was 5 (range: 5-18), indicating mild to moderate-severe stroke severity. Two patients had ASPECTS scores suggesting moderate early ischemic changes (5 for P002 and 7 for P001), while one patient had minimal early changes (ASPECTS 9 -P003). The baseline mRS for two of the patients was 2 (P001 and P003) and for the one experiencing the most extensive ischemic cerebral lesion was 5 (P002), resulting in sever disability. The complete patient demographics, clinical characteristics, treatments and outcomes can be seen in Supplementary Table 1.

3.2 Seizure Characteristics

All three patients experienced acute symptomatic seizures occurring at the onset of the ischemic stroke. **Patient 1(P001)** also experienced a focal motor seizure involving the left face with impaired awareness lasting 10-15 seconds, occurring 5 days after stroke onset, like the one at the onset of AIS. **P002** presented with the most severe seizure manifestation, a focal motor seizure affecting the right hemibody with secondary bilateral tonic-clonic generalization, evolving into status epilepticus lasting approximately 30 minutes. **P003** had a generalized tonic-clonic seizure.

All patients underwent continuous video-EEG monitoring (cEEG) initiated within 12 hours of hospital arrival. EEG findings revealed background asymmetry and moderate background slowing in all three patients, including lesional delta

waves at the ischemic focus. Epileptiform activity in the form of spikes and sharp waves was noted in all cases, localized to the ischemic hemisphere: right temporal in P001, left parietal in P002, and left fronto-parietal in P003. No electrographic seizures or nonconvulsive status epilepticus were detected during the monitoring period in any patient.

3.3 Quantitative EEG Indices

QEEG analysis revealed elevated slow-wave activity and interhemispheric asymmetry in all patients (Table 1).

Table 1. Quantitative EEG Indices, Prognostic Scores and Clinical Assessment Scores

Index/ Indices	P001	P002	P003	Median (Range)	Literature Thresholds
DAR (Delta/Alpha Ratio)	4.66	4.57	5.37	4.66	6.6+/-2.8 (poor outcome) [8,14,16]
DTABR	2.79	5.65	4.43	4.43	4.3+/-1.8 (poor outcome) [8]
BSI (Brain Symmetry Index)	0.09	0.15	0.29	0.15	>0.1 (abnormal) [10]
Prognostic Scores					
SeLECT-EEG Score	13%	77%	29%	29%	Higher = worse prognosis [15]
Clinical Assessment Scores					
Admission NIHSS	5	18	5	5	Higher = worse prognosis [7]
Discharge mRS	1	4	1	1	Higher = worse prognosis [7]

3.3.1 Delta/Alpha Ratio (DAR)

All three patients, as seen in Table 1, demonstrated markedly elevated DAR values ranging from 4.57 to 5.37 (median: 4.66). These values falls within the limits of the reported prognostic thresholds from the literature. In a study by Bentes et al. (n = 151), mRS was evaluated 12 months after stroke, and EEG was obtained less than 72 hours following the onset of ischemic stroke symptoms. Affected hemisphere median DAR (2.5 vs. 7.7) and unaffected hemisphere DAR (2.2 vs. 4.2) were lower in patients with mRS<3 than in those with mRS ≥3 [6]. More recent studies have reported frontal DAR values of approximately 3.3 as predictive of cerebral edema and poor outcomes after mechanical thrombectomy, with AUC of 0.80 [14].

Notably, P003 had the highest DAR (5.37) despite having the best ASPECTS score (9) and a relatively low admission NIHSS (5), suggesting that QEEG indices may capture functional impairment not fully reflected in structural imaging or clinical scales. Patient 002, who presented with status epilepticus and had the highest admission NIHSS (18), had a DAR of 4.57, which, while elevated, was not the highest in the series.

3.3.2 Delta/Theta-Alpha-Beta Ratio (DTABR)

DTABR values ranged from 2.79 to 5.65 (median: 4.43, Table 1), all confirming the reported optimal threshold for predicting poor outcomes. DTABR captures the overall shift from fast to slow frequencies and is considered a robust marker of cortical dysfunction. Meta-analytic data show that higher DTABR correlates with worse mRS (r = 0.32) and higher NIHSS (r = 0.49) [8].

P002 had the highest DTABR (5.65), consistent with the most severe clinical presentation (status epilepticus, NIHSS 18) and poorest functional outcome (discharge mRS 4). P001 had the lowest DTABR (2.79) and achieved excellent functional recovery (discharge mRS 1), suggesting a potential dose-response relationship between DTABR and outcome severity. Patient 003 had an intermediate DTABR (4.43) and also achieved good functional outcome (discharge mRS 1), though this patient had the highest DAR.

