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Case Report
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Bathing-Related Reflex Epilepsy in a Young Adult: First Reported Case from Palestine

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Abstract

Bathing-related reflex epilepsy (BRE) is an uncommon type of reflex epilepsy characterized by seizures that are reliably induced by contact with water during bathing or showering. In contrast to the more prevalent hot-water epilepsy (HWE), which is temperature-dependent, typically focal, and often benign, bathing-related epilepsy (BRE) can manifest with water of any temperature, display generalized features, and demonstrate resistance to pharmacological treatment. A 22-year-old male is reported with a five-year history of recurrent seizures predominantly occurring during bathing. Episodes were characterized by brief generalized tonic movements, eye rolling, frothy oral secretions, and transient loss of consciousness, succeeded by a prolonged postictal state lasting up to 30 minutes. Seizures occurred four to five times per month, primarily triggered by running water, and continued despite treatment with Lamotrigine, Carbamazepine, and Levetiracetam. Continuous 24-hour EEG monitoring recorded a habitual seizure triggered by water contact, revealing generalized high-amplitude rhythmic spike-and-wave discharges at 9 Hz, thereby confirming the diagnosis of BRE. This case underscores the diagnostic significance of ictal EEG and the necessity of differentiating between BRE and HWE. The identification of non-temperature-dependent triggers, the application of lifestyle modifications, and the evaluation of underlying genetic factors, especially SYN1 mutations, are critical for effective management and enhancement of long-term outcomes in patients with reflex epilepsies.

Keywords: Bathing-Related Reflex Epilepsy; Hot-Water Epilepsy; Reflex Seizures; EEG; SYN1 Gene; Pharmacoresistance; Case Report

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Introduction

Reflex epilepsies constitute a unique classification of seizure disorders, defined by epileptic episodes that are consistently and reliably provoked by particular sensory stimuli or cognitive processes. These triggers may include visual patterns, somatosensory stimuli, and more complex tasks such as reading or making choices. This shows how complicated the link is between outside factors and hyperexcitable neural networks in people who are more likely to be affected by them [8, 15]. Bathing-related reflex epilepsy (BRE) is a rare form of epilepsy that only happens when a person comes into contact with water while bathing or showering.

It is essential to differentiate BRE from the more commonly characterized hot-water epilepsy (HWE). HWE is generally a benign, focal epilepsy characterized by seizures induced by the application of hot water (40–50 °C) to the head, with the thermal stimulus regarded as a principal pathogenic factor [1–3]. On the other hand, BRE is not usually affected by the temperature of the water and can happen with water of any temperature. Moreover, BRE is commonly linked with generalized seizure semiology and exhibits a greater tendency for pharmacoresistance in contrast to the typically self-limiting or easily managed HWE [4–6]. This case report discusses the inaugural recorded occurrence of bathing-related reflex epilepsy from Palestine, highlighting the diagnostic difficulties and therapeutic impact of this uncommon condition.

This case report discusses the first observed case in Palestine, we believe it will add a lot to the literature and help physicians and healthcare providers to diagnose and deal with such cases, and consider further evaluation.

Case Presentation

A 22-year-old male, presented to a neurology clinic on 23rd of February 2025 with a five year history of recurrent episodes of seizures. The episodes occurred suddenly, while bathing. The patient described generalized body tonic movement lasting a few seconds followed by loss of consciousness and falling down, but no tonic clonic movements, associated with unrolling of eyes and frothy mouth secretions, and postictal state lasting for 30 minutes. Some events were witnessed by family members, while others occurred in private and were inferred based on post-event findings. Its frequency 4-5 times monthly, with a clear trigger identified in approximately 3 out of every 5 episodes, specifically, exposure to running water during showers.

There is no history of urinary or fecal incontinence, unusual behavior, as well as no motor or sensory deficits, fever, headache, gastrointestinal complaints, or visual complaints. The patient previously underwent treatment with Lamotrigine and Carbamazepine for two full years at an outpatient clinic with no improvement. There was also a trial of Paroxetine, Biperiden, and Risperidone for four months also without improvement. The symptoms kept worsening, thus, he sought treatment at another clinic and was started on Levetiracetam with multiple dose adjustments, but he continued having symptoms. Because of the lack of response to treatment with medication and ongoing seizures, the patient was admitted for further evaluation which included 24-hour EEG monitoring.

During the physical examination, the patient exhibited alertness, consciousness, and orientation to time, place, and person. The individual exhibited comfort and absence of pain, with no indications of cyanosis, jaundice, or respiratory distress. Vital signs were within normal ranges. The neurological examination yielded no significant findings, with no evidence of focal deficits, dizziness, or visual disturbances. On skin examination, there were no rashes or ulcers.

The EEG revealed abnormal background activity consisting of a symmetrical 9 Hz alpha rhythm in the posterior region, mixed with irregular theta and low-voltage beta activity (Figure 1)and normal features during sleep. During monitoring, the patient developed one of his habitual seizures triggered by water contact. The episode was captured on EEG with epileptic discharges, and it terminated spontaneously, confirming the diagnosis of reflex epilepsy, likely triggered by bathing.

