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Silent seizures in glioma: The role of preoperative EEG in detecting electrographic seizures

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Abstract

Seizures are a common presentation in glioma patients, yet electrographic seizures — subclinical seizure activity detected only on electroencephalography (EEG) are often overlooked due to the lack of routine EEG screening. This case report describes a glioma patient who presented with worsening occipital headaches and cognitive decline but no overt seizures. Preoperative EEG revealed an electrographic seizure originating from the left temporal region, despite the absence of clinical manifestations. The patient underwent surgical resection, and histology confirmed a WHO grade 4 IDH-mutant astrocytoma. This case highlights the importance of proactive EEG screening, even in the absence of clinical seizures, as it facilitated early seizure detection and treatment. Further studies are needed to evaluate the impact of treating electrographic seizures on long-term outcomes in glioma.

Keywords: Electrographic seizures, EEG screening, glioma, IDH mutant astrocytoma, cognitive decline

CASE REPORT

A previously healthy 52-year-old man presented with a one-year history of progressive occipital headaches. One week prior to his hospitalization, the headaches worsened and were accompanied by giddiness and vomiting. He also experienced persistent memory impairment and word-finding difficulties. There were no stereotypic episodes suggestive of focal seizures. Neurological examination revealed normal motor, sensory and speech functions except for persistent word-finding difficulties. Brain magnetic resonance imaging (MRI) revealed a left medial temporal contrast-enhancing mass. (Figure 1) Despite absence of clinical seizures, given the high seizure prevalence associated with gliomas, an electroencephalogram (EEG) was performed to screen for subclinical seizure activity. The EEG revealed persistent left temporal delta slowing, occasional left anterior temporal sharp waves, and one electrographic seizure originating from the left temporal region (Figure 2).

He was started on levetiracetam (500mg twice daily) and underwent left pterional craniotomy and tumor excision the following day. Histopathological examination (HPE) confirmed a WHO grade 4 IDH mutant astrocytoma. However, a follow-up MRI one month post-surgery revealed residual tumor at the left posterior temporal region with perilesional edema. (Figure 1) A second completion surgery was performed via a left parieto-occipital craniotomy. The patient showed significant post-operative improvement in word-finding ability and was started on adjuvant concurrent chemoradiotherapy (CCRT) 2 weeks post-surgery.

DISCUSSION

Seizures are common in glioma patients, accounting for approximately 80% of cases.[1] However, nonconvulsive status epilepticus (NCSE) is under-reported, in only 2% of brain tumor patients, primarily due to a lack of routine EEG screening.[1,2] Electrographic seizures, which are seizures detected only by EEG without clinical manifestations, are even less frequently reported, despite their potential contribution to cognitive decline and neuronal damage.[3] Studies of other neurological conditions (e.g., traumatic brain injury and stroke) suggested that electrographic seizures may lead to progressive cognitive impairment and secondary brain injury.[3]

