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Bathing epilepsy: report of two Caucasian cases

Emilio Franzoni[1], Valentina Gentile[1], Salvatore Grosso[2], Daniela Brunetto[1], Duccio Maria Cordelli[1,3], Paolo Balestri[2]

[1] Child Neuropsychiatry Unit, Paediatric Department, University of Bologna
[2] Department of Pediatrics, Pediatric Neurology Section
[3] PhD school in Applied Neurological Sciences, University of Siena, Italy

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ABSTRACT – Bathing epilepsy, also known as water-immersion epilepsy, refers to a rare form of benign reflex epilepsy in which seizures are precipitated by the normal process of domestic bathing. This condition has been confused with true hot water epilepsy, even though bathing in water at normal temperature is the trigger. Focal seizures predominate with a staring gaze, pallor and generalised features followed by prolonged postictal somnolence. A variable percentage of patients may also show unprovoked seizures. The prognosis is usually favourable, and modifying bathing habits may prevent further seizures. We report two Caucasian patients with bathing epilepsy. In one, seizures were provoked by water immersion. In the other, we noted an unusual triggering factor; pouring of lukewarm water over the genitalia.

Key words: reflex seizures, bathing epilepsy, water-immersion epilepsy

Bathing epilepsy refers to a specific type of reflex epilepsy triggered by domestic bathing in water at normal temperature (Nechay and Stephenson, 2009), with commonly a favourable prognosis. It is often confused with true hot water epilepsy (Bebek *et al.*, 2001; Satishchandra, 2003), a condition best known in southern India where there is a strong genetic predisposition (Ratnapriya *et al.*, 2009a, 2009b).

We describe two infants of Caucasian descent suspected to be affected by bathing epilepsy: in one, the seizures were caused by immersion in normal temperature water; in the other, the trigger was pouring of lukewarm water over the genitalia.

Case study

Patient 1

A five-year-old girl was born to healthy and nonconsanguineous parents, with an uneventful delivery. There was no family history of epilepsy or febrile convulsions. Psychomotor development was normal. At the age of 11 months, she started to present episodes of staring gaze, pallor, loss of awareness, ictal vomiting, generalised hypotonia and postictal somnolence each time she was bathed in normal temperature water. These episodes occurred within 20-30 seconds of the child being immersed in water and lasted 1-3 minutes. No seizures were observed if the child was washed using wet cloths. Physical examination was normal as well as biochemical investigations and interictal electroencephalography (EEG) recordings. The parents refused to give permission to perform ictal EEG while bathing.

Modifying the child's bathing habits by using wet cloths or showering prevented further seizures. One month later, the patient started to present non-reflex seizure episodes characterized by loss of consciousness, a staring gaze, pallor, ictal vomiting and

Correspondence:
E. Franzoni
Child Neuropsychiatry Unit, Paediatric Dept, University of Bologna, Via Massarenti 11, 40138 Bologna, Italy
<emilio.franzoni@unibo.it>

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generalised hypotonia lasting two minutes or more with prolonged postictal somnolence. The episodes occurred monthly, and each lasted about 1-3 minutes. Interictal EEGs showed sporadic spikes and sharp waves in the right centrotemporal area. Electrocardiogram (ECG) and Holter ECG were normal, as were metabolic investigations and brain magnetic resonance imaging (MRI). The patient was treated with carbamazepine (28 mg/kg/day). The EEGs became normal. At the last visit, the patient was seizure free.

Patient 2

A seven-year-old girl was born after an uncomplicated pregnancy. Her parents were healthy and unrelated. There was no family history of epilepsy and no past history of febrile convulsions. Psychomotor development was normal. The parents reported that, from the age of four months, she had developed convulsions when lukewarm water was poured over her genitalia. About 5-30 seconds after the stimulus, the child invariably presented with loss of consciousness, asymmetric tonic phases more prominent on the right side and initial tonic deviation of eyes to the right followed by a wandering gaze. Each episode, lasting 2-4 minutes, was followed by crying and prolonged postictal drowsiness. Water immersion, tactile stimuli of the genital area with wet or dry cloths did not precipitate seizures. Ictal Holter EEG showed generalised high amplitude waves over the scalp, more synchronous on the left central area, and apparently not preceded by any focal activity. MRI was normal.

Seizures were prevented by modifying bathing habits; immersing the child in water prior to being subjected to poured water. At follow-up the child was seizure free.

