J. Pharm. Res. 2026;25(3):206-208

Print ISSN: 0973-7200
Online ISSN: 2454-8405

Journal of Pharmaceutical Research

CASE REPORT

Beyond Herpes: A Case of Oral-Dominant Pemphigus Vulgaris Initially Misdiagnosed as Herpetic Stomatitis

Saijal Singh[1], Manmohan [2], Srenwentu Chakraborty[1*]

[1]*K.D. Dental College & Hospital, Dr. B.R. Ambedkar University, Agra, India*

[2]*K.D. Dental College and Hospital, Atal Bihari Vajpayee Medical University, Lucknow, India*

ARTICLE INFO

Article history:
Received 06-08-2026
Accepted 02-09-2026
Published 24-09-2026

** Corresponding author.*
Srenwentu Chakraborty
srenwentuchakraborty@gmail.com

https://doi.org/10.18579/jopcr/v25.i3.169

ABSTRACT

Pemphigus vulgaris (PV) is a rare, potentially life-threatening autoimmune vesiculobullous disorder that frequently presents first in the oral cavity, often before any skin involvement appears. Because early oral lesions are non-specific, PV is commonly misdiagnosed as herpetic stomatitis, aphthous ulceration, or other vesiculobullous conditions, delaying appropriate immunosuppressive treatment. We report the case of a 26-year-old female who presented with recurrent, painful oral blisters previously diagnosed and treated as herpetic stomatitis by other clinicians, without improvement. Clinical examination revealed flaccid bullae that ruptured to leave raw, eroded surfaces, with a positive perilesional Nikolsky sign confined to the oral mucosa; systemic features of herpetic infection (fever, lymphadenopathy) were absent. Incisional biopsy confirmed suprabasal acantholysis, consistent with pemphigus vulgaris. The patient was managed with systemic corticosteroids (methylprednisolone), intralesional triamcinolone acetonide, and supportive therapy, with significant clinical improvement and reduction in lesion burden. This case highlights the importance of considering pemphigus vulgaris in the differential diagnosis of persistent oral bullous lesions that do not respond to antiviral therapy and underscores the diagnostic value of the Nikolsky sign and biopsy in distinguishing PV from herpetic stomatitis.

Keywords: Pemphigus vulgaris; Oral bullae; Nikolsky sign; Suprabasal acantholysis; Herpetic stomatitis; Misdiagnosis

INTRODUCTION

Pemphigus vulgaris is an autoimmune mucocutaneous disease caused by IgG autoantibodies directed against desmoglein 3 (and, in mucocutaneous forms, desmoglein 1), leading to loss of keratinocyte adhesion and intraepithelial blister formation. The oral cavity is the initial and, in many cases, the only site of involvement for months before cutaneous lesions develop, making dental and oral medicine clinicians frequently the first point of contact for undiagnosed patients.

This is particularly relevant in the North Indian region where this case was managed, where prior work by our group has documented a substantial burden of tobacco and arecanut use, and associated oral mucosal lesions, among school-going adolescents[1, 2]. While tobacco use is not an established aetiological factor in pemphigus vulgaris, the high background prevalence of oral mucosal pathology in this population underscores the broader importance of careful, complete oral mucosal examination across age groups, and of maintaining a wide differential diagnosis rather than defaulting to the most common cause of oral ulceration.

Because oral PV lesions present as painful, shallow, irregular erosions following the rupture of fragile, short-lived bullae, they are frequently mistaken for more common causes of oral ulceration, particularly recurrent herpetic stomatitis, major aphthous ulceration, and erosive lichen planus. This diagnostic overlap can result in inappropriate antiviral therapy and delayed initiation of immunosuppressive treatment, during which the disease may progress to widespread mucocutaneous involvement. We report a case in which an initial diagnosis of herpetic stomatitis was revised to pemphigus vulgaris on the basis of clinical and histopathological findings, with a favourable early response to treatment.

