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Case Report

Hot Water Epilepsy: A Case Report with EEG Findings and Clinical Insights

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Abstract

Hot water epilepsy (HWE) is a rare form of reflex epilepsy triggered by exposure to hot water, typically during baths, and can be difficult to diagnose due to normal electroencephalogram (EEG) results. This case outlines a 30-year-old woman who experienced recurrent seizures over five years while bathing in hot water. Despite her history of generalized epilepsy, her initial EEG and MRI results were unremarkable. A subsequent sleep-deprived video EEG detected ictal discharges originating from the frontal lobes, confirming epilepsy. The patient was advised to continue her anti-seizure medication and to avoid hot baths. The discussion emphasizes the challenges in identifying the anatomical substrate of HWE, as most cases typically involve the temporal or parietal regions. This case contributes to a deeper understanding of HWE by identifying frontal lobe discharges as a potential correlate and highlights the vital role of EEG in guiding treatment adjustments.

Keywords: *Hot Water, Epilepsy, EEG, Reflex Epilepsy*

Introduction

Hot water epilepsy (HWE) is a form of reflex epilepsy that occurs when bathing in hot water (40–50 °C). [1,2] It is the second most common form of reflex epilepsy and can occur at any time in an individual's life. There is a lack of knowledge about the anatomical substrate in HWE, and often the electroencephalogram (EEG) is normal. This article describes the clinical presentation of hot water bath-induced epilepsy in a young woman, including the recording of electrographic seizure in the EEG and correlating the anatomical substrate of HWE.

Case Presentation

A 30-year-old female patient has been experiencing seizure episodes for the past six years, which were initiated by a traumatic experience. The first seizure occurred during the early hours, specifically around 2 a.m., following the distressing viewing of a video depicting her brother's house engulfed in flames. This initial episode was characterized by loud vocalization, deep snoring, frothing at the mouth, and involuntary movements of all four limbs, lasting approximately three minutes. She does not have any habituation and does not have any medical illness.

The patient was diagnosed with reflex (fire-photosensitive) generalized epilepsy and was subsequently prescribed brivaracetam, commencing with a dosage of 50 mg twice daily. However, two months after the initiation of treatment, she experienced a second seizure. This alarming episode transpired while she was bathing; she abruptly cried out, snored loudly, became unconscious, and collapsed while pouring hot water over her head. This incident corresponds with a diagnosis of hot water epilepsy (HWE). Following this seizure, the patient required nearly 30 minutes to regain her stability. Her brivaracetam dose was hiked up to 75 mg twice daily.

Baseline laboratory tests including blood sugar, electrolytes, thyroid functions, renal and liver function tests were normal. Further diagnostic evaluations, including brain MRI and EEG, conducted on two separate occasions, yielded normal results. Notably, there is no significant family history of epilepsy, prior childhood febrile seizures, or any identifiable traumatic incidents that could account for her condition. The most recent seizure occurred in November 2021 during another bathing session involving hot water, increasing her total count of hot water epilepsy episodes to four. To mitigate the risk of further seizures, she has proactively avoided hot baths.

As her marriage approached, her family sought advice in year 2022, regarding the continuation of her anti-seizure medication. In response, we conducted an additional sleep-deprived video EEG, which indicated ictal discharges originating from the frontal lobes, lasting 22 seconds without accompanying clinical manifestations [Figures 1, 2]. Given these findings, we decided to increase her anti-seizure medication dosage to brivaracetam 75 mg morning and 100 mg evening. A follow-up EEG conducted three weeks later revealed normal results. The patient was advised to maintain her medication regimen and implement lifestyle modifications to avoid exposure to hot water baths. In instances where she must bathe with hot water, we recommended administering clonazepam 0.5 mg half an hour before bathing to reduce the likelihood of experiencing another seizure episode. She is under follow up since then till date

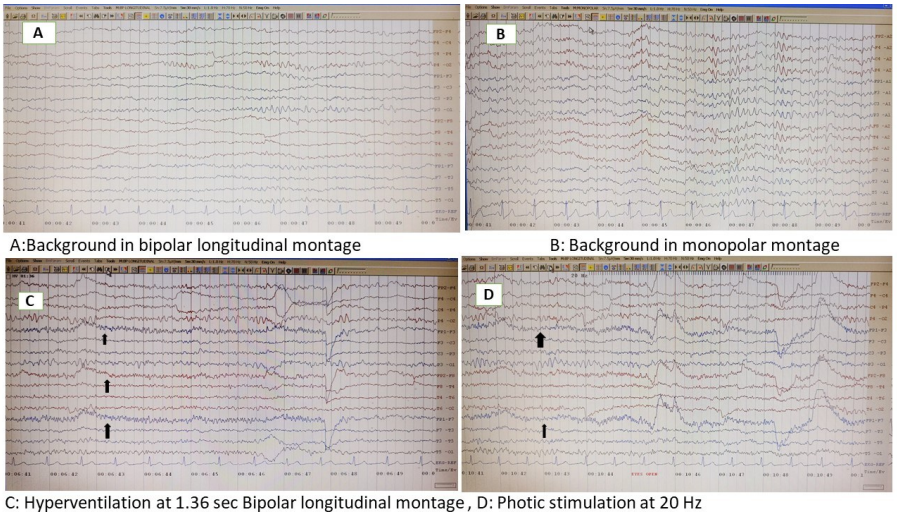

Figure 1. *(A) showing baseline normal background with alpha activity of 8hz as shown in bipolar longitudinal montage, (B) monopolar montage, (C) showing origin of fast activity low voltage from bilateral frontal region after hyperventilation and high frequency photic stimulation (D).*

