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Case Report

Quantitative EEG Findings in Post-Stroke Epilepsy Patients

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ABSTRACT

Post-stroke epilepsy presents a significant clinical challenge, often accompanied by cognitive impairments. The qEEG analysis in this case reveals notable abnormalities, including increased delta waves and decreased high-frequency activity, localized to specific brain regions. These findings not only strengthen diagnostic assessments but also provide valuable insights into the underlying pathophysiology of post-stroke epilepsy. Moreover, they serve as a basis for selecting personalized therapeutic interventions, such as neurofeedback therapy. By tailoring treatment to the captured brainwave frequencies during recording, non-invasive therapeutic options can be explored as viable alternatives for patients. Integrating qEEG findings into clinical practice enhances our understanding of post-stroke epilepsy's complex nature and guides optimal patient care and management strategies.

Keywords: quantitative EEG (qEEG); post-stroke epilepsy, cognitive impairments, diagnostic assessments, neurofeedback therapy

INTRODUCTION

Stroke stands as a significantly common trigger for seizures and epilepsy among adults. The occurrence of post-stroke epilepsy is noted in approximately 6% of the 3-6 million individuals affected by stroke. Post-stroke seizure episodes elevate metabolic stress and cellular demise, consequently enlarging infarct dimensions, escalating mortality rates, and diminishing functional outcomes in patients. Those who undergo seizures during the acute phase of stroke face a 60% likelihood of experiencing unprovoked seizures in subsequent conditions. Repeated seizures have the potential to diminish cognitive capabilities and the overall quality of life for patients.[1]

On the other hand, the development of quantitative EEG (qEEG) represents a significant advancement in diagnostic tools for cognitive function disorders, offering specific and quantitative assessments of brain dynamics at the cellular level. Unlike conventional EEG, qEEG has the capability to evaluate interactions

within groups of cells, individual cellular roles, cognitive circuits, and information flow with a level of precision that was previously unattainable.[2]

Exploring quantitative EEG (qEEG) findings in post-stroke epilepsy patients is crucial for comprehensively understanding the complex neurological ramifications of stroke and its interplay with epileptic seizures. Post-stroke epilepsy presents a multifaceted challenge in clinical practice, exerting profound impacts on the short-term and long-term prognoses of stroke survivors. By delving into the quantitative analysis of EEG data, researchers endeavor to unveil intricate neural signatures and patterns that characterize post-stroke epilepsy, thereby offering invaluable insights into diagnosis, prognosis, and potential therapeutic avenues. By elucidating the underlying mechanisms through quantitative EEG investigations, clinicians and researchers can better grasp the pathophysiological underpinnings of post-stroke epilepsy, paving the way for more targeted and efficacious treatment strategies. Thus, the exploration of quantitative EEG findings in post-stroke epilepsy patients represents a crucial endeavor aimed at unraveling the complexities of this neurological disorder and enhancing patient outcomes through evidence-based interventions and personalized care approaches. [1,2]

CASE REPORT

A 47-year-old female, has experienced recurrent seizures since March 2021, initially presenting with pre-ictal symptoms followed by ictal manifestations including lip smacking, eye blinking, hand stiffness, and upward eye deviation. Medical evaluation at Hospital revealed a suspicious brain tumor on MRI, but subsequent biopsy results were inconclusive for malignancy. In July 2021, she reported cognitive difficulties prompting a second opinion at another hospital, where a neurooncology board meeting in May 2022 attributed her symptoms to a vascular lesion obstructing the left middle cerebral artery. Despite ongoing seizures, she transitioned to levetiracetam due to aspirin and simvastatin use. Seizure frequency

remains 1-2 times per month as of January 2024. She denies hypertension, heart disease, or diabetes history, with no family history of similar conditions. Currently, the patient's regular medication regimen includes aspirin 1x80mg and levetiracetam 2x1000mg. Physical examination reveals cognitive impairment without other neurological deficits, as indicated by a Glasgow Coma Scale score of Eye 4, Motor 6, Verbal 5.

The first MRI findings disclosed multiple aging infarcts, ranging from acute to subacute stages, raising suspicion of a possible mass lesion. Consequently, a contrast-enhanced MRI brain was conducted at another hospital. Extensive pathological intensity (gliosis) manifested within the left middle cerebral artery territory, encompassing the left frontal lobe, insula, amygdala, and parahippocampal regions, along with compression of the left mesencephalon, indicative of subacute-chronic infarction. Notably, compared to prior imaging, the presence of edema was no longer evident. The MRI findings in second hospital showed in Figure 1.

