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Contact lens-associated ocular allodynia in migraine: A case report highlighting peripheral sensory input in central sensitization

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Abstract

Allodynia, defined as the perception of pain from stimuli that are normally non-painful, is a hallmark of central sensitization in migraine pathophysiology. While cutaneous allodynia is well-documented, the clinical significance of ocular allodynia, particularly in association with contact lens wear, remains underreported. Here, the case of a 42-year-old female with a long-standing history of episodic migraine, characterized by unilateral throbbing pain (VAS 7-8/10), nausea, and photo-phonophobia, typically triggered by sleep deprivation and menstruation was presented. The patient, a long-term user of soft contact lenses for -3.0 diopter myopia, reported a unique temporal pattern of allodynia emerging over the last two years. She described a localized hypersensitivity on the symptomatic side, occurring in the prodromal phase several hours before the headache onset. This sensation rendered contact lens wear intolerable during the attack, independent of photophobia. Ophthalmological evaluations, including intraocular pressure and fundus examinations, were unremarkable. Despite a history of chronic migraine managed with amitriptyline and onabotulinumtoxinA, her current episodic attacks reveal a distinct ocular-tactile sensitivity. To our knowledge, this is the first reported case of contact lens-related ocular allodynia in migraine. This case emphasizes the necessity of screening for ocular allodynia in contact lens users, suggesting that lens intolerance may serve as a clinical marker for evolving central sensitization in migraineurs. Further research is warranted to elucidate the trigeminovascular mechanisms underlying this specific allodynic manifestation.

Keywords: Migraine disorders, contact lenses, central sensitization, ocular allodynia, trigeminal nerve.

INTRODUCTION

Migraine is a common disabling primary headache disorder characterized by recurrent episodes of moderate to severe headache, often unilateral and throbbing, accompanied by nausea, photophobia, and phonophobia.[1] Sensory disturbances beyond headache, including cutaneous allodynia, are frequently reported and reflect altered pain processing within the central nervous system.[2] Allodynia is defined as pain due to a stimulus that does not normally provoke pain (e.g., light touch) and is distinct from hyperalgesia, which is an exaggerated response to painful stimuli.[3]

In migraine, allodynia occurs in approximately 40–70% of attacks and has been associated with higher attack severity and frequency, female sex, and central sensitization phenomena (4). The pathophysiology involves peripheral and central sensitization in trigemino-vascular and ascending nociceptive pathways, with dysfunction of brainstem and thalamocortical circuits mediating abnormal pain perception. Importantly, cutaneous allodynia is considered a clinical marker of migraine progression and therapeutic response, including potential reduced efficacy of acute treatments when present.[4,5]

Although sensory hypersensitivity to stimuli such as touch, combing hair, or wearing tight clothing or glasses has been described in the context of migraine allodynia, the specific interaction between contact lens wear and allodynia is seldom addressed in the literature. A small number of case observations suggest that discomfort from poorly fitted lenses may

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trigger migraine attacks or augment sensory hypersensitivity in predisposed individuals.[6] Herein, a case of unilateral migraine-associated allodynia in which soft contact lens wear precipitated or exacerbated ipsilateral sensory hypersensitivity prior to headache onset, despite normal ophthalmological findings is reported.

CASE REPORT

A 42-year-old female presented with a long-standing clinical history of episodic migraine since her early 30s. Her attacks were typically characterized by unilateral, throbbing pain, reaching a severity of 7-8 on the Visual Analogue Scale (VAS). The primary triggers were identified as the onset of the menstrual cycle and sleep deprivation. Accompanying symptoms included nausea, photophobia, and phonophobia.

In the last two years, the patient reported a shift in her clinical phenotype, marked by the emergence of localized allodynia preceding the headache phase by several hours. Specifically, she described a marked hypersensitivity on the ipsilateral side of the headache, where even innocuous tactile stimuli, most notably the insertion and wear of a soft contact lens, provoked significant discomfort and appeared to exacerbate the intensity of the subsequent migraine attack.

The patient's ophthalmic history revealed -3.00 D myopia, managed with an alternating regimen of spectacles and soft contact lenses for approximately 22 years. Routine ophthalmological evaluations, including slit-lamp examination, intraocular pressure measurement, and funduscopy, were consistently within normal limits. Notably, the patient denied symptoms of chronic dry eye or ocular surface disease outside the context of her migraine attacks. Her medical history was otherwise unremarkable, with regular menstrual cycles and no systemic comorbidities.