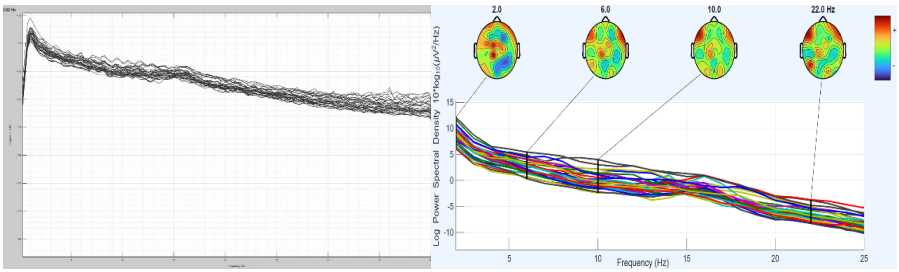
3.3.3 Brain Symmetry Index (BSI)

BSI values ranged from 0.09 to 0.29 (median: 0.15), as presented in Table 1, all exceeding the abnormal threshold of 0.1 [10]. These values indicate significant interhemispheric asymmetry in spectral power, reflecting the unilateral nature of the ischemic lesions and associated cortical dysfunction. P003 had the highest BSI (0.29), suggesting the greatest interhemispheric imbalance despite having the highest ASPECTS score, possibly related to the MCA-PCA junctional territory involvement affecting multiple cortical regions.

Literature reports on BSI correlation with functional outcomes have been inconsistent [8]. While some studies show inverse correlation with motor recovery and association with NIHSS [10] and pdBSI significantly correlated with mRS at 6 months [11], others found no significant association with functional outcomes in pooled analyses [8]. In our series, BSI did not clearly correlate with discharge mRS, as one patiens with good outcomes (P003) had the highest BSI value of 0.29, respectively, while the patient with poor outcome (P002) had a BSI (0.15) with a moderate value compared to the others included in the study.

3.4 Power Spectral Density and Spectrotopographic Analysis

Comprehensive PSD analysis with spectrotopographic mapping was performed for all three patients, revealing characteristic spectral alterations consistent with acute ischemic stroke. The analysis confirmed increased delta power and decreased alpha power in ischemic regions, with regional specificity corresponding to the vascular territories involved.

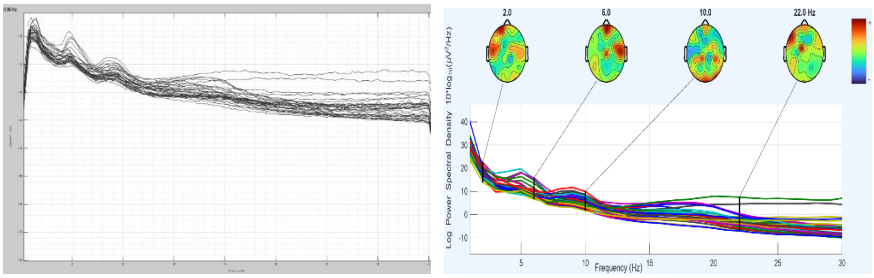


A. **B.**

Figure 1. Patient 001 - Right MCA Cortico-Subcortical Fronto-Insular Infarction

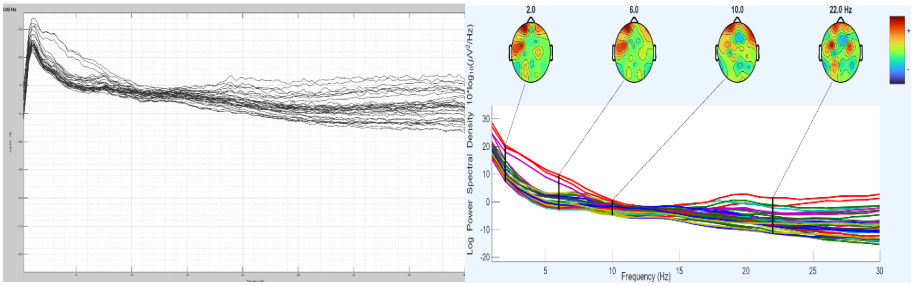
(A) Power Spectral Density: Multi-channel PSD plot showing elevated low-frequency power (0-10 Hz) with characteristic 1/f decay. Increased delta power evident across multiple channels with smooth decline toward higher frequencies.

(B) Spectrotopographic Mapping: Topographic distribution at 2.0 Hz (delta), 6.0 Hz (theta), 10.0 Hz (alpha), and 22.0 Hz (beta) demonstrating right fronto-temporal delta augmentation, right hemispheric theta elevation, left posterior alpha preservation, and relatively symmetric beta distribution.



A. **B.**

Figure 2. Patient 002 - Left MCA Superficial Territory Infarction with Status Epilepticus
(A) Power Spectral Density: Multi-channel PSD plot showing markedly elevated low-frequency power with prominent delta and theta peaks. Secondary peak around 6-8 Hz consistent with post-ictal slowing. Most severe spectral abnormalities in the series.
(B) Spectrotopographic Mapping: Topographic distribution showing profound left hemispheric delta augmentation (2.0 Hz), extensive theta elevation with multiple "hot spots" (6.0 Hz), near-complete left hemispheric alpha attenuation (10.0 Hz), and asymmetric beta reduction.



