"HOT-WATER EPILEPSY", "WARM-WATER EPILEPSY", OR BATHING EPILEPSY?

Report of three cases and considerations regarding an old theme

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ABSTRACT - Partial and generalized tonic-clonic reflex seizures related to hot water bathing have been described as temperature-related. We describe three cases of bathing epilepsy: a 28 year-old white male and a 30 year-old white female with spells triggered either by warm or hot water, and a 32 year-old female with spells triggered by hot water. The later two of the three cases presented localized epilepsy and a familial history of epilepsy. A complex tactile stimuli might play the most relevant role on seizure triggering, as well as water temperature with an additive effect over cutaneous stimulation.

KEY WORDS: epilepsy, epilepsy-generalized, epilepsy-localized, seizures-reflex, hot-water epilepsy.

Epilepsia do banho quente, do banho morno, ou apenas do banho? Relato de três casos e considerações sobre um velho tema

RESUMO - Crises reflexas a banhos quentes, tônicas-clônicas parciais e generalizadas foram descritas como relacionadas à temperatura. Descrevemos três casos de epilepsia do banho quente: um homem de 28 anos e uma mulher de 30 anos com crises provocadas por contato com água morna ou quente e uma mulher de 32 anos com crises ao contato com água quente. Os últimos dois casos apresentaram epilepsia localizada e um histórico familiar de epilepsia. Nesta forma de epilepsia, um estímulo táctil complexo parece ter o papel mais relevante na precipitação das crises, sendo potencializado pela temperatura da água.

PALAVRAS-CHAVE: epilepsia, epilepsia generalizada, epilepsia localizada, crises reflexas, epilepsia do banho quente.

Hot water epilepsy has been previously studied and reported, occurring mainly in India^{1,2,3}. Both partial and generalized tonic-clonic seizures related to hot water bathing have been described as temperature-related, often triggered by temperatures ranging from 40 to 50°C^{1,3}. As in many other types of reflex seizures, the same stimulus (hot water) triggers either generalized tonic-clonic or partial seizures, suggesting that generalized and localized epilepsies might be influenced by the same afferent pathways.

We describe three cases of bathing epilepsy, one triggered by hot water, and the other two either by warm or hot water. The pathophysiological significance of seizure triggering by warm water and the proper denomination of this syndrome is discussed.

CASES

Case 1 – A 28 year-old white male started having seizures at the age of 7. Seizures would start with dizziness, followed by left hemiclonic seizures starting on his left arm. He had had birth anoxia, and his brother also had epilepsy. Seizures were initially controlled with phenobarbital, but recurred at the age of 14, when partial seizures characterized by bilateral headache, cephalic paresthesia, and a negative motor phenomenon on his left upper limb were triggered either by warm (but not cold) or hot water. Symptoms lasted about 5 minutes. The CT was normal, but an interictal EEG showed a right rolandic focus and an independent left temporal spiking. Carbamazepine 1200mg/day reduced seizure frequency from 3/week to 4/month.

Case 2 – A 30 year-old white female started presenting seizures at the age of 26, during pregnancy. Seizures

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Received 15 August 2003, received in final form 29 November 2004. Accepted 31 January 2005.

occurred only during warm or hot shower bathing and consisted of a brief complex visual hallucinatory phenomenon and speech arrest, followed by vomiting and a generalized convulsive seizure. Post-ictal state was characterized by a severe throbbing headache. Seizures occurred twice-a-month, always during bathing. Her brother had atypical febrile seizures in childhood, followed by generalized seizures, controlled by sodium valproate. Her physical and neurological examinations were normal, as also wereher CT and MR imaging of the head. Carbamazepine had been ineffective for seizure control, but seizures abated when sodium valproate 3000mg/day was added to phenobarbitone 100mg/day, recurring only when the phenobarbitone dose was lowered.