Pharmacological history included a trial of amitriptyline and three courses of onabotulinumtoxinA five years prior for a period of chronic migraine, which successfully transitioned the patient back to an episodic pattern. Currently, the most striking clinical feature is the unique temporal relationship between contact lens wear and allodynia: wearing a soft contact lens during the prodromal phase on the symptomatic side acted as a potent allodynic stimulus. To objectively quantify the severity of these symptoms, the patient was instructed to complete the Allodynia Symptom Checklist-12 (ASC-12). Her self-reported scores consistently fell within the 'severe allodynia' range, further validating the clinical transition from localized ocular discomfort to a more generalized state of central sensitization. Importantly, while the removal of the contact lens did not abort the migraine process, the patient reported a subjective reduction in the intensity of the subsequent headache and a mitigation of allodynic discomfort.

DISCUSSION

Allodynia in migraine is a well-recognized clinical phenomenon reflecting the central sensitization of pain pathways, wherein normally non-noxious stimuli evoke a painful response.[4,7] This state of hypersensitivity results from the increased excitability of neurons within the trigeminal nucleus caudalis and the posterior thalamic nuclei, leading to dysfunctional sensory processing. Pioneering studies by Burstein et al. have demonstrated that cutaneous allodynia evolves during an attack through the sequential recruitment of spinal and supraspinal nociceptive neurons, marking the transition from peripheral to central sensitization.[8] Clinically, allodynia is reported in a significant portion of the migraine population and serves as a potent predictor of attack frequency, increased disability, and migraine chronification.[9,10] While manifestations like discomfort during hair combing or wearing spectacles are frequently documented, ocular-specific allodynia remains an under-investigated domain. Our case presents a distinct clinical scenario where the mechanical presence of a soft contact lens acts as a modulating sensory input during the prodromal and ictal phases.

Cutaneous allodynia is reported in approximately two-thirds of patients with migraine and is often assessed via the Allodynia Symptom Checklist (ASC-12).[11] While common manifestations include pain during hair combing, shaving, or wearing tight clothing, ocular-specific allodynia remains an under-investigated domain. Current literature on contact lens wear in migraineurs is predominantly limited to primary ocular surface issues, such as dry eye syndrome or improper lens fit (base curve mismatch).[6] Our case, however, presents a distinct clinical scenario where the mechanical presence of a soft contact lens acts as a focal stimulus for allodynia in the prodromal and ictal phases, despite normal ophthalmological findings and the absence of ocular surface disease.

The cornea is among the most densely innervated tissues in the body, receiving sensory supply primarily from the ophthalmic branch of the trigeminal nerve (V1).[2,4,12] It can be hypothesized that in a sensitized state, the sub-threshold mechanical stimulation provided by a contact lens functions as a secondary peripheral input that amplifies pre-existing trigeminal hypersensitivity. Importantly, while contact lens removal did not abort the migraine process in our patient, it was associated with a reduction in the severity of the allodynic sensation and the subsequent headache. This suggests that lens-related tactile stimulation acts as an exacerbating factor acting upon an already sensitized system, rather than a primary precipitating trigger. Such observations align with the broader understanding of migraine as a disorder of maladaptive sensory processing. This suggests that lens-related tactile input modulates the intensity of the attack through additive peripheral sensory input in the context of established central sensitization. Toriyama et al. demonstrated that a significant subgroup of migraineurs exhibits interictal widespread pressure hyperalgesia, supporting the classification of migraine as a central sensitivity syndrome.[13] In our case, the patient's localized ocular hypersensitivity to contact lens wear further exemplifies this generalized sensory dysregulation, suggesting that the ocular surface may also become a site for the clinical manifestation of such systemic central sensitization. The unique temporal relationship observed, where allodynia preceded the headache onset, indicates that ocular tactile sensitivity might serve as an early clinical marker of the migraine prodrome. Finally, the patient's severe allodynia score on the ASC-12[11] validates that this ocular sensitivity is a localized component of a broader, systemic state of neuronal hyperexcitability.

This case report underscores that in migraineurs with established central sensitization, contact lens wear may serve as a provocative sensory stimulus that exacerbates allodynic pain. Our findings highlight that ocular allodynia can occur independently of primary ocular pathology, representing a specific manifestation of trigeminal hypersensitivity. Clinicians should systematically assess for allodynia using standardized tools like the ASC-12 and actively explore potential modulating factors, including contact lens intolerance, even in patients with normal ophthalmologic examinations. Recognizing these specific sensory exacerbating factors is crucial, as it may influence the timing of acute pharmacological interventions (e.g., administering triptans at the earliest sign of ocular discomfort) and could serve as a phenotypic marker for prioritizing preventive strategies. Future large-scale studies are warranted to investigate the prevalence of ocular sensory aggravating factors and to further elucidate the underlying neurobiological mechanisms of lens-induced allodynia in migraine.

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DISCLOSURE

Ethics: The patient has given her written consent to this case report.

Data availability: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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