A. **B.**

Figure 3. Patient 003 - Left MCA-PCA Junctional Territory Infarction
(A) Power Spectral Density: Multi-channel PSD plot showing prominent low-frequency elevation with moderate delta and theta augmentation. Some residual alpha peaks visible in certain channels. Intermediate spectral severity between P001 and P002.
(B) Spectrotopographic Mapping: Topographic distribution showing left fronto-temporo-parietal-occipital delta augmentation (2.0 Hz), posterior-predominant theta elevation (6.0 Hz), significant left posterior alpha attenuation with right occipital preservation (10.0 Hz), and relatively preserved beta activity (22.0 Hz).

Power Spectral Density: The Power Spectral Density (PSD) plot serves as a fundamental quantitative tool in EEG analysis, decomposing the complex brain signal into its constituent frequencies to evaluate the distribution of electrical power. The horizontal axis (X-axis) represents the Frequency, measured in Hertz (Hz), typically ranging from 1 Hz to 25–40 Hz to capture the primary clinical bands (Delta, Theta, Alpha, and Beta). The vertical axis (Y-axis) represents the Log Power Spectral Density, expressed in 10 x log10 (muV^2/Hz). This logarithmic scale allows for the simultaneous visualization of high-amplitude slow waves and lower-amplitude rapid oscillations, providing a comprehensive profile of the patient's cortical excitability.

The PSD plots reveal a spectrum of cortical dysfunction severity across the three patients. All demonstrate characteristic 1/f spectral decay with elevated low-frequency power (0-10 Hz), but with varying magnitudes. As seen in Figure 1A, P001's PSD shows moderate delta elevation with smooth decline which indicates diffuse slow-wave augmentation without prominent alpha peaks, reflecting disruption of normal thalamocortical rhythms. Figure 2A represents the PSD of P002, which exhibits the most severe abnormalities with markedly elevated delta-theta power and a distinct secondary peak around 6-8 Hz suggesting post-ictal slowing, and near-complete absence of normal alpha peaks—a "shifted" spectral profile indicating dif-

fuse severe dysfunction. That may reflect post-ictal changes following status epilepticus. P003's PSD, as seen in Figure 3A, demonstrates intermediate severity with prominent delta-theta activity and some residual alpha peaks reflecting partial preservation in unaffected regions. The regional variability is reflecting the junctional territory involvement. The spectral profiles correlate with clinical severity also presented in Table 1: P001 (DTABR 2.79, mRS 1), P002 (DTABR 5.65, mRS 4), and P003 (DTABR 4.43, mRS 1).

Spectrotopographic Mapping: The topographic maps illustrate the spatial distribution of power across the scalp, where the color gradient represents the intensity of the signal. Specifically, the dark blue areas indicate regions of minimum power or suppressed activity, while the green and yellow zones represent intermediate power levels. The red and dark orange regions signify maximum power density, highlighting the dominant focal activity at the specific frequencies of 2.0, 6.0, 10.0, and 22.0 Hz. The topographic maps at representative frequencies from Figure 1B reveal the spatial distribution of spectral abnormalities, while Figure 2B reveals striking hemispheric asymmetry. Moreover, as seen in Figure 3B, the topographic maps reveal complex spatial patterns reflecting the MCA-PCA junctional territory.

✓ **Delta (2.0 Hz):** All three patients demonstrate marked hemispheric asymmetry with increased delta power (red-orange regions) ipsilateral to the infarct. P001 (Figure 1B) shows right frontotemporal predominance corresponding to the frontoinsular infarct. P002 represented in Figure 2B exhibits the most intense delta augmentation (bright red) throughout the left frontal, temporal, and parietal regions—the highest in the series—reflecting large superficial MCA territory involvement and status epilepticus. Interestingly, Figure 3B representative for P003 displays multiple elevated delta areas extending from frontal to occipital regions in the left hemisphere, reflecting MCA-PCA junctional watershed involvement. Contralateral hemispheres show relatively preserved distribution (blue-green) in all patients, with P003 showing mild increases in delta power suggesting possible remote effects or contralateral diaschisis.

✓ **Theta (6.0 Hz):** The maps demonstrate continued ipsilateral elevation in all patients with distinct topographic patterns. P001 (Figure 1B) demonstrates right frontotemporal gradient from high power (orange-red) to lower contralateral power (blue-green), indicating ischemic-healthy tissue transition zones. P002 shows in Figure 2B marked left central-parietal elevation with multiple "hot spots" possibly representing maximal cortical damage or epileptogenic foci, reflecting both ischemic changes and post-ictal slowing. P003, as seen in Figure 3B, exhibits distinctive posterior predominance in temporal and parietal-occipital regions with multiple left posterior quadrant "hot spots" consistent with PCA territory involvement in the junctional infarct.

