Clinical Correspondence

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Managing external cold-stimulus headache with preventive naproxen

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Abstract

The management of external cold-stimulus headache has previously focused on trigger avoidance, which can be an impractical and sometimes impossible solution. We describe the case of a 20-year-old woman who presented with a typical example of external cold-stimulus headache, and in whom a preventive regime of naproxen taken 30 min prior to cold exposure was associated with reliable prevention of cold-induced headache symptoms. This could be an effective strategy for managing patients with cold-stimulus headache for whom cold triggers cannot be avoided.

Keywords

clinical neurology history, cold-stimulus headache

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Introduction

Cold-stimulus headache (also termed ice cream headache or brain freeze[1]) is, in clinical practice, a relatively rare headache disorder typified by headache following ingestion of a cold substance. Cold exposure can however take different forms, and the International Classification of Headache (ICHD-3) also includes external cold-stimulus headache, which refers to headache caused by the external application of a cold stimulus.[2] The exact prevalence of this headache disorder is unknown.

No previous studies exploring therapeutic options for external cold-stimulus headache have been performed. While usually brief (lasting seconds or minutes in duration), headache can last for up to an hour following exposure, and although the usual treatment of cold-stimulus headache revolves around trigger avoidance, this is sometimes impossible.

We describe a typical case of prolonged external cold-stimulus headache and report an excellent response to a preventive strategy of oral naproxen.

Case

A 20-year-old university student presented with a 12-month history of persistent headache triggered by environmental cold exposure. She had a past medical history of asthma without a previous significant headache history. In particular, she had never experienced significant hangover or menstrual headaches. There was a family history of her mother, brother and sister having previously suffered migraine. Her medications included salbutamol taken on an as-needed basis and the combined oral contraceptive pill (ethinylestradiol 20 mcg and drospirenone 3 mg).

She described an intense (NRS headache score 5) bi-frontal headache invariably triggered by external cooling of the head, such as occurred during contact with cold wind (e.g. through the car window), exposure to air-conditioning or immersion of the head in cold water. These symptoms would arise almost immediately upon exposure to the cold stimulus and take 30–60 min to settle following removal of these triggers (see Online Supplemental video). Headache

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intensity increased proportionate to both time spent and magnitude of the cold environment. There was no change on physical activity. There was no associated nausea, vomiting or photophobia and no cranial autonomic symptoms such as lacrimation, nasal congestion, facial sweating or conjunctival injection noted at any time.

Neurological examination both during and immediately following a triggered attack was unremarkable.

Paracetamol 500 mg, ibuprofen 400 mg and aspirin 650 mg taken during headache episodes had all proven ineffective in controlling symptoms and only the application of warm headwear would provide mild alleviation to her pain.

The patient was diagnosed with external cold-stimulus headache and we prescribed a preventive regime of naproxen 750 mg taken 30 min prior to cold exposure. This was associated with reliable prevention of symptoms, to the extent she would experience either no symptoms at all or only mild discomfort during cold exposure. She took preventive naproxen 2–3 times a week for several months before this was no longer required.

Discussion

Headache experienced during external exposure to cold environments is rarely encountered in clinical practice. The underlying mechanism remains unknown although it has been proposed to be vascular, where sudden exposure to cold may trigger rapid constriction of vessels thus activating vessel wall nociceptors.[3] An alternative explanation may be pain from an ungated trigeminal afferent barrage triggered by the cold stimulus that is not modulated effectively. Certainly, trigeminocervical afferents can be shown to have a cyclooxygenase component experimentally, which could be invoked in the context of naproxen's effect.[4] Moreover, aspirin can modulate induced allodynia in migraineurs, which to some extent links these observations.[5] It is tempting to think of a role for the TRPM8 cold receptor, which is found in trigeminal neurons.[6] More could be done to understand the basis of headache with exposure to cold.

Trigger avoidance in cold-stimulus headache can range from being inconvenient to impossible and as such medical treatment strategies may in some situations be warranted. We report a case where pre-emptive use of naproxen was very effective in preventing external cold-stimulus headache.

Clinical implications

(1) External cold-stimulus headache can be experienced upon exposure to cold environments, with pain lasting up to 60 min after removal of the cold trigger.
(2) Pre-emptive use of naproxen may be effective in preventing external cold-stimulus headache in cases where trigger avoidance cannot be obtained.

Declaration of conflicting interests

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Informed consent

Informed consent was obtained in written form from the patient.

Supplemental material

Supplemental material for this article is available online.

References

1. Raskin NH and Knittle SC. Ice cream headache and orthostatic symptoms in patients with migraine. *Headache* 1976; 16(5): 222–225.
2. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. *Cephalalgia* 2018; 38(1): 1–211.
3. Starling AJ. Unusual headache disorders. *Continuum* 2018; 24(4): 1192–1208.
4. Kaube H, Hoskin KL and Goadsby PJ. Intravenous acetylsalicylic acid inhibits central trigeminal neurons in the dorsal horn of the upper cervical spinal cord in the cat. *Headache* 1993; 33(10): 541–544.
5. Akerman S, Karsan N, Bose P, et al. Nitroglycerine triggers triptan-responsive cranial allodynia and trigeminal neuronal hypersensitivity. *Brain* 2019; 142(1): 103–119.
6. Huang D, Li S, Dhaka A, et al. Expression of the transient receptor potential channels TRPV1, TRPA1 and TRPM8 in mouse trigeminal primary afferent neurons innervating the dura. *Mol Pain* 2012; 8(66): 1744–8069.