Following a re-evaluation at second hospital, the biopsy results unveiled reactive and degenerative brain parenchyma. The patient also underwent a Digital Subtraction Angiography (DSA) procedure, with the results indicating normal findings. However, an EEG administered in September 2022 identified abnormal results, including focal slowing and epileptiform activity within the left temporal region. This observation covered periods of both wakefulness and non-rapid eye movement (NREM) sleep stage I. Analysis of the QEEG showed heightened Delta wave activity and reduced Beta wave activity in absolute power. Additionally, irregular connections were noted in amplitude asymmetry, coherence, and phase lag.

To delve deeper into the analysis, we decided to conduct a quantitative EEG examination on the patient (figure 3). This additional examination was deemed necessary to obtain more detailed and quantitative data regarding the electrical activity of the brain. By employing quantitative EEG, we aimed to gain insights into the specific patterns and

dynamics of brain function, as well as to evaluate any abnormalities or irregularities in neural activity that may not be readily apparent through conventional EEG methods. This comprehensive approach allowed us to explore the nuances of the patient's brain activity and provided valuable information that could aid in diagnosis, treatment planning, and monitoring of their condition. Through the quantitative analysis provided by EEG, we were able to assess various parameters such as absolute power, relative power, amplitude asymmetry, coherence, and phase lag, enabling a thorough evaluation of the patient's neurological status. Overall, the utilization of quantitative EEG added an extra layer of depth to our understanding of the patient's brain function and facilitated a more comprehensive assessment of their condition.

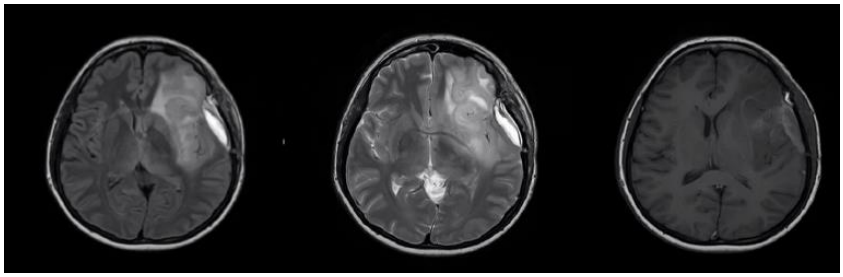

Figure 1. Non-Contrast Head MRI of Patient Post-Biopsy on T1, T2, and T2-Flair sequences.

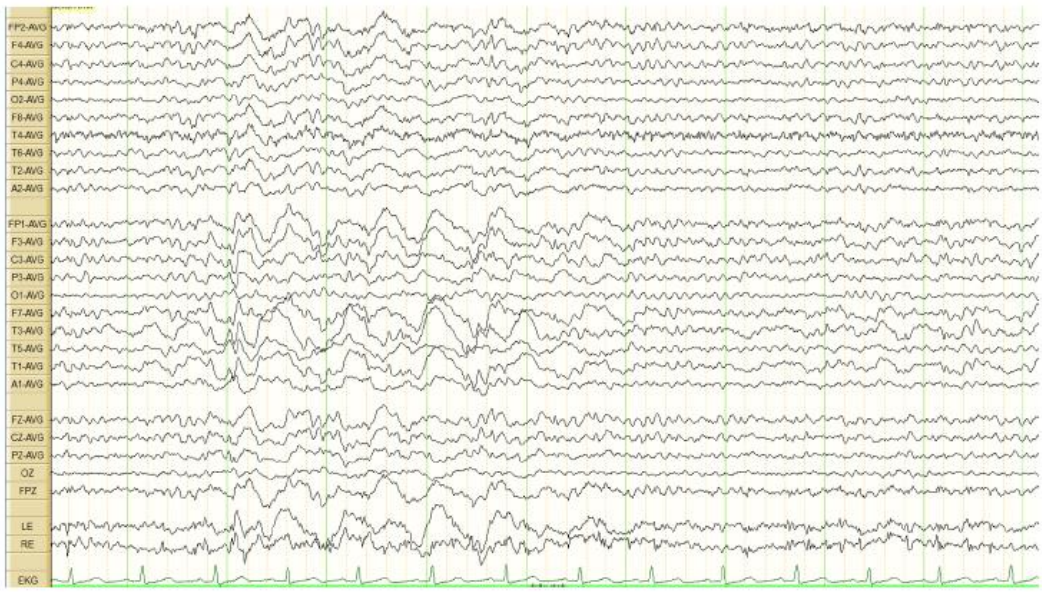

Figure 2. Patient’s EEG Findings.