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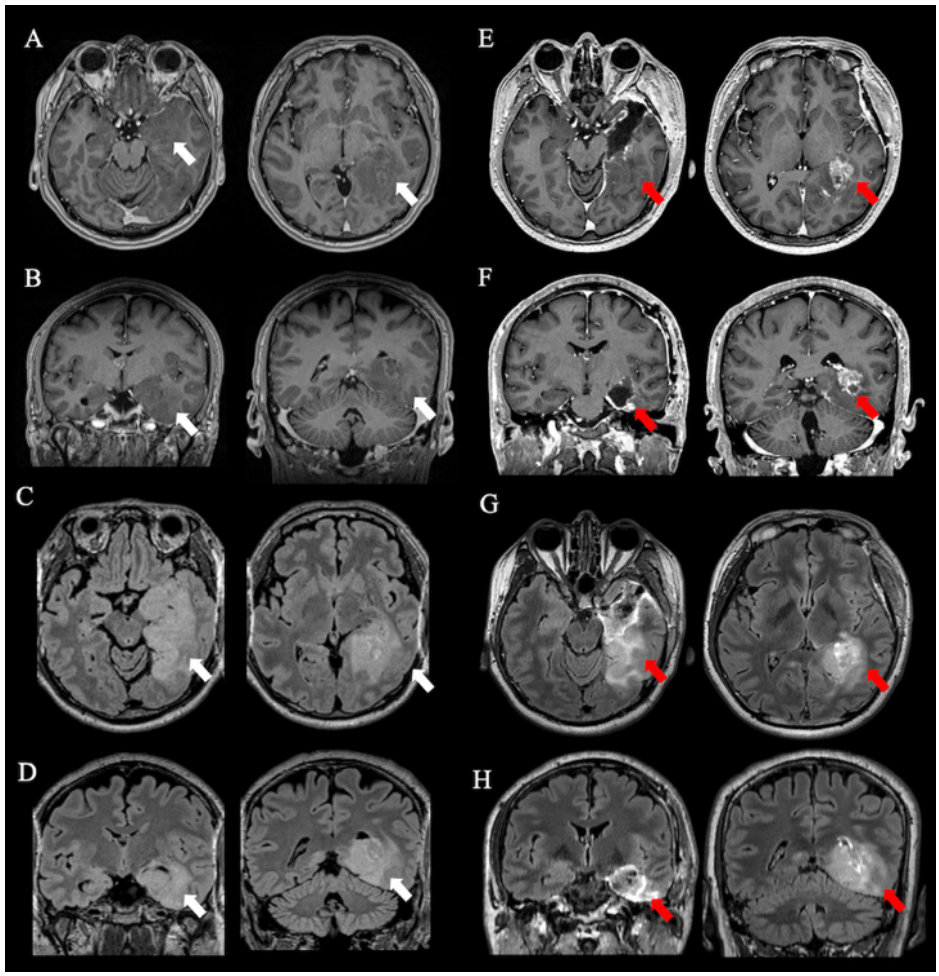

Figure 1. Pre-operative brain MRI (A-D) shows an ill-defined contrast-enhancing mass (white arrow) at the left medial and basal temporal lobe measuring approximately 2.0x1.4x1.6cm on contrasted T1-weighted sequence of axial (A) and coronal views (B). The mass showed hyperintense signal on fluid-attenuated inversion recovery (FLAIR) of axial (C) and coronal views (D). Repeated brain MRI one month post operation (E-H) showed contrast-enhancing residual tumour (red arrow) measuring 1.5x2.0x1.3cm at the left medial and basal temporal lobe on contrasted T1-weighted sequence at axial (E) and coronal views (F) with surrounding areas of FLAIR hyperintensities (G-H).

In current clinical practice, the diagnosis of seizure relies on clinical events rather than proactive screening for subclinical seizure activity. However, since gliomas frequently involve peritumoral epileptogenic zones, it is possible to postulate that electrographic seizures contribute to cognitive dysfunction in glioma patients.

This case highlights the importance of preoperative EEG screening in glioma patients, even in the absence of clinical seizures. As pre-operative EEG is not a standard practice in glioma diagnosis or management, this may underestimate the true burden of seizure activity. Preoperative EEG screening enables prompt antiseizure treatment, which may improve cognitive outcomes in glioma patients.[2]

Standard seizure management in brain tumor patients typically involves tumor resection and anti-seizure medications (ASMs). In brain tumor patients with NCSE, ASMs successfully resolved seizure activity in 92% of cases, leading to clinical improvement in 75% of patients.[4] However, it remains unknown whether treating subclinical seizures improves cognitive function, seizure burden, or alters long-term outcomes in patients with gliomas. Further research is needed to determine the significance of the proactive identification and treatment of electrographic seizures in gliomas.