Discussion

Reflex epilepsy due to bathing is referred to as "bathing epilepsy" or "water immersion epilepsy", according to the International League Against Epilepsy (ILAE). It is not to be confused with true hot water epilepsy which is more frequent in southern India (Satischandra, 2003) and Turkey (Bebek *et al.*, 2001), but uncommon in western countries; recent studies have pointed to specific genetic factors (Ratnapriya *et al.*, 2009a, 2009b).

According to Nechay and Stephenson (2009), and in our cases, contact between the child's body and water of normal temperature causes the seizures. In most European countries, a temperature around 37°C is the normal temperature of a bath for infants. Clinical semiology includes a staring gaze, pallor and hypotonia.

The pathophysiology of bathing epilepsy remains unknown. The complex partial nature of seizures and the focal features observed in the few ictal recordings performed, seem to suggest a temporal lobe involvement. However, a possible role of sensory cortex in the epileptogenic process might be involved in some cases (Grosso *et al.*, 2004).

It seems that a complex combination of multiple specific stimuli are needed to trigger seizures. In this context, in patient 1, seizures were precipitated by immersing her in water with a normal temperature. By contrast, in patient 2, seizures were trigged by the pouring of lukewarm water over the genitalia, whereas other tactile stimuli applied to the same area did not precipitate seizures.

Interictal EEGs are usually normal in patients with bathing epilepsy. Characteristically, no spikes or spikes and waves were present on ictal EEGs, which showed anomalies represented by focal, usually temporal, unilateral, rhythmic slow wave activity of high amplitude with secondary generalisation after a few seconds (Ceulemans *et al.*, 2008). Although no ictal EEG was available in patient 1, Holter system EEG performed on patient 2 recorded diffuse high-amplitude slow waves not preceded by a clear centrotemporal origin. No spikes or spikes and waves were observed, confirming previous reports (Ioos *et al.*, 2000).

Bathing epilepsy should be differentiated from the other paroxysmal events that may occur during bathing, in particular alternating hemiplegia of childhood (AHC) (Nechay and Stephenson, 2009; Incorpora *et al.*, 2008; Incorpora *et al.*, 2009).

Vasogenic syncope is a diagnostic possibility that has been suggested in subjects, as in patient 1, in whom the seizure was typified by staring gaze, pallor and generalised hypotonia. The use of ECG or Holter-ECG is helpful to identify patients with bathing epilepsy, since tachycardia, rather than bradycardia, is commonly present during and after clinical attacks, and there is no evidence that bathing in infancy is a trigger for non-epileptic syncope (Nechay and Stephenson, 2009).

The form of seizures of patient 2, with asymmetric tonic phases, was less compatible with a vasogenic type of syncope. In this patient, in contrast, the diagnosis of startle epilepsy should be considered. Although domestic bathing may be a trigger for hyperekplexia (Nechay and Stephenson, 2009), the normal neurological development and the longer duration of seizures observed in this patient exclude this diagnosis.

Although video-EEG monitoring with simultaneous ECG remains a cornerstone for the diagnosis, the recurrence of seizures while bathing, together with the absence of vagal symptoms during the attacks and the efficacy of modifications to bathing habits, are indirect arguments for diagnosis. Incorpora *et al.* (2008, 2009) reported twins first diagnosed with bathing epilepsy but who were subsequently shown to have AHC. In the neonatal period both twins had reflex seizures each time they were immersed in bath water temperature around 30°C but later had paroxysmal episodes, unrelated to water immersion, including alternating hemiplegia. In our two patients we can also exclude the diagnosis of AHC.

From a therapeutic point of view, modifying bathing habits (showering or sponging instead of bathing) commonly prevents further seizures. Anticonvulsants are usually efficacious but are only indicated when other measures are ineffective or when non-reflex seizures are noted in a patient's history (Grosso *et al.*, 2004). Using wet cloths or showering effectively prevented further seizures in patient 1. In contrast, gentle water immersion, rather than splashing water over the genitalia, was able to control seizures in patient 2.

In conclusion, we describe two further cases of Caucasian patients with bathing epilepsy. For one, the seizures were induced by exposing the genital area to flowing lukewarm water rather than by more typical water immersion. We agree with other authors (Nechay and Stephenson, 2009) who state that water bathing itself is the trigger of the seizures and that simply modifying bathing habits is sufficient to prevent them. The prognosis is commonly favourable: there is mostly spontaneous remission and psychomotor development remains normal (Ceulemans *et al.*, 2008). Exceptionally, bathing epilepsy may evolve into a non-reflex epilepsy. □

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Disclosure.

None of the authors has any conflict of interest to disclose.

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