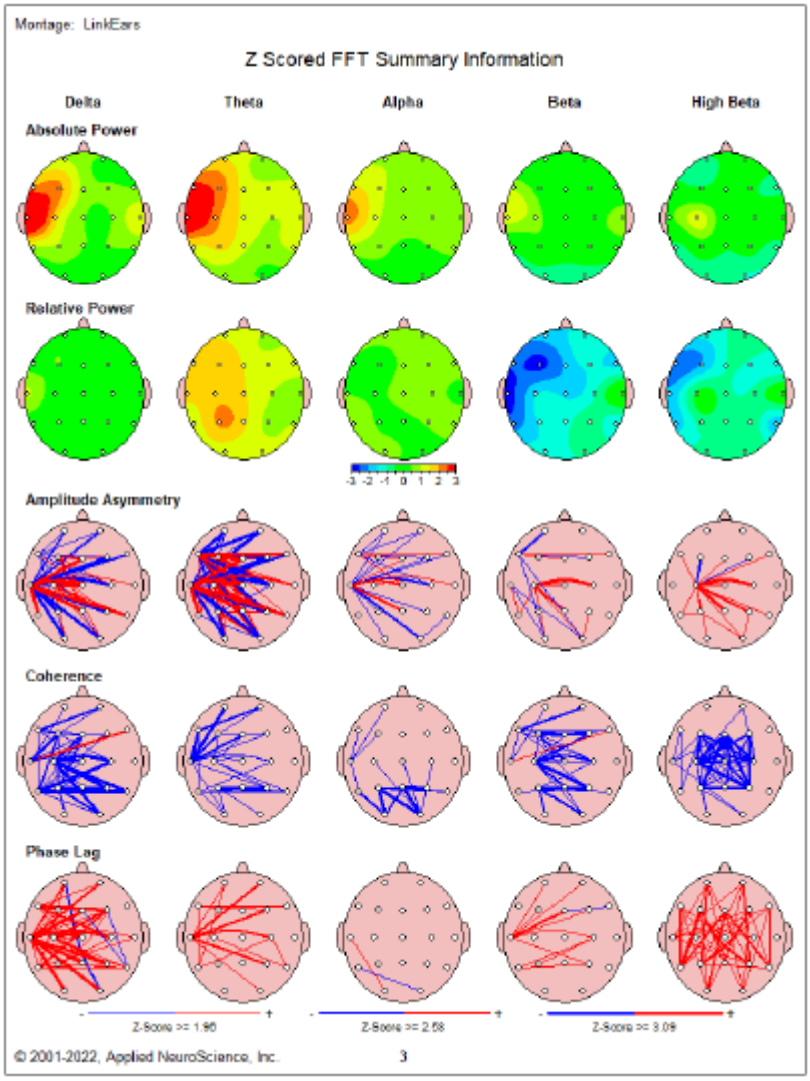

Figure 3. Patient's Quantitative EEG Findings

DISCUSSION

Post-stroke epilepsy presents a significant challenge for individuals who have experienced a stroke. According to a cohort study involving 100,000 subjects, the incidence rate of post-stroke epilepsy was found to be 7%. Recurrent seizures have been demonstrated to exacerbate prognosis and elevate mortality rates among stroke survivors. These seizures are classified into two categories: early and late, with a window of 7 days following the onset of stroke. Early seizures typically manifest within the initial days after stroke and are commonly known as acute symptomatic seizures, whereas late seizures occur after 7 days post-stroke and often reach their peak between 6

to 12 months later. Seizures following a stroke commonly demonstrate focal seizure features, although around one-third of cases involve generalized tonic-clonic seizures, while the remaining two-thirds typically show partial seizures. Early-onset seizures are more often characterized by focal onset, whereas late-onset seizures tend to be linked with generalized tonic-clonic seizures.[4,5]

