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Dynamic Mechanical (Brush) Allodynia in Cluster Headache

Background

Cutaneous allodynia is the perception of pain when a non-noxious stimulus is applied to normal skin.¹ It has been described in patients with migraine.²⁻⁴ Cutaneous allodynia is caused by sensitization of central nervous system neurons that receive convergent sensory input from both skin and intracranial structures.⁵ This phenomenon has not been previously described in patients with cluster headache. Although migraine and cluster headache (CH) may share some clinical features, the pathogenesis of these two primary headaches is different.⁶

The aim of this study was to examine the occurrence of dynamic mechanical (brush) allodynia (BA) in patients with CH.

Patients and Methods

We examined adult patients with episodic or chronic cluster headache (CH). Demographic data and the characteristics of each patient's CH (type of CH, phase in the disease, disease duration and disease severity) were obtained through a standardized questionnaire. Allodynia testing was performed by repetitively applying a 4x4-inch gauze pad at a rate of 2/sec to six skin areas bilaterally, in the trigeminal and cervical distributions (frontal (V₁), maxillary (V₂), mandibular (V₃), posterior neck (C₂, C₃), shoulder (C₅) and inner forearm (C₈)). For patients in acute attack, allodynia testing was performed before treatment and 10 and 20 minutes thereafter. The patients were asked to quantify the degree of allodynia on a 100 mm visual analog scale (VAS). They

were also asked to report on headache at the time of testing and to rate it on an 11-point verbal scale. The relations between the location and severity of headache and allodynia were assessed.

Results

Ten patients were included in the study. All were male. Their mean age was 39.3 years (range: 26-51). Seven patients had episodic CH (ECH) and three had chronic CH (CCH). Two patients were in acute attack when tested (both had ECH). Five patients were in cluster period but not in acute attack. The three CCH patients were not in acute attack. Disease duration ranged from 18 months to 38 years. One patient (who had ECH) also suffered from migraine with aura. Eight patients were on headache preventive medications. Seven of them were on monotherapy (four on verapamil, two on lithium and one on valproic acid) and one was on polytherapy (verapamil and valproic acid).

In total, four of the ten patients (40%) had brush allodynia, two of the seven with episodic CH (28.6%) and two of the three with chronic CH (66.7%) (Table 1). Of the two patients in acute attack (both had episodic CH), one had BA, which was ipsilateral to the pain. The allodynia decreased 20 minutes after treatment with oxygen (average allodynia score per site: before treatment - 16.66, after treatment – 7.16). Pain score also decreased after treatment, from 8 to 3 (on an 11-point verbal scale). The other patient in acute attack had no allodynia. Of the five patients in cluster period but not in acute attack, one (20%) had BA. Two of the three (66%) patients with chronic CH were found to have BA.

We found a trend for allodynia to occur in patients with longer disease duration (median disease duration – 22 years in patients with allodynia vs. 12 years in patients without allodynia-

Table 2). The difference, however, was not statistically significant, possibly due to the small number of patients.

Comments

This is the first report on the occurrence of cutaneous allodynia in cluster headache patients. Our results show that cutaneous allodynia may occur not only in migraine but also in other primary headaches.

Allodynia in migraine may be related to sensitization of neurons in the trigeminal nucleus caudalis (TNC) triggered by trigeminal firing.^{2,5} The trigeminovascular system is activated in both migraine and cluster headache.⁷ This provides a theoretical basis for the assumption that neurons in the trigeminovascular system are sensitized in patients with cluster headache, leading to cutaneous allodynia, as well as to head pain. The exact location and type of neurons or receptors (or both) that are sensitized in CH patients, leading to cutaneous allodynia, is currently unknown. The results of allodynia testing in this study may have been affected by the preventive medications that the majority of patients were on. However, the exact effect of preventive medications on the occurrence of cutaneous allodynia in CH, as well as in migraine, is still to be determined.

In this study there was a trend for allodynia to be more prevalent in patients with CCH (2/3, 66.6%) as opposed to ECH (2/7 28.6%). When correlating disease duration with the occurrence of allodynia, we found that in patients with allodynia median disease duration was longer than in patients without allodynia (22 vs. 12 years). Since patients with CCH have in average longer disease duration than those with ECH, these two results (Tables 1 and 2) are consistent. The allodynia in cluster headache may therefore result from a long-lasting time-

dependent process of neuronal or receptor sensitization. This has been suggested in a model of spinothalamic tract neuronal sensitization after the injection of capsaicin.¹ Alternatively, the development of allodynia may contribute to the transition from ECH to CCH or both processes may share a common cause.

In summary, in this study we show that sensitization of sensory neurons may occur in cluster headache and is not specific to migraine.

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TABLE 1: Occurrence of brush allodynia in the different patient groups

Allodynia	Episodic CH (in acute attack)	Episodic CH (not in acute attack)	Chronic CH (not in attack)	Total
Yes	1	1	2	4
No	1	4	1	6
Total	2	5	3	10

TABLE 2: Disease duration in patients with and without brush allodynia

Allodynia	Disease Duration (years)		
	Minimum	Median	Maximum
Yes	1.5	22	38
No	6	12	24