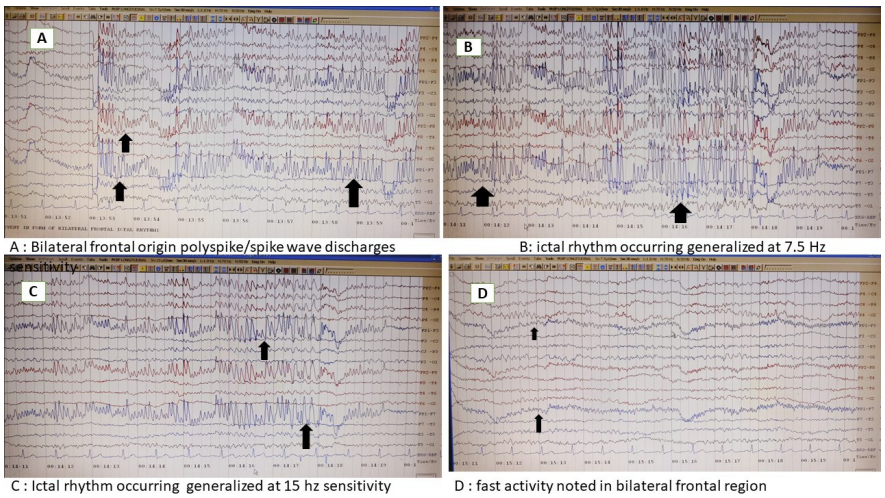

Figure 2. *(A) showing sudden onset high voltage polyspikes and spike wave discharges originating from bilateral frontal region as shown in bipolar longitudinal montages, with subsequent spread generalized activity (B) in all leads at a sensitivity of 7.5 microvolts. Figure (C) at sensitivity of 15 microvolts showing clear cut demarcation of polyspikes and spike wave discharges occurring generalized and (D) showing disappearance of ictal activity that lasted 22 seconds during the record followed by low voltage fast activity in bilateral frontal region.*

Discussion

Allen first described HWE in 1945 in New Zealand. [1] HWE is more prevalent in the South Indian population, as most of the cases in the world are reported from here. [3, 4, 5, 6] Prof. KS Mani, the father of Indian epileptology, first described HWE in South India in the year 1970. [3] Knowledge is scarce regarding the anatomical substrate of HWE. Patel M et al, [7] using multimodality including MRI, EEG, and SPECT, found the temporal as well as parietal focus of the origin of HWE. They found that either the medial or lateral temporal lobes were involved in HWE. Interictal EEG was normal among 5 out of the 6 cases they studied to demonstrate anatomical correlation. MRI brain was normal in all six cases. The MRI brain did not show structural abnormalities in the majority of case reports of HWE. Hence, there is difficulty in knowing the anatomical substrate of epilepsy. Only ictal and interictal SPECT could localize the lesion. There is no report of an isolated frontal lobe being involved in HWE. In a case series study of 21 cases with HWE reported by Bebek N et al, [8]the majority of cases had temporal lobe involvement, and only one case had frontal involvement. 40% of cases showed interictal EEG abnormality, while in 60% of cases, interictal EEG was normal. Overall pooled analysis from case series studies showed that only 15-20% of cases of HWE showed diffuse EEG abnormalities, while localized spike discharges were reported in a few cases. [9]

The exact cause of HWE remains uncertain. There are reports of seizures occurring even when hot water is not poured over the head, similar to the first event in our case. At a high temperature of 45 degrees Celsius, warmth receptors are most stimulated, leading to an abnormal thermoregulatory circuit that triggers an epileptic fit. [8, 9, 10] Clinicians should be aware of this rare form of reflex epilepsy, which accounts for only 6% of epilepsy. [10]

Reflex epilepsies (REs) are conditions marked by repeated seizures that are mainly brought on by particular types of motor, sensory, or cognitive stimulation. HWE and bathing-related epilepsy (BRE) rank among the most prevalent REs and are regarded as belonging to the same spectrum.[8] Recent findings indicate that these are separate conditions with unique genetics, triggers (such as hot versus pouring water), clinical manifestations, and associated comorbidities. An autosomal dominant inheritance pattern with reduced penetrance has led to the identification of two loci related to HWE located on chromosomes 10q21.3-q22.3 [11] and 4q24-q28, [12] while children affected by this condition exhibit otherwise normal development. In contrast, BRE has been linked to mutations in the X-linked gene SYN1, which encodes one of the three Synapsins (SYN1–SYN3), a group of phosphoproteins that play a role in synaptic development, functionality, and plasticity. [13, 14] Mutations in SYN1 are linked to various neurodevelopmental conditions, such as cognitive deficits and autism spectrum disorders (ASD). [15]