Discussion

A rare type of reflex epilepsy known as bathing-related reflex epilepsy (BRE) is typified by seizures that are consistently brought on by exposure to water while taking a bath or shower. This fits our patient's phenotype (triggered by running water, generalized semiology, prolonged post-ictal state, and drug refractoriness). In contrast to classic hot-water epilepsy (HWE), which is typically caused by pouring 40–50 °C water over the head, is frequently focal, and is generally benign, BRE may not depend on temperature, spread, or be drug resistant [1, 3].

Pathophysiology is regarded as sensory-driven: touch and thermal afferents from water exposure stimulate hyperexcitable somatosensory/parietal opercular networks, which extend to temporal–insular or generalized circuits. Case series and functional studies, such as video-EEG and ictal SPECT, indicate the involvement of temporal–insular areas and validate that both hot and cold water can induce seizures, emphasizing a network-based rather than a temperature-specific mechanism [4–6]. Reflex epilepsies are indicative of illnesses characterized by abnormal interactions between environmental stimuli and neural networks, with seizure manifestations contingent upon the hyperexcitable circuitry involved [7, 8].

Phenotypically, BRE includes localized impaired-awareness seizures with autonomic manifestations (pallor, cyanosis), Oro-buccal automatisms, and advancement to bilateral tonic–clonic seizures; certain patients additionally report towel rubbing or anticipatory thoughts of washing as precipitating factors [1, 6]. In contrast, HWE has male preponderance, recurrent temporal lobe semiology, and frequently self-limiting progression with straightforward preventive strategies, such as modifying water temperature [2, 3, 9].

Genetically, increasing evidence associates BRE with SYN1 mutations. Multicenter cohorts validate SYN1-associated X-linked pathways as a principal etiology of reflex bathing seizures, accompanied by ictal-temporo-insular alterations and neurodevelopmental comorbidities in certain carriers [10, 11]. Additional series confirm that bathing or showering can serve as repeatable triggers in SYN1-related epilepsies, alongside other reflex phenotypes like toothbrushing epilepsy, highlighting stimulus-synaptic dysregulation [12, 13].

Diagnosis demands an examination of semiology and ictal EEG

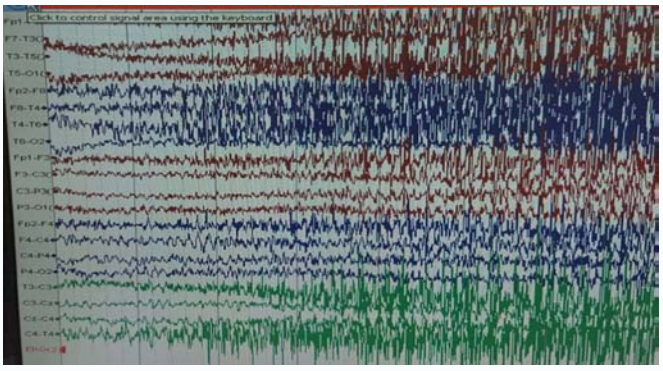

Figure 1: The EEG shows high-amplitude, generalized rhythmic spike-and-wave discharges; the discharges appear at 9Hz (9 cycles per second). Each complex consists of a spike, followed by a slow wave, forming the typical spike-and-wave morphology.

during induced events, as seen in our instance, with neuroimaging frequently appearing normal unless a symptomatic focal lesion is identified. Functional neuroimaging can provide additional delineation of ictal networks [6]. A full evaluation that includes EEG, MRI, and, if necessary, genetic testing makes sure that structural epilepsies are ruled out and the diagnosis is more accurate. The ILAE definition of reflex epilepsies meets BRE. Even though the seizures are only stimulus-locked and happen again and again, they nevertheless meet the criteria of epilepsy because the person is always at risk of having seizures [14, 15].

Management includes both changing triggers and using antiseizure drugs. Prophylactic steps such as exposing the scalp gradually, using lukewarm water, or taking clobazam before bathing may be sufficient in HWE [2, 9]. In BRE, more severe changes to behavior (such shorter showers, handheld spraying, sitting bathing, and supervision) are typically needed, along with long-term ASM. But reactions are different; some SYN1-positive cases have ongoing reflex seizures even after treatment. This shows how important it is to take lifestyle changes and safety precautions together with medication [1, 10].

Prognosis is varied. HWE is often benign and self-limiting during childhood; however, BRE may continue into adulthood and exhibit increased medication resistance, particularly in generalized or genetically influenced variants [3, 9, 16]. The patient's prolonged postictal phase, pharmacoresistant seizures, and temperature-independent seizures are more typical of BRE than HWE, indicating a worse prognosis. Important aspects of care still include genetic evaluation, strict trigger adjustment, and bathing safety procedures.

Conclusion

This case illustrates the significance of identifying bathing-related reflex epilepsy as a unique, non-temperature-dependent variant of reflex epilepsy. Being aware of this uncommon disorder can help avoid misdiagnosis, lead to the right EEG-based evaluation, and improve treatment by avoiding triggers and adjusting therapy to each individual.

Declarations

Conflict of Interest: The authors declare no conflicts of interest.

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