J Headache Pain (2004) 5:201–203
DOI 10.1007/s10194-004-0102-1

BRIEF REPORT

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Visual aura in ice cream headache

Received: 11 September 2003
Accepted in revised form: 26 May 2004

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Abstract We present a case of cold-induced visual symptoms in a 74-year-old woman with a history of migraine headaches. Migraine without aura was the starting symptom. It coexisted later with a typical pain of ice cream or *cold stimulus headache* that persisted after the migraine vanished. Finally the headache disappeared and the course was characterised by episodes of pure visual symptoms, always after swallowing cold material. The vasodynamic effects underlying migraine are invoked to understand this case, but the activation of the trigeminal system provides a better explanation for the symptoms of this patient. Thus trigeminal activated headache and aura could include a subform like this one, characterised by pure hemianopsia after ingesting cold material.

Key words Cold stimulus headache • Ice cream headache • Visual aura

Introduction

Ice cream headache is a form of cold stimulus headache. It is the result of the passage of cold material, solid or liquid, over the palate and posterior pharyngeal wall. Consequently, it has been termed headache attributed to ingestion or inhalation of cold stimulus in the new International Headache Classification [1]. This headache has been related to migraine, especially to active migraine sufferers with typical symptoms [2]. It would be a consequence of the pathophysiological vasodynamic mechanisms that are also present in migrainous patients [3, 4].

Because the vascular mechanisms are shared, other clinical features of migraine could equally be foreseen and explained in the ice cream or cold stimulus headache. The present case illustrates this point of view. It combines a prior history of migraine with a cold stimulus headache, which was finally transformed in episodes of pure visual hemianopsia after intake of cold material.

Case report

The patient was a 74-year-old woman that was remitted after having suffered multiple episodes of diminished visual acuity, which followed the intake of cold material.

Her past medical history was unremarkable. Between her adolescence and her last menses the patient had frequent episodes of recurring migraine without aura episodes. In her mid-40s they started to be associated with short bouts of frontal headache soon after the ingestion of cold food or liquids. In her early 50s she became migraine-free and thereafter she was aware of more com-

monly suffering short attacks of midfrontal pain a few seconds or a minute after the intake of any cold material. She did not seek medical attention because the cold stimulus headache vanished just by avoiding contact with cold beverages or food.

In her mid-50s she experienced her first episode of diminished visual acuity: It was shortly after the ingestion of a cold beverage. On this occasion she did not feel headache, but only visual symptoms. She perceived a dark spot in the centre of vision of both eyes, which persisted for about 30 min. The spots then cleared slowly. At the same time that hazy vision expanded laterally, with an outward zigzag line that finally adopted a right hemianopic pattern. This symptom was better tolerated and disappeared slowly, starting laterally and lasting an interval of several hours. Later on this woman experienced more occasional attacks of pure visual symptoms. They followed the involuntary intake of cold fruits, liquids or ice cream and tended to last longer: central scotomas could be recorded in a campimetric test 24 h after their appearance. A lateral and outer expansion of her right lateral extended area of diminished vision could also be tested, confirming her right hemianopic pattern.

Funduscopic and neurologic exams were otherwise normal. Routine blood analysis that included VDRL and HIV tests, antinuclear and anticardiolipin antibodies were within normal range. CT scan, brain MRI, carotid and vertebral ultrasonography, EKG and transthoracic echocardiography were irrelevant. Transcranial doppler and MR angiography disclosed patent basilar, posterior cerebral, carotid arteries and their distal branches, with no visible lesions. The patient has been reassured and reminded of the importance of avoiding the known triggers. At 76 years she has neither headache nor her disturbing visual episodes.

Discussion

This case supports the hypothesis that headache attributed to ingestion or inhalation of cold stimulus can be contemplated as a model of migraine. The postulated mechanism in this entity would also run for the cold stimulus form. A cerebral vasoconstriction has actually been proved by using transcranial Doppler ultrasonography once the headache has developed [5]. Local cooling would also lead to a sequence of waning and subsequent waxing arterial flow. Thus the pain may appear as a consequence of a direct cold pressor effect, as it has been described during surfing in winter and in divers [6], although autoregulation that protects the brain from any systemic vascular changes should be overtaken in order to get to the painful aftermath. The exposure of the head to low temperatures triggers a headache not distinct from the cold stimulus variety. The latter can be understood as a referred pain, by the trigeminal nerve when the pain is in the forehead or temple, and by the glossopharyngeal nerve when the pain is located in the ears [4].

Alternatively to the vascular theory, the trigeminal theory of migraine could also support the present case. The activation of the trigeminal system may lead not just to migraine, but equally to other headaches such as ice-pick headache, hemicrania continua and cluster headache. Thus these headaches might coexist and occur along with migraine or any other one among them. In addition, visual auras are no longer an event that only occurs with migraine. They have been known to occur without headache and moreover have recently been found with cluster headache [7], hemicrania continua [8] and chronic paroxysmal hemicrania [9]. In this way it is less likely that headache attributed to ingestion or inhalation of cold stimulus is a form of migraine, but rather a form of trigeminal activated headache. In similar terms, migraine aura is in fact a neurogenic phenomenon that so far had not been described as a one triggered by cold stimulus. Also worth noting is that there is no anatomical relationship between the trigeminal system and the visual system, a point that certainly makes it difficult to provide an explanation for this case through the trigeminal theory.

This patient's visual symptoms fit a typical visual aura when taking into account the clinical pattern and evolution. They were long lasting, as they persisted within minutes and 24 h. Nevertheless they do not fulfil the criteria for persistent aura without infarction, as the visual symptoms do not last more than a week, as is mandatory in the new classification [1]. Also worth noting is the absence of headache once the visual symptoms emerged, which matches the case of aura without headache [1]. They are more common in patients of advancing age, some of whom may alternate attacks of aura with headache with other attacks of aura alone or aura without headache [10]. But migraine and cold induced headache would just be concurrent, as in fact happens in the vast majority of patients with both of these headaches. Thus aura could not be linked to migraine. As a matter of fact, what we want to underline is that the auras were prolonged and induced by a cold stimulus, which has not been previously reported as far as we know.

A final diagnostic consideration takes into account transient ischaemic attacks playing a role in the development of the visual symptoms in this patient. Headache may in fact be associated with ischaemic cerebrovascular diseases. Nevertheless, this mechanism is unlikely: visual symptoms emerged in exactly equal bouts, without headache, multiple although always transient, and finally

with no responsible vascular lesion disclosed in neuroradiological or sonographic tests.

Not to be overlooked is the fact that this is a case report, and so it is not the most suitable form to discuss pathogenic mechanisms or to accept a proposal hypothesis. However, it may be of help for further discussion in the field of ice cream headache.

We believe that this case had different subforms of the same entity, shifting from one into another while sharing common underlying mechanisms. It can be contemplated according to the vascular theory of the cold stimulus headache, although the activation of the trigeminal system seems a more plausible mechanism. The clinical spectrum of trigeminal activated headaches is likewise broadened.

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