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Case Report

Startle epilepsy triggered by a light bump: a rare case confirmed with ambulatory electroencephelography

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ABSTRACT

Reflex epilepsy is characterized by seizures triggered by specific sensory or cognitive stimuli, with startle-induced epilepsy being a rare subtype often misdiagnosed due to non-specific findings on routine electroencephalography (EEG). We report the case of a 16-year-old boy with a history of hypoxic-ischemic encephalopathy who experienced frequent episodes of sudden muscle stiffness, myoclonic jerks, tonic posturing of limbs, and occasional loss of consciousness, consistently triggered by sudden tactile or auditory stimuli such as a light bump. The seizures began at age 11 and fluctuated in frequency, occurring up to 8–9 times per day. Previous magnetic resonance imaging (MRI) revealed bilateral temporal gliosis and frontal encephalomalacia, but routine EEGs at ages 12 and 15 were normal, leading to diagnostic uncertainty and ineffective treatment with several anti-seizure medications. A 24-hour ambulatory EEG successfully captured an ictal event triggered by a slight bump, revealing high-amplitude sharp and wave discharges over the bilateral temporal regions with frontal extension. Interictal epileptiform discharges and epileptic K-complexes were also noted. With the confirmation of startle epilepsy, the patient was started on appropriate antiepileptic treatment and advised behavioral modifications, resulting in a reduction of seizure frequency to 4–5 episodes per week. This case underscores the clinical value of prolonged EEG monitoring in cases of suspected reflex epilepsy, particularly when routine evaluations are inconclusive. Ambulatory EEG plays a crucial role in diagnosing rare seizure types and enabling timely, targeted therapy that can improve patient outcomes.

Keywords: Startle epilepsy, Reflex epilepsy, Ambulatory EEG, Tactile stimuli, Seizure, Case report

INTRODUCTION

Reflex seizures are always provoked by particular sensory or cognitive stimuli.[1] Reflexive is the term used when consistently provoked by particular stimuli or activities, which may be motor, sensory, or cognitive.[2] Startle epilepsy, a variant, is usually provoked by strong auditory or tactile stimuli.[3] Seizures provoked by subtle tactile stimuli are less commonly described. Reflex epilepsies constitute a subcategory of epilepsy syndromes in which all the seizures of an individual are activated by well-established sensory stimuli and reactive seizures are activated by transient systemic disturbances like alcohol intake, sleep deprivation, or intercurrent illness.

Reflex seizures have been observed in both generalised and focal epilepsy disorders, and they can also occur in response to structural brain lesions.[4] These seizures can be precipitated by extrinsic (e.g., flashing lights, music) or intrinsic (e.g., mental activity, emotions) stimuli, and they can also be classified according to complexity—simple stimuli (e.g., photic stimulation) versus more complex precipitants requiring higher cortical functions complicated reflex epilepsies include praxis-induced seizures, which occur while executing tasks or activities

involving complicated spatial processing, written arithmetic, or board games, as well as language-related reflex seizures caused by reading, speaking, writing, or listening to music.[5]

In this article, we report a case of startle-induced reflex epilepsy, a type of epilepsy where seizures are evoked by unexpected sensory stimuli like touch or noise. This is an important case because it highlights the value of high-quality electroencephalography (EEG) diagnostics, particularly ambulatory EEG (aEEG), in diagnosing reflex epilepsy when conventional EEG is negative. Additionally, it emphasizes the importance of correct diagnosis in shaping proper treatment regimens for patients with reflex epilepsy.

CASE REPORT

A 16-year-old male presented with recurrent episodes characterized by sudden muscle stiffening, myoclonic jerks, bilateral tonic elevation of extremities, and falls with or without transient loss of consciousness. These episodes, occurring 8-9 times daily, were consistently precipitated by a light bump or other startle stimuli. The patient had no known allergies, and his family history was unremarkable. Psychological assessment revealed normal cognitive function.

Seizure onset occurred at age 11, with a duration of 4 years at presentation. Seizure frequency varied from 4-5 episodes weekly to 2-3 episodes daily. The patient had also been diagnosed with reflex epilepsy triggered by sudden sound/noise, indicating a multifactorial trigger mechanism.

