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RECURRING RIDDLE: HERPETIFORM APTHOUS-A CASE STUDY

Oral Pathology

Dr. Sonali Khatri* Senior Lecturer, Department Of Oral And Maxillofacial Pathology And Microbiology, Genesis Dental College, Firozpur, Punjab, India. *Corresponding Author

Dr. Pawan Rebello Post-graduate Student, Sardar Patel Post Graduate Institute Of Dental & Medical Sciences, Lucknow, India.

Dr. Pallavi Khare Post-Graduate Student, Sardar Patel Post Graduate Institute Of Dental & Medical Sciences, Lucknow, India.

ABSTRACT

This case report presents the clinical characteristics, management, and recurrent nature of herpetiform aphthous ulcers (HAU) on the tongue in a 28-year-old female. The patient presented with a two-year history of recurrent, painful oral lesions, described as multiple small, blister-like sores coalescing into larger ulcers, occurring every 2-3 months. Intraoral examination revealed characteristic clustered ulcerations on the lateral aspects of dorsum of the tongue. A diagnosis of HAU was made based on clinical presentation. Management involved topical corticosteroids and chlorhexidine mouthwash, alongside patient education on avoiding trigger factors. Follow-up showed significant improvement and reduced recurrence frequency, emphasizing the importance of accurate diagnosis and patient-centred therapeutic approaches for this challenging oral condition. Further research is needed to fully understand the underlying mechanisms and develop more targeted long-term preventive strategies.

KEYWORDS

Herpetiform Aphthous Ulcers (HAU), Recurrent oral lesions, Tongue ulcerations, Topical corticosteroids

INTRODUCTION

Aphthous ulcers are a common oral mucosal disease characterized by painful, recurrent ulcerations. While most aphthous ulcers are solitary or few in number, the herpetiform subtype is characterized by multiple, small, clustered ulcers that can coalesce to form larger lesions [1]. This case report details the clinical presentation, management, and recurrent nature of aphthous ulcers herpetiform on the tongue and lip in a 28-year-old female. Aphthous ulcers are broadly classified into three main types based on their clinical presentation: minor, major, and herpetiform [2, 3].

- **Minor Aphthous Ulcers (MiRAU):** These are the most common type, accounting for about 80% of all cases [2]. They typically appear as small (less than 1 cm in diameter), oval or round ulcers with a yellowish-white pseudomembrane and an erythematous halo. They usually occur on non-keratinized movable mucosa (e.g., labial and buccal mucosa, floor of the mouth, ventral surface of the tongue) [2]. MiRAU heal spontaneously within 7-14 days without scarring.
- **Major Aphthous Ulcers (MaRAU):** Also known as Sutton's disease or periadenitis mucosa necrotica recurrens, MaRAU are less common but more severe [2]. They are larger (greater than 1 cm in diameter), deeper, and have irregular borders. MaRAU often take several weeks to months to heal and frequently result in scarring. They can occur anywhere in the oral cavity, including keratinized surfaces.
- **Herpetiform Aphthous Ulcers (HAU):** This is the rarest form, characterized by multiple (10-100 or more), small (1-3 mm), pin-point ulcers that often cluster together and can coalesce to form larger, irregularly shaped lesions [2]. Despite their name, they are not caused by the herpes virus [3]. HAU can occur on any oral mucosal surface, including the tongue, and are often intensely painful. They typically heal within 7-10 days, usually without scarring.

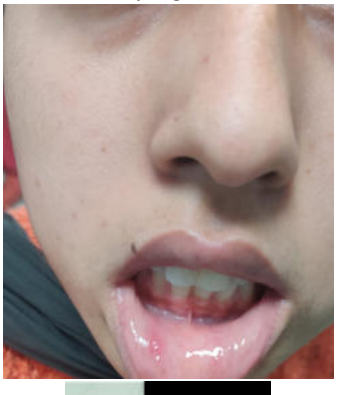
Case Study

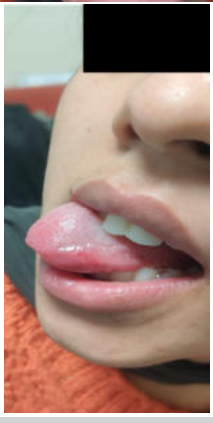
A 28-year-old female presented to the hospital with a chief complaint of recurrent, painful oral lesions in mouth for the past two years. The patient reported that the lesions typically appeared as small, blister-like sores that would rupture and coalesce, forming larger, intensely painful ulcers (Fig. 1,2). These episodes occurred approximately once every 2-3 months, lasting for 7-10 days, and significantly interfered with eating, speaking, and overall quality of life. The patient denied any systemic symptoms such as fever, weight loss, or gastrointestinal disturbances. Her medical history was unremarkable, and she was not on any regular medications. There was no family history of similar oral lesions.