✓ **Alpha (10.0 Hz)**: All patients show alpha attenuation with preserved contralateral activity ipsilateral to the ischemic area, creating interhemispheric asymmetry. P001 (Figure 1B) demonstrates marked right hemisphere reduction (blue) particularly in the infarct territory, reflecting loss of normal posterior dominant rhythm generation, with preserved left posterior activity (green-yellow) reflecting intact thalamocortical function contralateral to lesion. P002's map presented in Figure 2B exhibits the most severe attenuation with near-complete absence throughout the left hemisphere (blue), creating stark contrast with preserved right posterior alpha (orange-yellow)—the most pronounced disruption in the series reflecting severe thalamocortical network damage in the ischemic hemisphere. P003 shows in Figure 3B significant left posterior attenuation (blue) particularly striking given that PCA territory typically generates robust alpha activity, with preserved right occipital activity (green-yellow). The alpha disruption pattern localizes to ischemic territories and correlates inversely with hypoperfused volume ($\varrho \approx -0.66$) [17,18].

✓ **Beta (22.0 Hz):** All patients demonstrate relatively symmetric beta distribution with mild ipsilateral reduction, suggesting fast rhythms are less affected than slow rhythms in acute ischemia—consistent with preferential disruption of lower-frequency oscillations. P001 (Figure 1B) shows mild right frontal reduction, P002 (Figure 2B) exhibits asymmetric reduction in the left hemisphere though less pronounced than alpha attenuation, and P003 displays in Figure 3B relatively symmetric distribution with mild left hemisphere reduction and focal areas corresponding to infarct territory.

Clinical Correlation: The spectral patterns demonstrate clear severity gradients correlating with functional outcomes. P001's localized right fronto-insular dysfunction with preserved contralateral alpha and low DTABR (2.79) correlates with favorable outcome (mRS 1), validating anatomical localization. P002's severe extensive left MCA territory dysfunction with profound delta augmentation, near-complete alpha attenuation, and prominent theta activity correlates with the highest DTABR (5.65) and poorest outcome (mRS 4), reflecting both large ischemic lesion and additional metabolic stress from status epilepticus with post-ictal slowing persisting hours to days [19]. P003 presents an interesting discordance: despite having the highest DAR (5.37) and BSI (0.29) with moderate-to-severe junctional dysfunction and prominent posterior alpha attenuation, this patient achieved excellent recovery (mRS 1), suggesting that junctional territory location and favorable extent (ASPECTS 9) may outweigh spectral indices alone, with intermediate DTABR (4.43) potentially better reflecting true functional impairment.

Literature Comparison: These findings align with Ajčević et al. (2021), demonstrating that delta power correlates directly and alpha power inversely with hypoperfused volume ($\varrho \approx 0.65$ and -0.66 respectively), with regional spectral abnormalities localizing to ischemic territories [17,18]. P001's preserved contralateral alpha is consistent with unilateral MCA infarction without global dysfunction. P002's spectral abnormalities exceed typical uncomplicated stroke findings, consistent with the "double hit" of ischemia plus status epilepticus, where seizures increase metabolic demand in compromised tissue, potentially expanding dysfunction zones [20], aligning with reports of severe cortical dysfunction in large MCA infarctions with poor outcomes [6,9]. P003's junctional territory involvement creates complex spectral patterns with multiple cortical regions affected, and the discordance between high DAR/BSI and good outcome highlights the complexity of outcome prediction and the importance of considering lesion location and extent beyond QEEG indices alone.

3.4.1 Summary of Spectral Findings Across All Patients

Common Features:

✓ **Delta Power Augmentation:** All three patients demonstrated increased delta power (1-4 Hz) in ischemic hemispheres, with regional specificity corresponding to vascular territories. The magnitude of delta augmentation correlated with infarct size and clinical severity (P002 > P003 > P001).

✓ **Alpha Power Attenuation:** All patients showed reduced alpha power (8-13 Hz) in ischemic regions compared to contralateral hemispheres. The alpha attenuation was most pronounced in P002 (near-complete absence) and least severe in P001 (moderate reduction).

✓ **Interhemispheric Asymmetry:** Spectrotopographic maps consistently revealed stark asymmetry between ischemic and non-ischemic hemispheres, with preserved spectral characteristics contralaterally. This asymmetry underlies the elevated BSI values (0.09-0.29) observed in all patients.

✓ **Frequency-Dependent Effects:** Slow-wave activity (delta, theta) showed the most pronounced augmentation, while fast rhythms (beta) were relatively preserved. This frequency-dependent pattern explains the elevated DAR and DTABR values.

✓ **Regional Specificity:** Topographic maps demonstrated that spectral abnormalities localized to the ischemic territories, validating the anatomical correlation between EEG changes and structural lesions.

Literature Validation: These spectral patterns are fully consistent with established literature findings:

✓ Delta power correlates with hypoperfused/infarct volumes ($\rho \approx 0.59$-0.65) [17,18];

✓ Alpha power inversely correlates with hypoperfusion ($\rho \approx -0.66$) [17,18];

✓ Regional spectral abnormalities are located to ischemic territories [20];

✓ Spectral shifts correlate with clinical severity (NIHSS) and outcomes (mRS) [6,9].