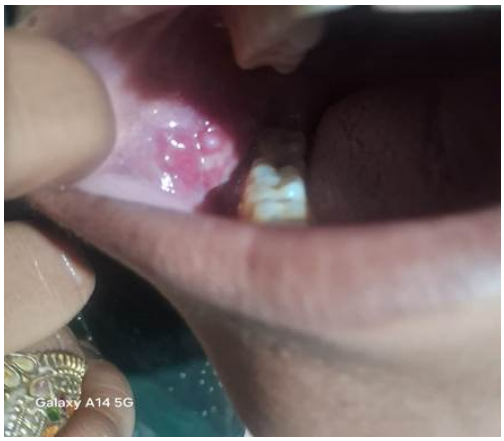
CASE PRESENTATION

A 26-year-old female presented to our clinic with a several-week history of painful blisters within the oral cavity. She had previously been evaluated elsewhere and diagnosed with herpetic stomatitis, for which she had received antiviral therapy without symptomatic improvement.

On examination:

- Multiple flaccid, thin-walled bullae were observed on the oral mucosa, which ruptured easily on minimal manipulation, leaving raw, eroded, erythematous surfaces Fig. 1.
- The Nikolsky sign was positive when elicited perilesionally around the oral bullae, but negative when tested on skin elsewhere on the body.
- No fever was recorded, and no regional or systemic lymphadenopathy was noted.
- No other mucocutaneous, ocular, or genital lesions were identified at presentation.

The absence of systemic features typically associated with primary herpetic infection (fever, lymphadenopathy), combined with the pattern of bullae rupture and a positive perilesional Nikolsky sign, raised suspicion for an immunobullous disorder rather than a viral aetiology.



Investigations

An incisional biopsy was performed from the margin of an active lesion. Histopathological examination demonstrated suprabasal acantholysis with intraepithelial clefting, consistent with pemphigus vulgaris. This finding, together with the clinical presentation, supported a diagnosis of oral-dominant pemphigus vulgaris rather than herpetic stomatitis.

Management

The patient was started on the following regimen:

- Methylprednisolone (Medrol) — systemic corticosteroid therapy
- Triamcinolone acetonide (Kenacort), 4 mg — intralesional injection
- Pantoprazole, 40 mg — gastric mucosal protection during corticosteroid therapy
- Folic acid supplementation — nutritional/mucosal support

On follow-up, the patient demonstrated significant clinical improvement, with a marked reduction in the number and severity of oral bullae and associated erosions. Continued monitoring and staged tapering of corticosteroid therapy, alongside consideration of a steroid-sparing agent, was planned given the chronic, relapsing nature of pemphigus vulgaris.

DISCUSSION

This case illustrates a diagnostically important distinction between pemphigus vulgaris and herpetic stomatitis, two conditions that can appear superficially similar but differ substantially in underlying pathophysiology, natural history, and required treatment.

Herpetic stomatitis typically presents with clusters of small vesicles that rapidly rupture into shallow ulcers and is frequently accompanied by systemic prodromal symptoms such as fever, malaise, and regional lymphadenopathy, particularly in primary infection. The absence of these systemic features in this patient, despite a clinical picture that had been treated as herpetic disease, was an important early clue prompting reconsideration of the diagnosis.

The Nikolsky sign — extension of a blister or induction of epidermal separation with lateral pressure on clinically normal-appearing skin or mucosa adjacent to a lesion — is a classic, though not universally present, clinical finding in pemphigus vulgaris, reflecting the loss of intercellular adhesion (acantholysis) that defines the disease. In this case, the sign was positive specifically around the oral lesions but negative on unaffected skin, a pattern consistent with early, oral-dominant disease that has not yet progressed to generalized cutaneous involvement. A negative Nikolsky sign at distant, unaffected skin sites should not be taken as

evidence against pemphigus vulgaris; the sign is most reliably elicited at or near active lesions.