His past medical history included hypoxic-ischemic encephalopathy (HIE), and brain imaging revealed mild gliosis in the bilateral temporal lobes. Magnetic resonance imaging (MRI) showed bilateral frontal encephalomalacia of unknown etiology. A cranial computed tomography (CT) scan at age 13 was unremarkable. Previous routine EEGs at ages 12 and 15 were normal.

The patient had been treated with multiple antiseizure medications (ASMs), including oxcarbazepine and levetiracetam, without sustained seizure control. Biochemical, immunological, serological, and hematological tests, including complete blood count (CBC) and free triiodothyronine (FT3), was within normal limits.

Given persistent clinical suspicion of reflex epilepsy, a 24-hour aEEG was performed. This captured an ictal event triggered by a light bump, revealing abnormal interictal epileptiform discharges (Figures 1-3). The ictal onset was localized to the bilateral temporal chains, demonstrating high-amplitude sharp and wave discharges, followed by frontal region involvement. Bilateral involvement was noted during sleep, with prominent fast activity in the parasagittal chain. Interictal epileptiform discharges with epileptic K-complexes were also observed figure 4. With proper diagnosis, seizure frequency was reduced to 4-5 per week.

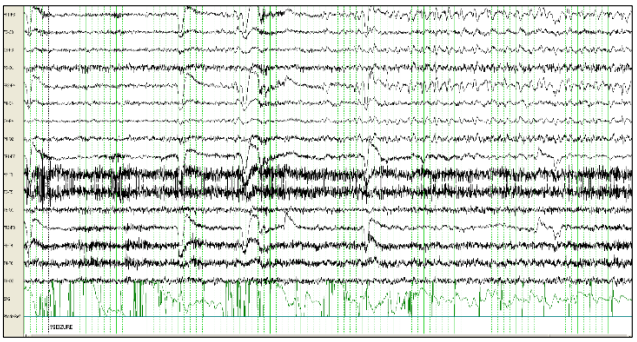

Figure 1: Evolution of rhythmic ictal discharges shortly after the patient experienced a light bump, with rhythmic parasagittal activity indicating a 1–4 Hz frequency, consistent with reflex seizure onset. The EEG was recorded with a high-frequency filter of 70 Hz, low-frequency filter of 1 Hz, notch filter off, sensitivity of 7 µV/mm, and a 10-second epoch.

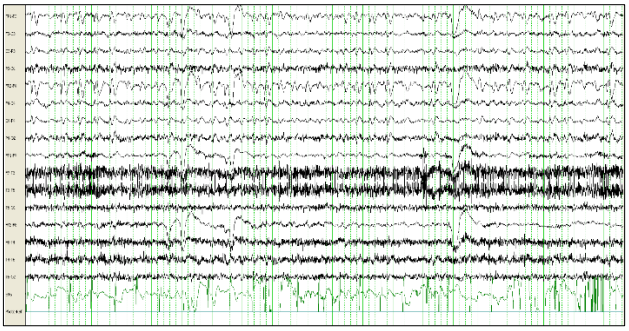

Figure 2: Evolution of the ictal event with rhythmic parasagittal activity in the 1–4 Hz range, indicating seizure progression. EEG settings: HFF 70 Hz, LFF 1 Hz, notch off, sensitivity 7 µV/mm, and 10-second epoch.

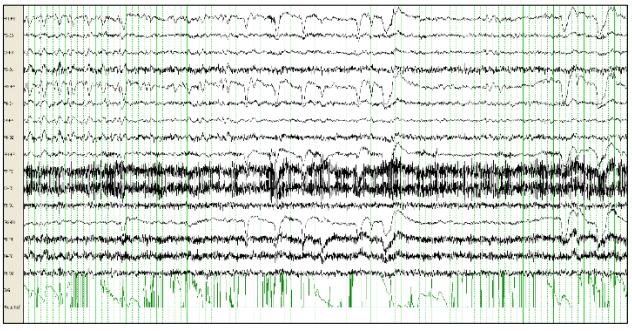

Figure 3: Late ictal phase with gradual attenuation of rhythmic activity, marking the resolution of the seizure, returning toward baseline. EEG recorded using HFF 70 Hz, LFF 1 Hz, notch off, sensitivity 7 µV/mm, and 10-second epoch.