Early seizures in post-stroke cases result from local cellular dysfunction, triggered by acute ischemia-induced blood-brain barrier disruption, leading to increased glutamate levels and subsequent neuronal dysfunction. Late seizures occur when the brain has predisposed conditions such as gliosis, chronic inflammation, and synaptic changes. Both types of seizures can exacerbate brain damage and contribute to post-stroke epilepsy development.[4,6]

Cognitive impairment represents a prevalent challenge within the population of individuals afflicted with post-stroke epilepsy. This dual condition, marked by both epilepsy and the aftermath of a stroke, often manifests in various cognitive deficits that significantly impact daily functioning and quality of life.[7]

The complexities of cognitive impairment in post-stroke epilepsy patients stem from the combined effects of stroke-related brain damage and the epileptic activity that ensues. The structural alterations resulting from the stroke, such as neuronal loss, gliosis, and disrupted neural networks, contribute to cognitive dysfunction. Additionally, the recurrent seizures characteristic of epilepsy further exacerbates cognitive deficits through mechanisms such as neuronal excitotoxicity and disruption of neurotransmitter balance.[7]

Furthermore, the cognitive impairment in post-stroke epilepsy patients encompasses a broad spectrum of deficits, including memory impairment, attention deficits, executive dysfunction, and language difficulties. These cognitive impairments can vary in severity and may worsen over time, posing significant challenges for patients in managing daily activities, social interactions, and vocational responsibilities.[7]

Quantitative Electroencephalography (qEEG) plays a crucial role in recognizing cognitive impairment in patients with post-stroke epilepsy. This advanced neuroimaging technique provides a quantitative assessment of the brain's electrical activity, offering valuable insights into the underlying neural mechanisms contributing to cognitive dysfunction. By analyzing qEEG data, clinicians can identify aberrant patterns of brain activity associated with cognitive deficits, such as abnormalities in oscillatory rhythms, connectivity measures, and functional networks. Moreover, qEEG enables the detection of subtle changes in brain function that may not be evident through traditional neuroimaging methods, allowing for early intervention and targeted treatment strategies.[8]

The normative QEEG database consists of metrics derived from EEG data collected from a large population, enabling comparison with individual metrics to identify electrophysiological markers associated with specific disorders. It includes data from active and resting states, allowing for the calculation of z-scores to determine deviations from the population mean. These metrics cover absolute power, relative power, power ratio, amplitude asymmetry, coherence, and phase, providing insights into brain functioning.[9]

Clinically, cognitive abnormalities are associated with observed changes in brain waves. Excessive focal delta waves may indicate localized injury or impairment, while global changes suggest general pathology or systemic issues. Theta wave excess often signifies brain dysregulation due to reduced activity. Asymmetrical alpha waves affect mood, while beta wave abnormalities manifest as restlessness, insomnia, migraines. Imbalances in Delta/Theta lead to cognitive disturbances, hyperactivity, while alpha imbalances suggest depression, irritability, and beta imbalances result in restlessness, migraines, insomnia, or panic attacks.[9]

In the patient's case, it's evident that a lesion exists in the left frontal area, corresponding to the QEEG waveform findings. There's a prevalence of delta waves in absolute power, while relative power shows a

dominance of low beta waves. This disparity in frequency levels is known to contribute to cognitive disturbances, particularly affecting executive functions due to the frontal area lesion. The patient finds it challenging to process information and stay focused on forming new memories, which corresponds to observations seen in epilepsy patients exhibiting heightened delta and theta waves alongside diminished high-frequency waves.

In terms of the depiction of asymmetrical amplitude, there is an apparent imbalance between the hemispheres, particularly notable with frequency differences primarily observed on the left side. The abundance of frequency disparities, especially prevalent on the left side as indicated by the blue and red lines, is often associated with focal seizures.

In the management of epilepsy patients, pharmacological interventions may prove insufficient, prompting consideration of neurofeedback training as an alternative approach. Alpha wave therapy could be utilized to elevate delta waves, while ongoing SMR therapy aims to reduce seizure occurrences in epilepsy cases.

