Case Report Life Conflux

A Unique Case of Migraine Without Aura Triggered by Palpation of V2-innervated Chronic Facial Folliculitis

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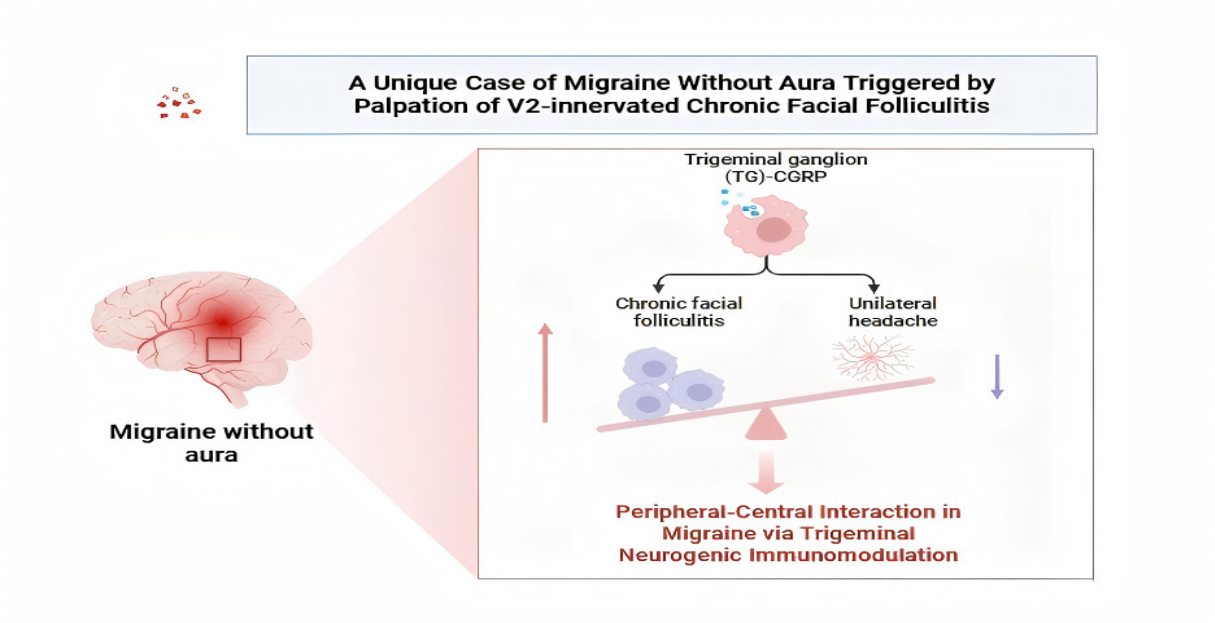
Graphical Abstract



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A Unique Case of Migraine Without Aura Triggered by Palpation of V2-innervated Chronic Facial Folliculitis

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Abstract

Accumulating research suggests that peripheral pathological changes participate in the pathogenesis of migraine, and supporting evidence has been reported in many clinical cases. However, few cases have directly demonstrated the bidirectional interaction between migraine and peripheral pathological lesions. Here, we present a case of severe migraine without aura (MwoA) whose attacks were elicited by palpation of chronic folliculitis lesions around the unilateral nasal ala.This unique case reveals a distinct peripheral-central interaction in migraine, which may be mediated by neurogenic immunomodulation of the trigeminal system.

Keywords: Facial folliculitis; Migraine without aura; Trigeminal nerve; Neurogenic immunomodulation; Peripheral trigger; CGRP

Introduction

It has long been established that migraine patients frequently exhibit tenderness in multiple pericranial regions, including the shoulder, neck, sternocleidomastoid muscle, anterior temporal area and occipital insertion sites. A large proportion of these tender regions act as classic trigger points that can induce migraine attacks, which strongly suggests that peripheral pathological mechanisms play a non-negligible role in the onset and recurrence of migraine [1]. As a common otogenic inflammatory disease, petrous apicitis is often accompanied by headache as a prominent clinical manifestation, and several clinical case reports have pointed out that the headache phenotype induced by petrous apicitis is highly consistent with the characteristics of migraine in some patients[2-3]. Recently, Mancini et al. reported a rare case where a patient with petrous apicitis developed de novo migraine without aura (MwoA), and the migraine attacks achieved sustained remission after the patient received mastoidectomy combined with a long-course antibiotic therapy targeting the primary petrous apicitis [4]. This finding further confirms that inflammatory lesions of the petrous apex can cause pathological changes in the trigeminal nerve fibers innervating the area, which in turn initiates and maintains the recurrent attacks of migraine.