Biopsy remains essential to confirm the diagnosis. Suprabasal acantholysis with an intact basal cell layer ("row of tombstones" appearance) is the histopathological hallmark of pemphigus vulgaris, distinguishing it from herpetic infection (which shows viral cytopathic changes such as multinucleated giant cells and ballooning degeneration) and from other immunobullous diseases such as mucous membrane pemphigoid (subepithelial rather than intraepithelial split). Where available, direct immunofluorescence demonstrating intercellular IgG and C3 deposition in a characteristic "chicken-wire" pattern would further strengthen diagnostic confidence and is recommended in subsequent evaluation of this patient.

Regarding management, systemic corticosteroids remain the cornerstone of initial pemphigus vulgaris treatment, often combined with intralesional corticosteroids for localized, symptomatic oral lesions, as used in this patient. Current international consensus increasingly favours the early addition of rituximab alongside corticosteroids for moderate-to-severe disease, given evidence of more durable remission and reduced long-term steroid exposure compared with corticosteroid monotherapy. Steroid-sparing immunosuppressants such as azathioprine or mycophenolate mofetil are commonly introduced to reduce cumulative corticosteroid burden in patients requiring longer-term therapy. Supportive measures — gastric protection with a proton pump inhibitor, as used here, along with calcium and vitamin D supplementation during prolonged corticosteroid therapy — are important adjuncts to reduce treatment-related morbidity.

This case reinforces that persistent oral bullous or erosive lesions failing to respond to antiviral therapy, particularly in the absence of systemic infectious features, warrant biopsy and consideration of an immunobullous aetiology rather than continued empirical antiviral treatment. It also adds to a broader pattern observed by our group in this region, where atypical or clinically ambiguous oral mucosal presentations have previously been shown to mask significant underlying systemic disease — as in a prior report of an oral hemorrhagic lesion mimicking melanoma that was ultimately attributed to severe thrombocytopenia and suspected immune thrombocytopenic purpura[3]. Taken together, these cases underscore the importance of histopathological and, where indicated, systemic haematological or immunological work-up for oral mucosal lesions that do not conform neatly to a common, benign clinical picture.

CONCLUSION

Oral pemphigus vulgaris can closely mimic herpetic stomatitis in its early presentation, and misdiagnosis can result in delayed, ineffective treatment. Clinicians should maintain a high index of suspicion for pemphigus vulgaris in patients with recurrent, non-healing oral bullae or erosions that do not respond to antiviral therapy, particularly when systemic infectious features are absent. A positive perilesional Nikolsky sign and biopsy demonstrating suprabasal acantholysis are key diagnostic findings that support prompt initiation of appropriate immunosuppressive therapy, as illustrated in this case.

DECLARATIONS

Informed consent was obtained from the patient for publication of this case report and any accompanying clinical images.

The authors declare no conflict of interest.

References

1. Chakraborty S, Mathuriya D, Singh S, Singh CD. Peer Pressure and Early Adoption of Arecanut and Smokeless Tobacco Among Schoolchildren: A Cross-Sectional Study in Public Health Dentistry. *Asian Pacific Journal of Cancer Nursing*. 2025; :20251201 . Available from: https://doi.org/10.31557/apjcn.2207.20251201
2. Chakraborty S. Prevalence of Tobacco and Arecanut Use and Associated Oral Mucosal Lesions Among Adolescent School Children of Mathura City. *Asian Pacific Journal of Cancer Nursing*. 2025; :111-116 . Available from: https://doi.org/10.31557/apjcn.2116.20251026
3. Kukar S, Kankariya H, Srivastava S, Chakraborty S. Oral Hemorrhagic Lesion Mimicking Melanoma: A Case Report of Severe Thrombocytopenia with Suspected Immune Thrombocytopenic Purpura. *Asian Pacific Journal of Cancer Nursing*. 2026; . Available from: https://doi.org/10.31557/APJCN.2918.20260714