The comprehensive spectral analysis in our series validates the QEEG methodology and confirms that seizure-onset stroke patients exhibit characteristic patterns of slow-wave augmentation and fast-wave attenuation consistent with acute cortical dysfunction.

3.5 Correlation Between QEEG Indices, Spectral Patterns, and Outcomes

Examining the relationship between QEEG indices, spectral characteristics, and functional outcomes (see Table 1) in this small series reveals several observations:

1. **DTABR showed the strongest correlation with discharge mRS:** The patient with the highest DTABR (5.65) had the worst outcome (mRS 4), while the patient with the lowest DTABR (2.79) had an excellent outcome (mRS 1). This is consistent with meta-analytic findings showing DTABR correlation with mRS ($r = 0.32$) [8].
2. **Spectral severity correlated with outcomes:** Patient 002, with the most severe spectral abnormalities (near-complete alpha loss, extensive delta augmentation), had the worst outcome. P001, with moderate, localized spectral abnormalities, had the best outcome.
3. **DAR alone did not clearly correlate with outcome:** The patient with the highest DAR (5.37) achieved good functional recovery (mRS 1), while the patient with intermediate DAR (4.66) also had good outcome (mRS 1). This suggests that DAR alone may not be sufficient for outcome prediction in seizure-onset stroke.
4. **BSI showed no consistent pattern:** BSI values did not correlate with discharge mRS in this series, consistent with literature reports of inconsistent BSI-outcome associations [8].
5. **Posterior alpha preservation may be protective:** P003, despite having the highest DAR and BSI, showed some preservation of alpha activity in spectral plots, which may have contributed to the good outcome.
6. **SeLECT-EEG score showed promise:** The patient with the highest SeLECT-EEG score (77%) had the worst outcome (mRS 4), while the patient with the lowest score (13%) had the best outcome (mRS 1).
7. **Clinical severity (NIHSS) showed correlation with DTABR:** The patient with the highest admission NIHSS (18) had the worst outcome and only correlates with DTABR, emphasizing that QEEG indices should complement, not replace, clinical assessment.

4. Discussion

This case series presents detailed QEEG analysis of three acute ischemic stroke patients with epileptic seizures, demonstrating elevated slow-to-fast frequency ratios (DAR, DTABR) and interhemispheric asymmetry (BSI) consistent with

literature findings. Power spectral density analysis with spectrotopographic mapping confirmed increased delta power and decreased alpha power in ischemic regions with regional specificity corresponding to vascular territories, validating the neurophysiological substrate of acute brain ischemia.

4.1 QEEG Indices in Seizure-Onset Stroke: Comparison with Literature

Our patients' DAR values (range: 4.57-5.37, median: 4.66, Table 1) and DTABR values (range: 2.79-5.65, median: 4.43, Table 1) fall within literature-reported ranges. Studies report frontal DAR values of approximately 3.3 as predictive of cerebral edema and poor outcomes (AUC 0.80) [14], while Wang et al. (2021) reported a higher DAR threshold of 14.3 for predicting poor 90-day outcome (sensitivity 90.9%, specificity 90.0%) [7]. Meta-analytic data show DTABR correlates with worse mRS ($r = 0.32$) and higher NIHSS ($r = 0.49$) [8]. In our series, DTABR showed the strongest outcome association, with the highest DTABR (5.65) corresponding to the worst outcome (mRS 4) and the lowest DTABR (2.79) to excellent recovery (mRS 1) and also correlated with admission NIHSS (see Table 1).

Several factors may explain our intermediate DAR/DTABR positioning: (1) all patients had cortical infarctions generating more pronounced EEG abnormalities than subcortical strokes [21]; (2) acute seizures and epileptiform activity may transiently increase slow-wave activity beyond structural lesion expectations, with postictal slowing persisting hours to days [19], particularly evident in P002's prominent theta activity following status epilepticus; (3) EEG timing within 12 hours of hospital arrival (typically 24-36 hours post-onset) during peak hyperacute abnormalities [17,18]; and (4) measurement methodology variations in electrode montages, frequency definitions, and artifact rejection [8].

Despite elevated values, two of three patients achieved excellent outcomes (mRS 1), suggesting DAR elevation in seizure-onset stroke may not carry the same prognostic implications as in unselected populations, warranting further investigation, but DTABR showed clinical correlations.

The superior prognostic performance of DTABR compared to DAR or BSI may be explained by its ability to simultaneously capture both pathological slow-wave burden (delta+theta) and residual fast-wave activity (alpha+beta), thereby providing a more comprehensive assessment of the balance between ischemic dysfunction and preserved cortical integrity. A systematic review and meta-analysis demonstrated that DTABR showed stronger correlations with functional outcomes than DAR (mRS: $r=0.32$ vs $r=0.26$; NIHSS: $r=0.49$ vs $r=0.42$), and when added to clinical prediction models, contralesional DTABR explained the greatest additional variance (adjusted $r^2=0.60$, $P<0.001$), suggesting that the composite ratio captures unique neurophysiological information related to neurovascular coupling and tissue viability that single-band ratios may miss [8]. This bidirectional quantification of both injury severity and functional preservation likely accounts for DTABR's enhanced discriminative capacity in seizure-onset stroke outcomes.