On the basis of previous research, we report a clinical case of severe MwoA triggered by palpation of chronic facial folliculitis around the unilateral nasal ala, a peripheral lesion that was refractory to conventional antibiotic therapy but completely resolved after effective migraine prophylactic treatment. This case not only provides new clinical evidence for the peripheral trigger mechanism of migraine, but also for the first time reveals that the central neural modulation of migraine can in turn regulate the progression of peripheral inflammatory lesions. It further enriches the understanding of the peripheral-central interaction mechanism in migraine and provides a novel perspective for exploring the cross-regulation between the trigeminal nervous system and peripheral immune inflammation.

Case Presentation

A 45-year-old male presented to our department with an 8-month history of severe headaches, which were solely induced by facial contact activities such as face washing and shaving that involved the facial folliculitis lesions around his left nasal ala. Each episode of contact or palpation of this affected area was capable of precipitating an acute severe headache attack. Preceding the onset of the severe headache, an abrupt, transient sharp stabbing pain would radiate instantaneously from the left nasal ala to the surrounding regions of

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the left face. The headache was strictly unilateral on the left side, localizing to the left frontal, temporal and parietal lobes, and occasionally extending to the entire left cerebral hemisphere. Characteristically, the headache manifested as throbbing and pulsating pain, with occasional stabbing sensations, and had an average intensity of 8 to 10 on a 10-point numerical rating scale. Associated symptoms included nausea (with or without vomiting), photophobia, phonophobia and irritability. The headache was exacerbated by head movement, ambulation and physical exertion, while mild relief was achieved with recumbency and rest. Each headache attack typically persisted for half a day or resolved spontaneously following an overnight sleep.

On admission to our department, neurological and physical examinations yielded unremarkable findings, and brain magnetic resonance imaging (MRI) scans were normal. Routine blood tests, as well as additional investigations including erythrocyte sedimentation rate and screening for sexually transmitted diseases, all fell within normal reference ranges. Psychological assessments for anxiety and depression were performed during a headache-free interval, revealing a Hamilton Anxiety Rating Scale (HAMA) score of 10 (a score ≥14 indicates mild anxiety) and a Hamilton Depression Rating Scale (HAM-D) score of 8 (a score >7 indicates mild depression). Dermatological examination of the skin around the left nasal ala identified small pustular lesions and several keloids with varying degrees of pigmentation, which the patient reported were sequelae of previous pustular lesions (Figure 1). Based on the clinical features of the headache and the normal results of all auxiliary examinations except for the local cutaneous findings, a diagnosis of migraine was made. The headache attacks conformed to the diagnostic criteria for migraine without aura (MwoA) as defined by the International Classification of Headache Disorders, 3rd edition (ICHD-3), with the only exception being the specific peripheral trigger factor.

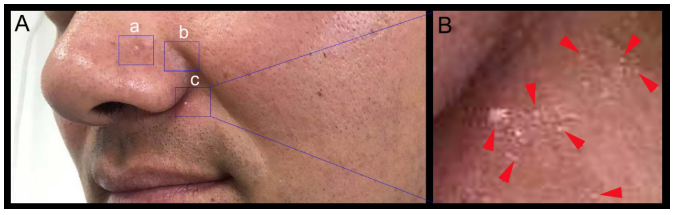

Figure 1. Clinical manifestations of chronic facial folliculitis adjacent to the left nosewing. The skin over the left nosewing region exhibited small purulent papules (white arrows) and multiple keloidal lesions with varying degrees of pigmentation (black arrows), which the patient described as sequelae of prior purulent folliculitis. These lesions were restricted to the cutaneous area innervated by the second division (V2) of the trigeminal nerve and served as the sole trigger for migraine without aura (MwoA) attacks in this patient.

The patient was initiated on prophylactic treatment with sodium valproate (500 mg twice daily) and discontinued all topical antibiotic ointments for 2 months. During the first week of treatment, palpation of the pustular lesions still triggered migraine attacks, but pain intensity and duration were markedly reduced. One week after starting sodium valproate, the pustular lesions around the left nasal ala resolved completely, and palpation or contact stimuli such as face washing no longer induced headaches. During the subsequent 2 months of treatment and 6-month follow-up, the patient remained free of migraine attacks and recurrent pustular folliculitis.