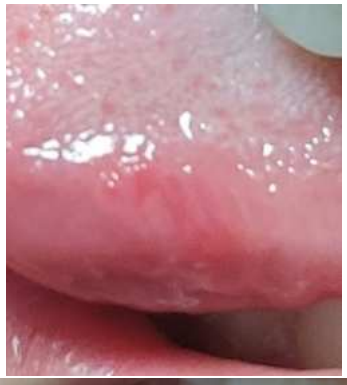
On intraoral examination, multiple small (1-3 mm) ulcerations clustered together were observed on the lateral aspects of the tongue and inner lip. The ulcers were shallow, and surrounded by an erythematous halo (Fig. 3,4). Regional lymph nodes were not palpable.

Based on the clinical presentation of recurrent, multiple, small, clustered ulcers on the tongue, a diagnosis of aphthous ulcers herpetiform was made while in the differentials, Secondary HSV infections, Pemphigus vulgaris and Mucous membrane pemphigoid were considered but ruled out due to lack of any major systemic symptoms and absence of vesicles preceeding the ulcers.

The exact pathophysiology of recurrent aphthous ulcers (RAU) is not fully understood, but it is believed to involve a complex interplay of genetic, immunological, microbiological, and environmental factors, leading to a localized immune dysregulation.







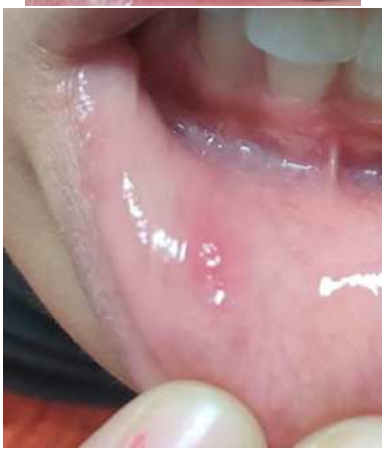


Fig 1 & 2: Patient's presentation with long-standing ulcers on the lip and tongue.
Fig 3: Coalesced ulcers on the tongue exhibiting a red patch.
Fig. 4: An aggregation of tiny ulcers in a close-up view.

The Exact Pathophysiology Is Unknown And Is Attributed To:
Immunological Factors:

- **T-cell Mediated Immune Response:** RAU are considered to be a T-cell mediated inflammatory disorder. An increase in CD4+ T lymphocytes and a decrease in CD8+ T lymphocytes are observed in the peripheral blood of RAU patients during active phases. These T cells are believed to be activated and target oral mucosal epithelial cells, leading to ulceration.
- **Cytokines:** An imbalance in pro-inflammatory (e.g., TNF-α, IL-6, IL-1β) and anti-inflammatory (e.g., IL-10) cytokines is consistently reported. Elevated levels of pro-inflammatory cytokines contribute to the tissue destruction and inflammation characteristic of the ulcers.
- **Autoimmunity:** While not definitively proven, some theories suggest an autoimmune component, where the immune system mistakenly attacks components of the oral mucosa. Antibodies against oral mucosal antigens have been detected in some patients.

Genetic Predisposition:

- A strong familial predisposition is observed, suggesting a genetic component. Certain HLA (Human Leukocyte Antigen) types, particularly HLA-B51 and HLA-DRB1, have been associated with an increased susceptibility to RAU, indicating a genetic influence on immune response.

Microbiological Factors:
While not a primary infectious disease, changes in the oral microbiome have been implicated. Streptococcus sanguinis and other oral bacteria have been suggested to play a role in triggering or perpetuating the immune response.

Environmental And Precipitating Factors:
Trauma: Local trauma (e.g., biting, vigorous brushing, dental procedures) can act as a trigger by initiating an inflammatory cascade in susceptible individuals.

Stress: Psychological stress is a well-recognized precipitating factor, possibly by modulating the immune system.

Nutritional Deficiencies: Deficiencies in iron, folic acid, zinc, and vitamin B12 are sometimes associated with RAU and may impair mucosal integrity and immune function.

Hormonal Changes: Some women report a correlation between ulcer outbreaks and their menstrual cycle, suggesting hormonal influence.