In conclusion, glioma patients frequently

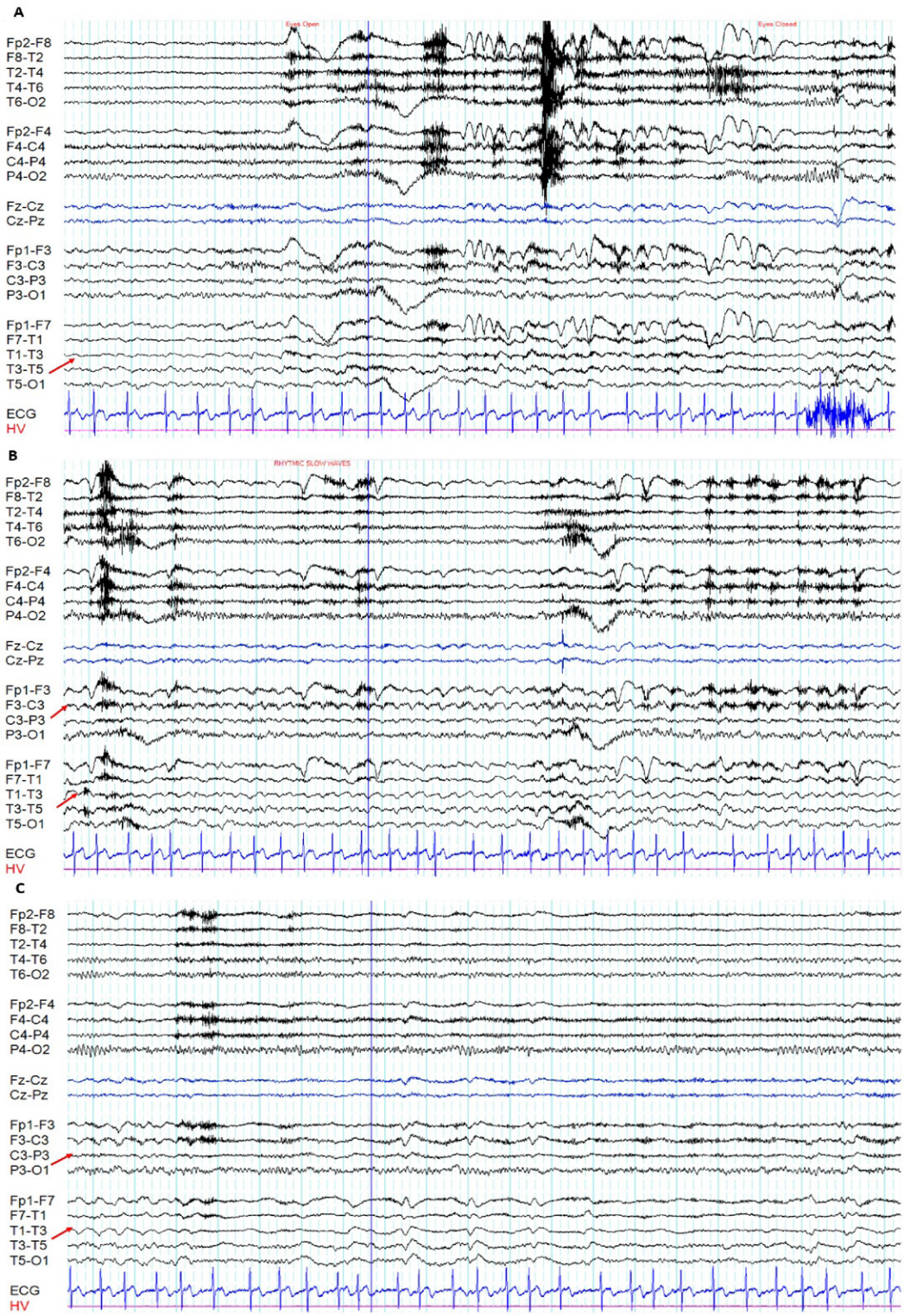

Figure 2. Subclinical seizure began as arrhythmic slowing at the left temporal (A) with evolution into 2 to 2.5Hz rhythmic activities at the left hemisphere (B-C).

experience seizures, yet electrographic seizures remain underdiagnosed due to the lack of routine EEG screening. This case highlighted the importance of early EEG screening, even in the absence of clinical seizures.

DISCLOSURE

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