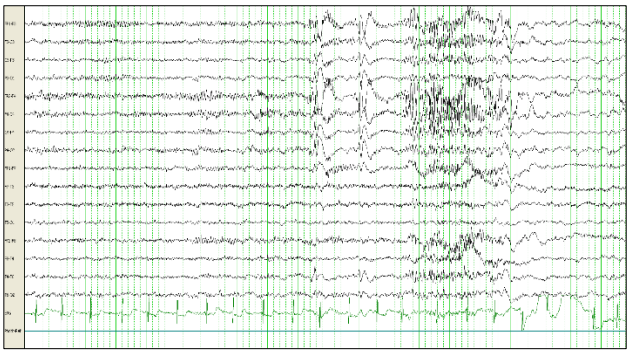

Figure 4: The EEG was recorded using a high-frequency filter (HFF) of 70 Hz, low-frequency filter (LFF) of 1 Hz, with the notch filter turned off, a sensitivity of 7 µV/mm, and a time base of 10 seconds per epoch. Shows the presence of interictal epileptiform discharges with epileptic K-complexes, further supporting the reflex nature of the seizures.

DISCUSSION

We present a case of reflex epilepsy in which seizures were triggered by a specific tactile stimulus, a light bump. The instance highlights the importance of high-quality diagnostic modalities for accurately diagnosing reflex seizures, which are frequently misdiagnosed due to poor-quality EEG recordings. Startle epilepsy, which typically manifests in infancy or adolescence, is frequently associated with structural brain abnormalities produced by prenatal or early-life trauma.[6] The stated causes are perinatal HIE, perinatal stroke, and meningitis, encephalitis, and porencephalic cysts. In other situations, focal dysplastic lesions have been described alongside normal MRI results.[7] In this example, an MRI revealed bilateral frontal encephalomalacia, and the patient had a history of HIE, confirming the presence of structural abnormalities in startle epilepsy.

Ambulatory EEG (aEEG) was critical in confirming the diagnosis, especially in cases where standard EEG had previously failed to detect epileptiform activity. Unlike normal EEG, which only provides a brief window of observation, aEEG enables for continuous monitoring in a patient's natural surroundings, increasing the possibility of recording seizures that would otherwise go undetected.[8] In this case, preceding conventional EEGs revealed no epileptiform discharges, resulting in an incorrect first diagnosis.

However, aEEG revealed significant information about the epileptogenic network, with ictal beginning in the contralateral temporal chains followed by frontal involvement. This pattern points to a complicated interaction between these regions in reflex epilepsy. Furthermore, bilateral participation during sleep and the presence of parasagittal rapid activity suggest extensive network disruption. The occurrence of interictal epileptiform discharges with epileptic K-complexes adds to the reflex nature of the seizures.

The precise pathophysiology of startle epilepsy remains unknown, but it is assumed to be caused by hyperexcitability of sensory networks, notably the supplementary motor area (SMA) and brainstem structures.[9] Earlier research has linked startle-induced seizures to hypersynchronous cortical-subcortical discharges, particularly in the premotor and parietal cortices. The patient's EEG results demonstrate rapid secondary generalisation following a minor tactile stimulation, lending support to the concept that certain people have increased cortical excitability in response to specific stimuli.[10] Though tactile and auditory stimuli are frequently reported in reflex epilepsy, there have been infrequent cases of seizures triggered by cognitive stimulation, such as thinking about a specific object or memory. Another patient with a left temporal focus on EEG had seizures when brushing his teeth but also on thinking of a toothbrush.[11]

Treatment of startle epilepsy is difficult due to the recurrent and, in many cases, refractory nature of the seizures. The correct diagnosis using an EEG significantly reduced the seizures in this patient. The patient experienced approximately 8-9 seizures the day before being exposed to an EEG. After adequate electroclinical correlation and therapy adjustments, seizure frequency decreased to 4-5 per week.

This research emphasises the importance of modern diagnostic equipment in fully optimising the treatment approach, as well as the dangers of making an inaccurate diagnosis with underutilised EEG techniques.

CONCLUSION

This case report describes a unique presentation of startle epilepsy caused by a mild bump, emphasising the need of detecting reflex epilepsy with atypical triggers. Ambulatory EEG played a critical role in detecting seizure occurrences that conventional EEG missed, allowing for an accurate diagnosis and targeted care. Our findings lend credence to the role of structural brain pathology in reflex epilepsy and emphasise the need for long-term observation in cases with abnormal normal standard EEG readings. Additional research into the pathophysiologic under-pinnings of startle epilepsy has the potential to lead to better, more personalised treatment options, including non-medication approaches.

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