BSI values (range: 0.09-0.29, median: 0.15) all exceeded the abnormal threshold of 0.1 [10], indicating significant interhemispheric asymmetry, but showed no clear outcome relationship. This aligns with literature reports of inconsistent BSI-outcome associations [8]. Paradoxically, P003 had the highest BSI (0.29) with excellent outcome (mRS 1), while P002 had middle-ranged BSI (0.15) with worst outcome (mRS 4). This may relate to lesion characteristics: P003's MCA-PCA junctional infarct created greater interhemispheric asymmetry across multiple regions, while P002's large superficial MCA infarct caused severe bilateral background slowing, reducing relative asymmetry despite worse absolute dysfunction. These observations highlight BSI interpretation complexity and the need to consider lesion-specific factors.

4.2 Spectral Analysis: Regional Patterns and Clinical Correlations

Comprehensive spectral analysis with topographic mapping provided critical insights complementing global QEEG indices. Spectrotopographic maps consistently demonstrated spectral abnormalities localizing to ischemic territories with preserved contralateral rhythms across all frequency bands, validating QEEG methodology and confirming that spectral changes reflect true cortical dysfunction rather than artifacts. These findings align with Ajčević et al. (2021) and Finnigan et al. (2004), who demonstrated strong correlations between regional spectral abnormalities and infarct/hypoperfused volumes [17,18,23].

The analysis revealed frequency-dependent effects with preferential slow-wave augmentation (delta, theta) and relative beta preservation, reflecting selective vulnerability of lower-frequency oscillations to ischemia. Delta and alpha rhythms generated by thalamocortical circuits are particularly sensitive to metabolic compromise, whereas beta rhythms involve more distributed cortical networks with greater resilience [24]. Preserved beta activity in all patients, even in regions with severe delta augmentation and alpha attenuation, suggests residual cortical processing capacity with implications for recovery potential and rehabilitation.

Alpha power attenuation observed in all patients, particularly in P003's posterior regions with PCA involvement, highlights thalamocortical network disruption. Alpha rhythm (8-13 Hz) is generated by reciprocal thalamic-cortical interactions and represents the dominant rhythm in awake, relaxed individuals [25]. In acute stroke, alpha power decreases in ischemic regions, correlating inversely with hypoperfused volume ($\rho \approx -0.66$) and clinical severity [17,18]. Alpha attenuation degree correlated with outcome severity: P002 showed near-complete alpha loss with worst outcome, while P001 and P003 demonstrated partial preservation with good outcomes. Spectrotopographic maps at 10.0 Hz clearly visualized this asymmetry, providing intuitive evidence useful for clinical communication and prognostic counseling.

P002's prominent theta peak (6-8 Hz) following status epilepticus likely reflects post-ictal slowing from neuronal exhaustion and metabolic depletion [22]. This complicates QEEG interpretation in seizure-onset stroke, as elevated DAR and DTABR values reflect both ischemic injury and superimposed seizure effects. Disentangling these contributions requires serial EEG recordings tracking spectral evolution as post-ictal effects resolve.

4.3 Seizures and Prognostic Implications

All three patients experienced acute symptomatic seizures within 5 days of stroke onset. Post-stroke seizure mechanisms involve acute excitotoxicity, ionic imbalances, blood-brain barrier disruption, inflammation, and altered GABAergic inhibition [26,27]. Seizures may elevate QEEG indices through: (1) ictal and post-ictal slowing persisting hours to days [22]; (2) metabolic stress in compromised tissue expanding dysfunction zones [20] through the "double hit" explaining spectral abnormality magnitude, particularly in P002; (3) interictal epileptiform discharges contributing to background slowing [28]; and (4) acute cortical network reorganization altering excitation-inhibition balance[27].

The SeLECT-EEG prognostic score showed promise, with the highest score (77%) corresponding to worst outcome (mRS 4) and lowest (13%) to best outcome (mRS 1) [15]. However, P003's excellent recovery despite highest DAR (5.37) and BSI (0.29) demonstrates that elevated QEEG indices do not invariably predict poor outcomes, underscoring the importance of comprehensive assessment including clinical severity, imaging findings (ASPECTS, lesion location), comorbidities, and treatment response.

Potential clinical applications include: (1) risk stratification identifying high-risk patients for aggressive intervention (DTABR most reliable); (2) treatment response monitoring through serial QEEG measurements; (3) prognostic counseling with objective data and visual spectral displays; (4) clinical trial enrichment; and (5) seizure risk assessment guiding ASM decisions.

4.4 Limitations and Future Directions

Primary limitations include small sample size (N=3), single-center design, lack of control group, and single QEEG recording at variable times relative to stroke/seizure onset, preventing statistical inference, causal attribution, and assessment of temporal evolution or long-term prognosis (follow-up only to discharge). Heterogeneous lesion locations, seizure timing variability, and absence of quantitative imaging-EEG correlation limit interpretability.