Systemic Conditions: While the patient in this case had no systemic symptoms, RAU can sometimes be a manifestation of underlying systemic diseases such as Behçet's disease, inflammatory bowel disease, or celiac disease. In such cases, the pathophysiology is linked to the broader systemic inflammatory or autoimmune processes.

In aphthous ulcers herpetiform, the multiplicity of lesions suggests a more widespread or intense localized immune reaction, potentially due to a heightened sensitivity or dysregulation of the immune response to triggering factors. The small, clustered nature of the ulcers might reflect a focal and intense inflammatory attack on multiple adjacent epithelial cells, leading to rapid coalescence.

The patient was advised on symptomatic management and preventive measures. Topical corticosteroids (0.05% clobetasol propionate ointment) were prescribed to be applied directly to the lesions three times a day after meals. Additionally, a chlorhexidine gluconate mouthwash (0.12%) was recommended twice daily to prevent secondary infection and promote healing. The patient was educated on avoiding trigger factors such as spicy or acidic foods, trauma to the oral mucosa, and stress. Nutritional counselling was also provided, emphasizing the importance of a balanced diet and adequate vitamin intake.

At the two-week follow-up, the patient reported significant improvement in pain and healing of the ulcers. The lesions had almost completely resolved. Over the subsequent six months, the patient experienced one milder recurrence, which was managed effectively with topical corticosteroids. The patient was advised to continue with meticulous oral hygiene and to report any significant changes or increased frequency of recurrences.

CONCLUSION

Aphthous ulcers herpetiform, though less common than other forms of recurrent aphthous stomatitis, can cause significant discomfort due to the multiplicity and coalescing nature of the lesions [7]. The etiology of aphthous ulcers remains unclear, but it is believed to involve a multifactorial interplay of genetic predisposition, immunological dysfunction, microbial factors, and various precipitating factors such as stress, trauma, hormonal changes, and nutritional deficiencies [8, 9]. The recurrent nature of the lesions in this case highlights the chronic and relapsing course of aphthous ulcers herpetiform [10]. Management primarily focuses on symptom relief and reducing the frequency and severity of recurrences [11]. Topical corticosteroids are the mainstay of treatment during acute episodes, while other agents like immunomodulators may be considered for severe, recalcitrant cases [12]. Patient education on identifying and avoiding trigger factors is crucial for long-term management.

Further research is warranted to fully elucidate the underlying mechanisms and to develop more targeted and effective long-term preventive strategies for this condition.

REFERENCES:

1. Scully C, Porter S. Oral mucosal disease: recurrent aphthous stomatitis. Br Dent J. 2008 Apr 12;204(6):307-14.
2. Ship II. Epidemiologic aspects of recurrent aphthous ulcerations. Oral Surg Oral Med Oral Pathol. 1972 Aug;33(2):404-9.
3. Woo SB, Greenberg MS. Recurrent aphthous ulcers: a review. Adv Dermatol. 1999;15:159-87.
4. Scully C, Porter S. Oral mucosal disease: recurrent aphthous stomatitis. Br Dent J. 2008 Apr 12;204(6):307-14.
5. Ship II. Epidemiologic aspects of recurrent aphthous ulcerations. Oral Surg Oral Med Oral Pathol. 1972 Aug;33(2):404-9.
6. Woo SB, Greenberg MS. Recurrent aphthous ulcers: a review. Adv Dermatol. 1999;15:159-87.
7. Jurge S, Kuffer R, Scully C. Recurrent aphthous stomatitis. Oral Dis. 2006 Jan;12(1):1-21.
8. Porter SR, Hegarty A, Kaliakatsou F, Hodgson TA, Scully C. Recurrent aphthous stomatitis. Crit Rev Oral Biol Med. 2002;13(4):306-21.
9. Akintoye SO, Greenberg MS. Recurrent aphthous stomatitis: a review of the etiology, pathogenesis and management. Dent Clin North Am. 2014 Apr;58(2):299-311.
10. Preeti L, Magesh KT, Rajkumar K, Karthik MR. Recurrent aphthous stomatitis. J Oral Maxillofac Pathol. 2011 May;15(2):166-70.
11. Rivera C, Hotz C, Rivera P, Peñaloza C, Cortés R. Recurrent aphthous stomatitis: clinical features and immunopathogenesis. Int J Oral Sci. 2014 Jun;6(2):65-72.
12. Noli K. Aphthous stomatitis: a review of clinical presentation, etiology, and management. J Clin Aesthet Dermatol. 2011 Oct;4(10):24-9.