

Transient glabellar and diffuse facial erythema during migraine attacks: A rare autonomic manifestation

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Abstract

Migraine is a primary headache disorder frequently accompanied by cranial autonomic symptoms, and recent reports have described cutaneous phenomena such as the “red forehead dot” and glabellar erythema, suggesting a link with localized autonomic dysfunction. We present two male patients with migraine who developed transient glabellar erythema during attacks. In the first case, unilateral erythema appeared during the peak of the migraine attack and resolved within 12–24 hours, whereas in the second case, symmetrical diffuse erythema involving both sides of the glabella were observed prior to headache onset and gradually regressed as the attack subsided. In both patients, neurological examination, laboratory tests, and cranial imaging were normal during the interictal period. These findings suggest that glabellar erythema may represent a rare manifestation of migraine-related autonomic dysfunction with variable distribution patterns. Recognition of this phenomenon may help prevent misdiagnosis and unnecessary diagnostic procedures.

Keywords: Red forehead dot, migraine, autonomic dysfunction, trigeminovascular system.

INTRODUCTION

According to the ICHD-3, migraine is a primary headache disorder characterized by recurrent attacks of headache lasting 4–72 hours. The headache is typically unilateral, pulsating in quality, of moderate to severe intensity, and aggravated by routine physical activity. It is commonly associated with nausea and/or vomiting, as well as photophobia and phonophobia.¹ Cranial autonomic symptoms are common in migraine, occurring in up to 56–70% of patients, and are associated with more severe attacks and a higher risk of chronification.² In recent years, migraine-associated cutaneous manifestations have gained attention, although they remain rarely reported. The “red forehead dot” phenomenon, as well as cases with ecchymosis or erythematous lesions in the forehead and periorbital regions, suggest that trigeminovascular activation and neurogenic inflammation may also manifest in the skin.^{3,5} Additional rare entities, such as red ear syndrome and facial erythromelalgia,

further support this association.^{6,7} Migraine pathophysiology involves trigeminovascular activation with neuropeptide release (including CGRP), vasodilation, and plasma extravasation, which may explain these cutaneous findings.⁸ In this context, we present two cases of migraine with distinct patterns of forehead and facial erythema, expanding the spectrum of migraine-associated cutaneous manifestations.

CASE REPORT

Patient 1

A 35-year-old male presented with a 10-year history of recurrent headaches occurring 4–6 times per month. The attacks were predominantly unilateral and localized to the frontal and glabellar regions. The headaches were accompanied by photophobia, phonophobia, and nausea. The patient had no known comorbidities. Routine blood tests and cranial MRI were unremarkable. During severe migraine attacks, unilateral erythema confined to one side of the glabella and

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Date of Submission: 31 March 2026; Date of Acceptance: 9 April 2026

<https://doi.org/10.54029/2026axh>



Figure 1. **a:** Asymmetric erythema in the forehead region during a migraine attack; **b:** Following nonsteroidal anti-inflammatory treatment.

corresponding to the side of the headache was observed (Figure 1a). The erythematous area was well-demarcated, blanchable on pressure, and not associated with increased local temperature or edema. The erythema typically appeared at peak pain intensity and was reported in approximately 30–40% of attacks. Following treatment with nonsteroidal anti-inflammatory drugs, both the headache and the erythema resolved completely within 12–24 hours (Figure 1b).

Patient 2

A 46-year-old male presented with recurrent migraine-like headaches occurring four or

more times per month, aggravated by physical activity and accompanied by photophobia and phonophobia. The headache was unilateral in nature. In most attacks, a transient erythematous lesion involving both sides of the frontal region, including the glabella, appeared prior to headache onset (Figure 2) and resolved completely as the attack subsided, without residual skin changes. The erythematous areas were blanchable on pressure, had indistinct borders, and were not associated with increased local temperature or edema. The lesions were non-elevated and not accompanied by pain or pruritus. Neurological examination, laboratory tests, and cranial imaging were normal during the interictal period.



Figure 2. Transient bilateral erythematous lesion of the glabellar region before migraine attack in Case 2.

Based on the temporal association, the findings were interpreted as a migraine-associated autonomic phenomenon consistent with a red forehead dot.

DISCUSSION

In these two cases, erythema limited to the glabellar and forehead regions during migraine attacks fits within the rarely described spectrum of “cutaneous manifestations of migraine” while exhibiting a distinctive pattern. The presence of unilateral glabellar involvement in one patient and bilateral diffuse forehead/facial erythema in the other, occurring exclusively during attacks, suggests a direct relationship with ictal trigeminovascular activation.⁹ Cranial autonomic symptoms and facial involvement are more common in migraine than previously recognized, occurring in approximately 2–9% of patients; however, cutaneous findings such as non-blanchable erythematous plaques or patches on the forehead remain extremely rare.^{5,10} The strict temporal association of erythema with the ictal phase and its complete resolution during the interictal period support the hypothesis of a transient neurovascular phenomenon.

From a pathophysiological perspective, activation of meningeal afferents during migraine leads to trigeminovascular neurogenic inflammation, characterized by CGRP release, vasodilation, and plasma extravasation. Given that the forehead and facial skin share segmental innervation via the ophthalmic (V1) branch of the trigeminal nerve, it is biologically plausible that these processes manifest as localized or diffuse erythema.⁹ The unilateral glabellar involvement observed in one case parallels migraine phenotypes associated with unilateral cranial autonomic symptoms.¹¹

Clinically, these findings are important for differential diagnosis. Transient facial erythema may mimic dermatological or systemic conditions such as rosacea, vasculitis, or angioedema. However, the absence of persistent erythema, papules, pustules, or telangiectasia, along with normal systemic and imaging findings and resolution with migraine treatment, argues against primary dermatologic disorders and supports a diagnosis of migraine-related transient ictal facial erythema.^{12,13} Recognition of this phenomenon may help avoid unnecessary diagnostic investigations.

These cases highlight that migraine-associated cutaneous manifestations are likely

underrecognized. Clinicians should consider migraine when encountering facial erythema that is temporally related to attacks and resolves interictally. Further studies are needed to clarify its relationship with migraine severity, cranial autonomic features, and chronification risk.²

In conclusion, these two cases suggest that glabellar and diffuse facial erythema occurring during migraine attacks may represent a rare manifestation of migraine-related autonomic dysfunction. This finding may serve as a clinical clue for migraine, particularly when erythema is attack-related and not explained by a dermatologic condition. Awareness of this phenomenon may help avoid misdiagnosis and unnecessary investigations.

DISCLOSURE

Ethics: Written informed consent for publication of the clinical information and accompanying photographs was obtained from both patients.

Financial support: None

Conflict of interest: None

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