Future research should prioritize larger prospective cohorts (N=50-100) with standardized QEEG protocols and serial measurements (admission, 24h, 72h, 7d) to capture temporal evolution and resolve post-ictal effects, incorporating long-term outcome assessment (90-day, 6-month mRS) and case-control designs comparing seizure-onset versus non-seizure stroke patients. Multi-center collaborations are essential for standardization and consensus guidelines. Key focuses should include multimodal integration of QEEG with neuroimaging, biomarkers, and genetics using machine learning; analysis beyond spectral ratios to functional connectivity patterns (coherence, graph theory); and intervention trials evaluating QEEG-guided therapies (optimized ASM, neuromodulation) and prophylactic strategies with cost-effectiveness analysis.

4.5. Proposed Clinical Algorithm: Integrating QEEG into Acute Stroke Workflow

STEP 1: Initial Assessment (ED/Stroke Unit Admission)

Clinical Triage:

- Altered consciousness, fluctuating alertness, or unexplained deterioration?
- Focal motor phenomena or clinical suspicion of seizure?

→ YES to any: Proceed to continuous EEG (cEEG) monitoring urgently

→ NO: Proceed to standard EEG within 72 hours for prognostication

STEP 2: EEG Acquisition & QEEG Analysis

Timing: Within 72 hours (ideally) or up to 7 days post-stroke

QEEG Indices to Calculate:

- DTABR: (delta+theta)/(alpha+beta) ratio
- DAR: delta/alpha ratio
- BSI: Brain Symmetry Index

STEP 3: Interpretation & Risk Stratification

QEEG Finding	Clinical Action
Epileptiform activity or periodic discharges	→ Escalate to prolonged cEEG → Neurology/epilepsy consult → Consider prophylactic ASM → Counsel on high PSE risk
Marked background slowing (↑DTABR/DAR) + asymmetry (↑BSI)	→ Integrate into poor outcome prognostication → Early rehabilitation planning → Discuss elevated seizure & disability risk with family
Normal QEEG + low clinical suspicion	→ Routine stroke care → No additional EEG monitoring required

STEP 4: Follow-Up & Discharge Planning

High-Risk Patients (abnormal QEEG):

- Schedule outpatient neurology follow-up within 1–2 weeks
- Consider ASM at discharge if epileptiform activity present
- Provide seizure precautions and education

Standard-Risk Patients:

- Routine stroke follow-up at 3 months
- Educate on late seizure warning signs

Quick Reference: QEEG in Acute Stroke (4-Step Protocol)

1. TRIGGER	Seizure suspicion → Order EEG within 72h
2. MEASURE	Calculate DTABR, DAR, BSI
3. STRATIFY	• Epileptiform activity → cEEG + ASM + Consult • High DTABR/BSI → Poor prognosis counseling • Normal → Routine care
4. FOLLOW-UP	High-risk patients → Early neurology appointment + ASM consideration

5. Conclusions

This case series demonstrates that acute ischemic stroke patients presenting with epileptic seizures exhibit significantly elevated QEEG slow-to-fast frequency ratios (DAR, DTABR) and interhemispheric asymmetry (BSI) compared to established norms. Comprehensive power spectral density analysis confirmed increased delta power and decreased alpha power corresponding to the ischemic vascular territories. Specifically, DTABR showed the strongest association with functional outcome in our limited series, with the highest value linked to the worst outcome (mRS 4) and the lowest to excellent recovery (mRS 1).

These findings suggest that QEEG analysis, particularly DTABR and spectrotopographic mapping, provides valuable, objective markers of cortical dysfunction that can complement clinical and imaging assessments for risk stratification. However, two out of three patients still achieved excellent functional outcomes (mRS 1) despite highly abnormal QEEG indices, emphasizing the complexity of outcome prediction. The prognostic implications of elevated QEEG values are likely modulated by factors such as lesion location, seizure timing, and post-ictal effects. Larger, prospective studies are necessary to validate these observations, establish seizure-specific prognostic thresholds, and define the optimal role of QEEG in clinical decision-making.

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, A.M.N. and I.D.C.; methodology, R.R.; software, L.E.V; validation, A.M.N.; formal analysis, A.M.N.; investigation, R.R., M.I.S.; resources, L.E.V.,M.I.S.; data curation, I.D.C.; writing—original draft preparation A.M.N.; writing—review and editing, R.R.; visualization, L.E.V.; supervision, I.D.C.; project administration, A.M.N.; funding acquisition, A.M.N. 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.
Funding: Please add: This research received no external
Conflicts of Interest: The authors declare no conflict of interest.

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Supplementary Table 1. Patient Demographics, Clinical Characteristics, Treatments, and Outcomes

Complete clinical data for three patients with seizure-onset acute ischemic stroke, including baseline characteristics, EEG findings, quantitative EEG indices, antiseizure medication management, and clinical outcomes.

