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Correspondence and Clinical Notes

Post-Traumatic Cluster Headache: From the Periphery to the Central Nervous System?

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A correlation between head trauma and cluster headache is believed to exist. We report a case of post-traumatic episodic cluster headache that fulfills the criteria of the International Classification of Headache Disorders, 2nd edition. The distinctive features of this case are: a close temporal relation between head trauma and headache onset; pain ipsilateral to the side of trauma; mild severity of trauma; episodic course well-responsive to low doses of verapamil. Given the close temporal relation between the 2 events, multiple hypotheses can be advanced about a possible role of head trauma in the pathogenesis of cluster headache.

Key words: cluster headache, mild head trauma, post-traumatic cluster headache

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INTRODUCTION

Cluster headache (CH) is a primary headache currently classified among trigeminal autonomic cephalgias.[1]

Some evidence indicates that a major role in CH pathogenesis is played by the hypothalamus,[2,3] which is involved in pain modulation, controls the autonomic parasympathetic pathway, and also regulates biological rhythms, including hormones secretion.[3] Secondary CH due to organic diseases has been reported in the literature.[4] The only CH case secondary to head trauma that fulfills the 1988 International Headache Society (IHS) criteria[5] was described by Turkewitz et al, but the follow-up was too short for any conclusive evidence on the headache's temporal pattern.[6] Other authors reported CH onset following head trauma, but in these cases the headache did not fulfill the 1988 IHS criteria for post-traumatic headache.[7-9] In the International Classification of Headache Disorders, 2nd edition (ICHD-II), post-traumatic CH is coded to group 5 ("Headache attributed to head and/or neck trauma"). It is a very rare entity, with a rate of occurrence comparable to that of other secondary CH forms.[4]

We report a case of chronic post-traumatic CH with an episodic temporal pattern and a 19-year follow-up. Possible relations between CH and head trauma are discussed.

CASE REPORT

The patient, a 48-year-old woman with no family history of migraine or CH, had never complained of headache before. Except for mild mitral valve prolapse, the patient reported nothing significant in her past medical history. Around 9 PM August 28, 1989, the patient suffered a mild head injury (HI) at home: she accidentally banged her head against an iron ladder and remained in a state of dazed consciousness for about 2 hours. At 3 AM in the night, she felt an excruciating "stabbing" pain radiating from the

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injured right parietal region down to the right retro-orbital, temporal and zygomatic regions. The pain lasted for about 1 hour untreated and was accompanied by ipsilateral lacrimation, conjunctival injection, rhinorrhea and ptosis, but was not associated with psychomotor agitation. At 8 AM the next morning, the patient had a new headache attack identical to the previous one. Based on normal results from an urgent brain CT scan, the neurologist made a diagnosis of CH and recommended preventive therapy with verapamil 80-mg tid during active periods. From then on, the patient reported 2 active periods in a year, the first in early January and the second in early September. Both periods lasted 40 days on average. The attacks recurred regularly at 3 AM in the night and at 8 AM in the morning. They always started in the right parietal region and were never accompanied by restlessness. No other headache subtypes were present during or between CH periods. In 1991, 2 years after CH onset, the patient sought treatment at our Headache Center. A gadolinium-enhanced brain magnetic resonance imaging (MRI) proved negative. We did not perform scans of the pituitary region or angio-MRI of the intracranial arteries to exclude rather frequent causes of secondary CH, eg, pituitary adenoma or carotid dissection,[10,11] because the patient did not show atypical headache features and presented a stable clinical picture at 2 years from onset.

We made a diagnosis of episodic CH (coded to 3.1.2 in the 1988 IHS Classification and to 3.1.1 in ICHD-II) and of chronic post-traumatic headache attributed to mild HI (coded to 5.2.2 in the 1988 IHS classification and in ICHD-II), confirmed the therapy with 80-mg verapamil tid, and prescribed acute treatment with sumatriptan 20-mg nasal spray, which proved effective within 15 minutes. Oxygen therapy 100% at 7 L/min for 15 minutes was also effective after a few minutes.

At 19 years' follow-up, the headache was still showing the same features as at onset.

DISCUSSION

In this paper we report a case of secondary CH that developed 6 hours after mild head trauma. New-onset headache following minor HI is not a rare event.[12] It frequently has the same features as tension-type headache, occurs daily, and can be associated with a "litigation/compensation syndrome," which eventually affects the severity and duration of the headache.[13] In our case, however, psychological factors such as litigation/compensation could be excluded because the HI was self-inflicted.

Based on 19 years' follow-up, the most important clinical aspects in our patient was the absence of atypical CH features. Unlike our case, those reported by Reik and Mathew[7,14] could not be classified as post-traumatic CH because of the long intervals between HI and CH onset, the temporal pattern tended to be chronic from the beginning and the patients did not respond well to therapy. In the case reported by Turkewitz[6] the headache had a chronic course and responded well to lithium and valproic acid, but the 1-year follow-up was too short for any conclusive evidence. Like in previous reports, in our patient, too, pain was ipsilateral to HI.[7]

The pathogenetic mechanisms of post-traumatic chronic headache[1] are not entirely clear yet. Some authors have suggested a neuropathic etiology and assigned a causative role to frontal lobe dysfunction, which is frequently involved in head trauma.[15] A range of hypotheses have been advanced for the role of head trauma in post-traumatic CH pathogenesis. One of these hypotheses is peripheral pathogenesis of this type of headache,[6,7] secondary to abnormal changes in the trigeminal pathway due to direct damage in the site of head trauma and to modulation of some central pain pathways. It has also been suggested that the pathogenetic mechanisms of secondary CH may differ from those of primary CH,[16] which is currently considered a central nervous system (CNS)-originated headache where the trigeminal-autonomic reflex does not seem to be absolutely necessary for the expression of headache attacks.[17]

We believe that head trauma may stimulate nociceptive sensory afferents by causing some mechanical damage locally and by provoking the kind of biochemical modifications described by Hayes and Packard for mild head trauma. These modifications include: transiently increased levels of certain neurotransmitters (glutamate, serotonin), abnormal production of endogenous opiate peptides, and abnormal release of local neurotransmitters (CGRP, substance

P, VIP).[18,19] As a result, the neurovascular system would reorganize and activate itself, eventually precipitating CH attacks. What is more difficult to explain is how mild brain damage may act on central structures such as the hypothalamus, which is likely involved in determining the typical temporal pattern of CH.[17] A possible hypothesis is that patients predisposed to develop CH may have intrinsic hypothalamic dysfunction.[20] Periodically, this dysfunction could predispose the hypothalamus to greater or lesser vulnerability to exogenous factors – such as head trauma, surgery for removal of a brain lesion, or a CNS disorder – thus precipitating headache through peripheral activation in patients who are certain to develop CH at some point in their lifetime. It would then be useful to find a biochemical marker reflecting variations in this "hypothalamic susceptibility." In conclusion, we cannot exclude a purely coincidental relation between HI and CH onset, but the hypothesis of a peripheral genesis of CH appears reasonable. In predisposed subjects, head trauma may eventually trigger the trigeminal-vascular system, activating also CNS mechanisms that are normally involved in CH pathogenesis.

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