Ten months after initial sodium valproate initiation (and 8 months after drug discontinuation), pustular lesions relapsed around the left nasal ala, and their palpation again triggered migraine attacks identical to the original presentation. Each headache typically lasted 12 hours or resolved overnight after sleep. On one occasion, topical lidocaine hydrochloride gel applied to the pustules reduced headache intensity after attack onset; repeated application prevented palpation-triggered headaches, but therapy was stopped due to its short duration of action (only 2-6 hours). The patient was restarted on sodium valproate at 500 mg twice daily. Five days after resumption, pustular lesions resolved fully, and no headaches were triggered by skin palpation or face washing. The patient has remained on continuous sodium valproate with sustained remission up to manuscript submission.

Written informed consent was obtained from the patient for the publication of this case report, and all identifying information of the patient has been anonymized to protect privacy.

Discussion

Migraine attacks can be triggered by multiple factors via trigeminal nerve (TN) or non-trigeminal pathways [5-7]. Although trigeminal nerve block is effective for migraine, direct TN stimulation as an independent trigger of migraine attacks has rarely been reported. In this case, severe migraine without aura was exclusively elicited by palpation of pustular folliculitis in the V2 distribution in a patient with no prior headache history, providing direct clinical evidence that peripheral TN stimulation can initiate migraine under sensitized conditions. Psychiatric factors were excluded by normal anxiety and depression scores, and contralateral symmetrical skin palpation did not induce headaches. Topical lidocaine hydrochloride gel transiently blocked palpation-induced headaches, supporting the key role of peripheral trigeminal terminal activation in migraine attack initiation [8].

Recent studies have demonstrated microstructural changes and myelin sheath disruption in the TN of migraine patients [9], suggesting that migraine may induce functional and structural abnormalities in the TN [10] . Notably, our case is the first to show that antibiotic-refractory chronic facial folliculitis in the V2 territory completely resolved after migraine prophylaxis, indicating that central migraine-related neural activity can regulate peripheral cutaneous immune responses through the trigeminal nerve. The trigeminal nerve acts as a critical bridge for neuro-immune crosstalk, and its neuropeptides mediate peripheral immune regulation and inflammation [11-12]

Folliculitis is mainly caused by Staphylococcus aureus and Streptococcus pyogenes [13]. Bacterial toxins activate nociceptors, followed by calcitonin gene-related peptide (CGRP) release, which inhibits neutrophil phagocytosis and prolongs infection [14].Meanwhile, CGRP from the trigeminal ganglion (TG) maintains peripheral and central sensitization, increasing migraine susceptibility [15]. TG-derived CGRP also modulates macrophage polarization and exerts local immunomodulatory effects [12]. We therefore propose a vicious cycle: nasal ala folliculitis activates TG neurons and increases CGRP release, leading to trigeminal and central sensitization that enables palpation to trigger migraine. Concurrently, CGRP suppresses

local antibacterial immunity, worsening chronic infection. This bidirectional interaction between peripheral inflammation and central migraine pathogenesis highlights a novel peripheral-central mechanism mediated by CGRP-related neurogenic immunomodulation.

Conclusion

This case observation demonstrates a bidirectional regulatory relationship between migraine attacks and chronic cutaneous infection in the trigeminal nerve (TN) innervated facial region. Moreover, this peripheral-central interaction in migraine pathogenesis is likely mediated by calcitonin gene-related peptide (CGRP) secreted from trigeminal ganglion (TG) neurons.

Abbreviations

Migraine Without Aura: MwoA; Second branch of trigeminal nerve: V2; Calcitonin Gene-Related Peptide: CGRP; Magnetic Resonance Imaging: MRI; Hamilton Anxiety Rating Scale: HAMA; Hamilton Depression Rating Scale: HAM-D; International Classification of Headache Disorders, 3rd edition: ICHD-3; Trigeminal Nerve: TN; Trigeminal Ganglion: TG.

Author Contributions

Conception YW, DX. Acquisition of data YW, DX. Data interpretation YW, DX. Manuscript draft DX. Revision for intellectual content YW. All Authors read and approved the manuscript.

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Ethics Approval and Consent to Participate

Not Applicable.

Competing Interests

The authors declare that they have no existing or potential commercial or financial relationships that could create a conflict of interest at the time of conducting this study.

Data Availability

All data needed to evaluate the conclusions in the paper are present in the paper or the Supplementary Materials. Additional data related to this paper may be requested from the authors.