Case 3 – A 32 year-old female started having seizures at the age of 20, occurring mostly during bathing. The seizures were partial complex, fugacious, with the left hand and lips closing for about 1 to 2 minutes. She felt bad the rest of the day in post-ictus. In the beginning, she had 6 spells per month, most of them as a result of water-contact, evolving to almost daily spells. She made use of phenytoin and carbamazepine. During her only pregnancy her seizures decreased and recurred after delivery. Her cervical radiography, CT and MRI of the head were normal and four seizures triggered by water contact on the upper limbs recorded at a video-EEG without concomitant specific EEG-abnormalities. A deeper psychiatric evaluation excluded a DSM-IV disorder and the hypothesis of non-epileptic nature for the events. Carbamazepine 200mg had been ineffective for seizure control, but seizures decreased when clonazepam 0.5mg b.i.d. was associated to carbamazepine 400mg b.i.d.

DISCUSSION

Bathing epilepsy occurs in both localized and generalized epilepsy, as a reflex to immersion or shower bathing¹⁻³. Hot water bathing is an especially strong precipitating factor for severe myoclonic epilepsy in infancy or its peripheral form⁴. Some patients reported bathing at lower temperatures or with a gradual exposure to water, starting with areas showing minor tendency to trigger the attacks, as a seldom effective way to avoid seizures (Arruda F, personal communication). The major findings in bathing epilepsy are that it has a male preponderance with ratios higher than 70% for men^{3,5}, can be selfinduced, is known as benign and self-limited, often performed for pleasure, has complex triggering factors, and shows temporally-related abnormalities in the EEG tracings^{1,5}.

Although bathing epilepsy has been claimed to be temperature-related, its mechanisms remain uncertain. However, there is in the literature reports of seizures related to bathing irrespective of water temperature⁶. In India, where this kind of reflex seizure seems to be more frequent than elsewhere³, ritual immersion baths at temperatures around 45°C are reported to trigger epileptic fits, often complex partial or generalized seizures⁷. This temperatureis the one at which the warmth receptors come into action at the highest rates, and temperatures above this level, also known as "noxious heat", we redescribed to prog ressively turn off the warmth receptors. Another phenomenon that occurs at temperatures within the "noxious heat" bracket is the perception of pain through the activation of heat nociceptors^{7,8}. The patients reported in this paper did not complain of heat-triggered pain, suggesting that pain fibres were not activated during the spells.

The fact that these types of spells have not been reported in other places of increased heat, such as saunas or furnaces, suggests that complex tactile stimuli, rather than temperature, might play the most relevant role on seizure triggering. The reports of the latter two of the three cases here described reinforce this view, since bathing at a water temperature, quoted as warm but not hot, also triggered the seizures. This finding reinforce our view that the expression "bathing epilepsy" rather than "hot-water epilepsy" should be preferred, when referring to patients afflicted by this (these) disorder(s). Another aspects regarding both aforementioned cases is that both presented localized epilepsy and a familial history of epilepsy, reinforing the role of a genetically-based lower threshold for seizures.

A reasonable mechanism would be a defective inhibitoryinfluence over afferent volleys of somatosensorystimuli, such as warm or hot water over large body surfaces. In this sense, water temperature might have an additive effect over cutaneous stimulation. Reflex epilepsy to hot-water in children suggests a mechanism similar to febrile seizures^{4,9}. The relationship between sensory stimulation, epilepsy and blood-brain barrier function still remains as a mystery. Ibay et al. suggeste that the opening of the blood-brain barrier caused by acute triggers may be an early etiological mechanism for seizures or, by other hand, an important consequence of seizures^{10,11}.

The treatment of hot-water epilepsy may not need antiepileptic drugs usage; lowering the both temperature or changing the method of washing may be enough to control the seizures, and to avoid the trigger-factor^{3,12}. Antiepileptic drug therapy is only indicated when those measures fail or when other types of epilepsy are involved. Since seizures f requently show a tendency to decrease spontane o-

usly, withdrawal of medication should be carefully undertaken only after several years^{13,14}.

Post-scriptum - After this paper submittal, Guilhoto and Degenszajn have reported another single case of bathing epilepsy, whose seizures were controlled by sodium valproate and lamotrigine. The authors considerate that in patients with refractory seizures, bathing epilepsy should be sought, and that seizures susceptibility might reflect focal phenomena in a generalized excitable cortex (Guilhoto LMFF, Degenszajn J. J Epilepsy Clin Neurophysiol 2004;10(Suppl 1):8.

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