Differentiating between BRE and HWE can be quite challenging. Although there are some commonalities, there are notable distinctions between the two: BRE is primarily triggered by the act of bathing or showering, particularly when the individual is submerged in water or subjected to changes in water temperature while bathing. [16] Seizures associated with BRE can occur during the process of entering or exiting the bath, or even during showering. These seizures are not necessarily linked solely to hot water but may result from the overall bathing experience. The exact mechanism remains unclear, but being immersed in water, along with other environmental factors (like stress, relaxation, reflecting lights, voice of water, and temperature variations), may contribute to seizure onset.[17] BRE EEG abnormality has been reported in temporal lobes .In contrast, HWE is triggered specifically by exposure to hot water. This can happen during bathing, showering, or other interactions with hot water, including immersion in hot tubs or saunas. Seizures linked to hot water epilepsy occur solely when a person encounters hot water. It is believed that the sudden shift in body temperature and/or the physical sensations associated with hot water may provoke a seizure in predisposed individuals.[8,18] The seizures are thought to stem from the rapid temperature change and the physiological stress resulting from being in hot water.

In a study by Bakgetir F and colleagues,[19] performed a long-term follow-up on 50 patients diagnosed with HWE for up to 15 years. A good prognosis was characterized by patients whose seizures were managed with or without preventive treatments and who did not need antiepileptic medication. Conversely, a poor prognosis was described as patients whose seizures persisted despite preventive treatments and necessitated antiepileptic therapy. The researchers discovered that hot water epilepsy, contrary to prior understanding, is not a benign condition that resolves on its own. About three-quarters of the cases showed a favourable prognosis, while the remainder exhibited chronic epilepsy. The low effectiveness of antiepileptic treatments implies that the underlying mechanisms of HWE might differ from those of other forms of epilepsy. The median age of patients were 15 years . A comparable study was carried out by Hanci F and colleagues [20] focusing on the prognosis of 11 patients who experienced HWE during childhood, with ages ranging from 12 months to 13 years, and observed over a brief period of 18 months.

It was noted that the types of seizures included generalized motor seizures, absence seizures, and atonic seizures. Children diagnosed with childhood HWE exhibited normal neuromuscular development and had unremarkable neurological evaluations. These children need prophylactic measures or seizure management using a single antiepileptic medication, indicating that this condition is a self-limiting form of reflex epilepsy. The patient described here presents with adult-onset HWE. Despite being administered antiseizure medication, she continues to exhibit electrographic seizures, which suggests the presence of chronic epilepsy rather than a self-limiting course typically observed in pediatric cases.

HWE may involve complex mechanisms beyond warmth receptors and thermoregulatory circuits. While these factors initiate seizures in response to temperature changes, other neurophysiological pathways likely contribute to this phenomenon. Research suggests HWE could be linked to altered brain excitability in areas like the temporal lobe or brainstem, which are involved in sensory processing and autonomic regulation. Abnormal ion channel function or neurotransmitter regulation may also lead to increased sensitivity to temperature changes [21]

This case is significant as it enhances our understanding of how environmental factors, like hot water exposure, can trigger seizures in some individuals and highlights the need for further research into the mechanisms involved. Insights from neuroimaging and electrophysiological studies are essential for exploring specific brain regions and pathways. The broader implications include improved diagnosis and management of atypical epilepsy forms, particularly when triggers are not obvious. Understanding these mechanisms could lead to targeted therapies and better patient outcomes.

One limitation of the study is the dependence on EEG for localization, which might not offer the specificity required to identify subtle structural or functional abnormalities. In contrast to more advanced imaging methods such as SPECT or fMRI, EEG delivers less comprehensive information, which could result in an over interpretation of the involvement of the frontal lobe. Furthermore, the anatomical basis of HWE is still unclear, which adds to the complexity of accurately identifying the underlying mechanisms.

Conclusion

The case description presented herein illustrates a novel phenomenon, demonstrating an electrographic seizure without an accompanying clinical event in individuals with HWE. It highlights the significance of ASM in the management of HWE. This case emphasizes the crucial role of EEG in guiding the appropriate tapering of ASM in patients diagnosed with HWE, even in the absence of overt clinical seizures. Moreover, it adds to the existing literature surrounding HWE within the framework of generalized epilepsy, pinpointing the frontal lobe as a relevant anatomical correlate associated with HWE.

Patient Consent Declaration

The authors confirm that all necessary consent forms have been obtained, with patients granting permission for their images and clinical details to be published in the journal.

Conflict of Interest

The authors declare no conflict of interest.

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