References

[1] DO T P, HELDARSKARD G F, KOLDING L T, HVEDSTRUP J, SCHYTZ H W. Myofascial trigger points in migraine and tension-type headache [J]. The journal of headache and pain, 2018, 19(1): 84.http://doi.org/10.1186/s10194-018-0913-8.

[2] PLODPAI Y, HIRUNPAT S, KIDDEE W. Gradenigo's syndrome secondary to chronic otitis media on a background of previous radical mastoidectomy: a case report [J]. Journal of medical case reports, 2014, 8: 217.http://doi.org/10.1186/1752-1947-8-217.

[3] ISERSON K V. The ethics of emergency medicine [J]. The Journal of emergency medicine, 1985, 3(2): 161-2.http://doi.org/10.1016/0736-4679(85)90050-2.

[4] MANCINI A J, GLASSMAN R D, CHANG Y M, BURSTEIN R, ASHINA S. Headache in Petrous Apicitis: A Case Report of Chronic Migraine-like Headache Due to Peripheral Pathology [J]. Headache, 2019, 59(10): 1821-6.http://doi.org/10.1111/head.13643.

[5] TAI M S, YET S X E, LIM T C, POW Z Y, GOH C B. Geographical Differences in Trigger Factors of Tension-Type Headaches and Migraines [J]. Current pain and headache reports, 2019, 23(2): 12.http://doi.org/10.1007/s11916-019-0760-6.

[6] SILVA-NéTO R P, PERES M F, VALENçA M M. Odorant substances that trigger headaches in migraine patients [J]. Cephalalgia : an international journal of headache, 2014, 34(1): 14-21.http://doi.org/10.1177/0333102413495969.

[7] SILVA-NéTO R P, RODRIGUES Â B, CAVALCANTE D C, FERREIRA P H, NASI E P, SOUSA K M, et al. May headache triggered by odors be regarded as a differentiating factor between migraine and other primary headaches? [J]. Cephalalgia : an international journal of headache, 2017, 37(1): 20-8.http://doi.org/10.1177/0333102416636098.

[8] RAM S, KUMAR S K, CLARK G T. Using oral medications, infusions and injections for differential diagnosis of orofacial pain [J]. Journal of the California Dental Association, 2006, 34(8): 645-54

[9] GUYURON B, YOHANNES E, MILLER R, CHIM H, REED D, CHANCE M R. Electron microscopic and proteomic comparison of terminal branches of the trigeminal nerve in patients with and without migraine headaches [J]. Plastic and reconstructive surgery, 2014, 134(5): 796e-805e. http://doi.org/10.1097/prs.0000000000000696.

[10] ZIEGELER C, MAY A. Facial presentations of migraine, TACs, and other paroxysmal facial pain syndromes [J]. Neurology, 2019, 93(12): e1138-e47.http://doi.org/10.1212/wnl.0000000000008124.

[11] BARON R, BINDER A, WASNER G. Neuropathic pain: diagnosis, pathophysiological mechanisms, and treatment [J]. The Lancet Neurology, 2010, 9(8): 807-19.http://doi.

org/10.1016/s1474-4422(10)70143-5.
[12] TAO J, WANG X, XU J. Expression of CGRP in the Trigeminal Ganglion and Its Effect on the Polarization of Macrophages in Rats with Temporomandibular Arthritis [J]. Cellular and molecular neurobiology, 2024, 44(1): 22.http://doi.org/10.1007/s10571-024-01456-7.
[13] JACOBS M R, JONES R N, GIORDANO P A. Oral beta-lactams applied to uncomplicated infections of skin and skin structures [J]. Diagnostic microbiology and infectious disease, 2007, 57(3 Suppl): 55s-65s.http://doi.org/10.1016/j.diagmicrobio.2006.11.020.
[14] PINHO-RIBEIRO F A, BADDAL B, HAARSMA R, O'SEAGHDHA M, YANG N J, BLAKE K J, et al. Blocking Neuronal Signaling to Immune Cells Treats Streptococcal Invasive Infection [J]. Cell, 2018, 173(5): 1083-97.e22.http://doi.org/10.1016/j.cell.2018.04.006.
[15] IYENGAR S, JOHNSON K W, OSSIPOV M H, AURORA S K. CGRP and the Trigeminal System in Migraine [J]. Headache, 2019, 59(5): 659-81.http://doi.org/10.1111/head.13529.