Demographics & Baseline

Variable	P001	P002	P003
Patient ID	P001	P002	P003
Age	58	65	64
Sex	F	M	M
Baseline mRS	2	5	2
Charlson Comorbidity Index	2	4	2
Hypertension	Yes	Yes	Yes
Diabetes	Yes	No	No
Atrial Fibrillation	Yes	Yes	Yes
Prior Epilepsy	No	No	No
Prior Seizures	No	No	No
Prior Stroke	No	No	No

Stroke Characteristics

Variable	P001	P002	P003
Stroke Type	Ischemic	Ischemic	Ischemic
Vascular Territory	RIGHT MCA	LEFT MCA	LEFT MCA-PCA
Cortical Involvement	Yes	Yes	Yes
ASPECTS Score	7	5	9
Territories included	Cortico-subcortical fronto insular	Superficial ACM	Junctional ACM-ACP
Hemorrhagic Transformation	N/A	N/A	N/A
Symptom Onset Time	2023-06-17 18:00:00	2023-04-31 23:30:00	2023-05-13 20:00:00
Hospital Arrival Time	2023-06-18 20:50:00	2023-05-01 05:00:00	2023-05-16 21:00:00
Admission NIHSS	5	18	5
APACHE II Score	14	15	11
GCS Score	15	15	14

Acute Treatments

Variable	P001	P002	P003
IV Thrombolysis	No	No	No
Thrombolysis Time	0	0	0
Endovascular Therapy	No	No	No
EVT Time	0	0	0
TICI Score	0	0	0
Neurosurgical Procedure	No	No	No

EEG Monitoring

Variable	P001	P002	P003
EEG Modality	cEEG	cEEG	cEEG
Montage	10-20	10-20	10-20
Sampling Rate Hz	512 Hz	512 Hz	512 Hz
Time to First EEG Hours	12	12	12
Total Monitoring Duration Hours	3	3	3
Continuous Monitoring	Yes	Yes	Yes

EEG Findings

Variable	P001	P002	P003
Background Asymmetry	Yes	Yes	Yes
Background Slowing	Moderate	Moderate	Moderate
Epileptiform Activity	No	No	No
EA Type	Spikes/Sharp waves	Spikes/Sharp waves	Spikes/Sharp waves
EA Location	Right Frontal	Left parietal	Left fronto-parietal
Seizures Clinical	Yes	Yes	Yes
Seizures Electrographic	No	No	No
NCSE	No	No	No
Status Epilepticus	No	Yes	No

Quantitative EEG

Variable	P001	P002	P003
qEEG Performed	Yes	Yes	Yes
DAR Ratio	4.66	4.57	5.37
DTABR Ratio	2.79	5.65	4.43
BSI Index	0.09	0.15	0.29

Seizure Details

Variable	P001	P002	P003
Acute symptomatic seizure	Yes	Yes	Yes
Type of acute symptomatic seizure	Left facial focal motor seizure lasting 10-15 seconds with impaired awareness	Right hemicorporeal focal motor onset with secondary bilateralization lasting approximately 30 minutes - Status epilepticus	Generalized tonic-clonic motor onset
Onset time of acute symptomatic seizure	24 h 5 days	<24h	3 days

Antiseizure Medication

Variable	P001	P002	P003
ASM Prescribed	Yes	Yes	Yes
ASM Indication	Therapeutic	Therapeutic	Therapeutic
First ASM Drug Name	Levetiracetam	Levetiracetam	Levetiracetam
First ASM Dose	1000mg	1000mg	1500mg
ASM Start Time	2023-06-18 21:30:00	2023-05-01 05:00:00	2023-05-16 21:10:00
ASM Changes Made	No	No	No
ASM Change Reason	0	0	0
Second ASM Drug	Yes	No	No
Second ASM Drug Name	Sodium Valproate	0	0
Second ASM Dose	1000 mg	0	0
ASM Duration Days	7	13	4

Hospital Course

Variable	P001	P002	P003
Hospital Length Stay Days	10	13	4
ICU Length Stay Days	0	0	0
Mechanical Ventilation	No	No	No
Ventilation Duration Days	0	0	0
Neurological Deterioration	Yes	Yes	Yes
Deterioration Cause	Seizures	Seizures	Seizures
EEG Triggered ASM Change	No	No	No
Clinical deterioration triggers ASM Change/ adding second ASM	Yes	No	No
Cause of clinical deterioration for ASM change	Seizure	0	0

Outcomes

Variable	P001	P002	P003
Discharge NIHSS	3	15	3
Discharge mRS	1	4	1
In Hospital Mortality	No	No	No
Discharge Destination	Home	Home	Home
Post Stroke Epilepsy	Yes	No	No
Time to First Unprovoked Seizure Days	1	0	3
ASM at Discharge	Yes	Yes	Yes
Cognitive Assessment	Normal	Impaired	Normal
Prognostic Score Used	SeLECT-EEG	SeLECT-EEG	SeLECT-EEG
Prognostic Score Value	0.13	0.77	0.29