Neurofeedback is a component of biofeedback, which teaches subjects to control their brain function by measuring brainwaves and providing feedback signals with audio or video. There are treatment protocols which can be provided to patients according to their clinical needs. The Alpha Protocol involves utilizing brain alpha waves for relaxation and mood enhancement. These waves are associated with creativity and are utilized in relaxation techniques, leading to sleep. Alpha therapy, typically ranging from 7 to 10 Hz, is used for pain relief, stress reduction, memory enhancement, and improving mental performance. It's also effective for treating conditions like anxiety and brain injuries.[2,10]

Delta waves, the slowest brain waves linked with stages 3 and 4 of sleep, signify heightened comfort, pain relief, and deep sleep. As a result, they are employed to mitigate headaches, traumatic brain injuries, learning difficulties, and to address rigid and acute muscle contractions through delta wave stimulation at 1-3

Hz. Additionally, they diminish anxiety levels and enhance the quality of sleep. The Sensorimotor Rhythm (SMR) is linked with alertness, fine motor activity with the intention of boosting SMR waves and diminishing theta and high beta waves. It can be delivered through auditory cues like basic sounds, leading to heightened focus, anxiety, and improved motor coordination and stability. [2,10]

CONCLUSION

QEEG findings in post-stroke epilepsy patients offer valuable insights into the neurological sequelae following a stroke event. By examining the quantitative EEG data, clinicians can identify specific abnormalities in brainwave patterns associated with epilepsy, providing crucial information for diagnosis and treatment planning. Cognitive deficits can develop in individuals experiencing post-stroke epilepsy, as evidenced by qEEG results showing elevated delta waves and reduced high-frequency activity localized to distinct brain areas. These observations strengthen diagnostic evaluations and offer a framework for choosing neurofeedback interventions. By customizing therapy according to the brainwave frequencies recorded, non-invasive treatment modalities can be explored. The integration of qEEG insights into clinical protocols not only advances comprehension of the underlying pathophysiology of post-stroke epilepsy but also directs tailored therapeutic approaches, thereby optimizing patient care and management strategies.

REFERENCE

1. Galovic M, Ferreira-Atuesta C, Abraira L, Döhler N, Sinka L, Brigo F, et al. Seizures and Epilepsy After Stroke: Epidemiology, Biomarkers and Management. Vol. 38, Drugs and Aging. Adis; 2021. p. 285–99.
2. van Tuijl JH, van Raak EPM, van Oostenbrugge RJ, Aldenkamp AP, Rouhl RPW. Cognition and quality of life in patients with poststroke epilepsy: A case–control study. Epilepsy and Behavior. 2020 Mar 1;104.
3. Sutcliffe L, Lumley H, Shaw L, et al. Surface electroencephalography (EEG) during the acute phase of stroke to assist with diagnosis and prediction of prognosis: a scoping review. BMC Emerg Med. 2022; 22(29)
4. Feyissa AM, Hasan TF, Meschia JF. Stroke-related epilepsy. Eur J Neurol. 2019;26:18.
5. Galovic M, Ferreira-Atuesta C, Abraira L, Döhler N, Sinka L, Brigo F, Bentes C, Zelano J, Koepp MJ. Seizures and Epilepsy After Stroke: Epidemiology, Biomarkers and Management. Drugs Aging. 2021 Apr;38(4):285-299
6. Agarwal A, Sharma J, Padma Srivastava MV, Bhatia R, Singh MB, Gupta A, Pandit AK, Singh R, Rajan R, Dwivedi S, Upadhyay A, Garg A, Vishnu VY. Early Post-Stroke Seizures in Acute Ischemic Stroke: A Prospective Cohort Study. Ann Indian Acad Neurol. 2021 Jul-Aug;24(4):580-585.
7. Novak A, Vizjak K, Rakusa M. Cognitive Impairment in People with Epilepsy. J Clin Med. 2022 Jan 5;11(1):267.
8. Doerrfuss JI, Kilic T, Ahmadi M, Holtkamp M, Weber JE. Quantitative and Qualitative EEG as a Prediction Tool for Outcome and Complications in Acute Stroke Patients. Clin EEG Neurosci. 2020 Mar;51(2):121-129.
9. Fingelkurts AA, Fingelkurts AA. Quantitative Electroencephalogram (qEEG) as a Natural and Non-Invasive Window into Living Brain and Mind in the Functional Continuum of Healthy and Pathological Conditions. Vol. 12, Applied Sciences (Switzerland). MDPI; 2022.
10. Renton T, Tibbles A, Topolovec-Vranic J. Neurofeedback as a form of cognitive rehabilitation therapy following stroke: A systematic review. PLoS One. 2017 May 16;12(5):e0177290.