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Bath-Related Headache

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Abstract
Purpose of review:
The purpose of this review is to summarize the most up-to-date literature on bath-related headache, a rare disorder.
Recent findings:
Initially described in middle-aged Asian women, it is now reported in a wider demographic. More information is available about the pathophysiology of bath-related headache, including its classification as a subtype of reversible cerebral vasoconstriction syndrome (RCVS). Nimodipine can be effective in patients both with and without vasospasm
Summary:
Bath-related headache is a rare form of thunderclap headache. Although its mechanism is still unclear, it is associated with vasospasm and RCVS. Controlled trials investigating the use of nimodipine and other agents may be useful in furthering our understanding of and treatment of this phenomenon.

Key words
Bath-related headache; thunderclap headache; reversible cerebral vasoconstriction syndrome (RCVS)

Introduction

Bath-related headache is a rare form of thunderclap headache that has primarily been described in case reports and case series. One of its most distinguishing features is the trigger, which is typically bathing or other water-related activity. It is likely underreported.

Clinical characteristics

Bath-related headache was first described in 2000 by Negoro et al [1*]. It is more common in Asian populations and has been reported more recently in Europe and South America [2, 3]. There is a female predominance, but at least one case in a man has been described [4]. The most common age of onset is in the fifth to seventh decade; the youngest reported age in the literature in the second decade [5, 4]. A minority of patients have a history of other headache types such as migraine or tension headache [4, 5].

The semiology of pain varies, but in general is provoked by contact with water [5]. Bath-related headache is typically abrupt in onset, severe in intensity, reaches a peak within one minute, and lasts between 10 minutes and 9 hours [1*, 5]. Liao first suggested in 2003 that the bath-related headache is a variant of idiopathic thunderclap headache [6, 7].

Different precipitating activities are reported, including pouring hot water over the head, diving into a swimming pool, warm washcloth on the lower half of the body, warm or cold showers, or soaking in hot water [1*, 5, 6]. Less frequent causes include brushing teeth with cold water, exposure to steam, and micturition [5, 6].

Location of pain also varies, and descriptions include holocephalic, unilateral, frontal, occipital, bitemporal, vertex, and cervical [4, 5]. In one series, pain quality was most often explosive or pulsatile and typically bilateral or midline [8**]. There can be associated scalp tenderness, nausea, vomiting, and pain between attacks [5].

Bath-related headaches are typically self-limiting. The usual duration of triggered headaches prior to remission is between one week and three months without subsequent clusters,

although one case series indicates bath-related headache can be recurrent for a period of greater than 15 years [2, 4].

Pathophysiology

The pathophysiology of bath-related headache is as of yet unknown and might be multifactorial. There are several proposed mechanisms. Multisegmental vasospasm has been detected via vessel imaging (MRA, conventional angiogram, CTA) in some patients with bath-related headache [5, 6]. One large case series shows vasospasm within the Circle of Willis in the majority (62%) of patients, most frequently in the middle cerebral artery or posterior cerebral artery [8**]. Posterior leukencephalopathy has been seen on MRI in association with vasospasm [5, 8**]. Other abnormalities seen on imaging in the setting of vasospasm include asymptomatic cerebellar infarct in one patient, and both subarachnoid hemorrhage and delayed intracerebral hemorrhage in another patient [5, 9].

The detection of reversible vasospasm (resolution determined by repeat vessel imaging or normalization of velocities on transcranial Dopplers) does not always correlate with resolution of headache [6]. Vasospasm can resolve prior to headache remission, and resolution of headaches can occur prior to reversal of vasospasm [10, 6]. This suggests a complex mechanism.

An overactive neurogenic vasomotor response might be responsible for the vasospasm seen in bath-related headache and other thunderclap headaches [11**]. It has been suggested that bathing can be a trigger of RCVS, similar to other activities such as straining or coughing [8**]. In fact, under the International Classification of Headache Disorders, Third edition (ICHD-3), thunderclap headaches related to bathing and showering are "attributed or probably attributed" to reversible cerebral vasoconstriction syndrome (RCVS) [7].

Other proposed mechanisms for bath-related headaches exist. There is possibly a predisposition based on age, sex, and ethnicity given the demographic described [3]. It has been suggested that low levels or fluctuations of estrogen, such as occurs in menopause, might be directly impactful [8**]. Aquadynia is a term used for an overactive sympathetic response to skin stimulation by water and is a possible contributor to bath-related headache [12]. Alternatively, these patients could have highly sensitive temperature receptors on the face and scalp which are triggered by either excessively hot or cold water [1*]. This might share a common mechanisms with cold-stimulus headache [1*, 4]. Although not described in the literature, a temperature-sensitive sodium channelopathy is another possibility.

Treatment

One logical choice of management is to avoid the known trigger (eg, pouring hot water over the head, taking a shower, or soaking in hot water) [1*]. This has been reported as effective although may not be practical.

Alternatively, pharmacologic therapy has been used, both preventive and abortive. Nimodipine is a calcium channel blocker that is often used in preventing or treating vasospasm in subarachoid hemorrhage and has been used effectively in bath-related headache [11**]. One study describes the use of oral nimodipine for patients without vasospasm and higher dose IV formulation for patients with vasospasm [11**, 6]. In another series, oral or IV nimodipine at a dose of 30-60 mg q4 hours for a course of 3-4 weeks was effective in the majority (16/19, or 84%) [8**]. The exact mechanism by which nimodipine works in the patients without vasospasm is unclear, but the time course presented in studies suggests against spontaneous remission or placebo effect [11**]. Other preventive medications reported include amitriptyline, gabapentin,

sodium valproate, and topiramate [5, 13]. Their effectiveness is unclear given bath-related headache's high rate of spontaneous remission and lack of controlled trials.

Many abortive medications have been used anecdotally, including non-steroidal anti-inflammatory medication, acetaminophen derivatives, and triptans [5, 10]. Most are poorly effective. Triptans have been reported as efficacious, although the safety of using them given the association of vasoconstriction is questionable [10].

As bath-related headache is also possibly associated with an overly active sympathetic nervous system, medications targeted at the autonomic nervous system such as clonidine and propranolol could theoretically be helpful, although their use has not been reported [12].

Conclusion

Bath-related headache is an unusual type of thunderclap headache with a unique trigger and varied semiologies. Initially thought to be primarily seen in middle-aged Asian women, it is now known to be present in others and likely is underreported. Its association with vasospasm is highly suggestive of a subtype or trigger for RCVS, although the extent of this relationship is not clear and other factors are likely involved given the lack of correlation between resolution of headache and resolution of vasospasm. Avoidance of triggers is an effective if impractical approach. Nimodipine can be used effectively in patients with or without vasospasm. More research is needed to further our understanding of this rare headache phenomenon.

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