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Herpetic Ulcer: A Case Report with Clinical Perspectives

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Abstract

Herpetic infections of the oral cavity are common viral lesions caused by herpes simplex virus (HSV), characterized by episodic ulceration preceded by vesicle formation. Although primary infections are frequently seen in children, recurrent lesions may occur in adults and can mimic other ulcerative conditions. It classified into primary herpetic gingivostomatitis, which presents as multiple painful oral ulcers with fever and gingivitis; recurrent intraoral herpes, which occurs due to reactivation and appears as small clustered ulcers on keratinized mucosa like the hard palate and attached gingiva; and herpes labialis (cold sores), which affects the lips and is triggered by stress, fever, or sunlight.The condition is treated with antiviral therapy and supportive therapy like analgesics and antiviral mouthwash.antiviral drugs include valacyclovir,acyclovir are used especially in prodronal phase to reduce severity and duration.early recognition of lesion pattern and site plays a key role in differentiating herpetic ulcers from other oral ulcerative disorders.this report describes a case of herpetic ulcer in 50 year old female patient who reported to the department of oral medicine and radiology.

Introduction

Herpetic ulcer is a painful vesiculo-ulcerative lesion of the oral mucosa resulting from infection with Herpes Simplex Virus is a DNA virus responsible for oral herpetic infections[1,2]. It primarily causes herpetic stomatitis (primary infection), commonly seen in children as widespread painful vesicles and ulcers associated with pyrexia and gingivitis[3,4].After the primary infection, the virus remains latent in the trigeminal ganglion and may later reactivate to produce recurrent herpetic ulcers, which are usually localized, smaller, and occur on keratinized oral mucosa such as the hard palate and gingiva, It is a self-limiting condition in immunocompetent individuals, typically healing within 7–10 days for recurrent lesions and 10–14 days for primary infection without scarring.[5],[6]Recurrence episode arise from the reactivation of the latent virus in the trigeminal ganglion, often triggered by factors such as stress, fever, trauma, sunlight exposure, or immunosuppression[6],[7].Etiopathologically, the virus targets the epithelial cells resulting in ballooning degeneration, vesicle formation, and subsequent ulceration, while

pathophysiologically it travels along sensory nerves to the trigeminal ganglion where it stays latent and later reactivates to produce recurrent lesions[2,3].Types include primary herpetic gingivostomatitis (diffuse painful ulcers with systemic symptoms), recurrent intraoral herpes (localized ulcers on keratinized mucosa such as hard palate and gingiva), and herpes labialis (cold sores on lips)[3,4]. Herpetic ulcers are characterized by a prodromal phase, during which patients often report burning, tingling, or discomfort prior to ulcer formation[6]. Early identification is crucial for appropriate management[3].Investigations include Tzanck smear, viral culture, PCR, and serology[5]. Management involves supportive care such as maintaining oral hygiene, adequate hydration, and a soft diet, along with symptomatic relief using analgesics, topical anesthetics, and antiseptic mouthwashes[3,4]. Antiviral drugs may be used as preventive or therapeutic agents in certain cases[5,6].This case report highlights the clinical presentation, diagnosis, and management of herpetic ulcers of the oral cavity, emphasizing the importance of prompt recognition and appropriate treatment in dental practice.

Case Report

A 50-year-old female patient presented the Department of Oral Medicine and Radiology with a chief complaint of burning sensation in the upper right palatal region for the past 3 months.Patient was apparently normal before 3 months after which she developed persisting burning sensation and pain which is sudden in onset , intermittent and pricking type , that aggravated on intake of spicy foods.It was also associated with fever. No relevant past medical, dental and surgical history. .Patient consumes mixed diet , brushes once daily , is under stress On General examination Patient was conscious , cooperative and oriented to time , place and person.On extraoral examination no facial asymmetry present and Palpable regional lymph nodes detected in head and neck region. On TMJ examination reveals normal mouth opening. And normal tmj movement .On intraoral soft tissue examination(Fig:1) , Presence of multiple small crusted ulcers approximately 1 × 2 mm in size, with well defined margin, , with erythematous border surrounding central whitish pseudomembranous slough located on the right posterior palatal region in relation to 16 ,17 ,18. On palpation mild tenderness and burning sensation present. No other secondary changes present. Intraoral hard tissue examination reveals , dental caries in 36 with calculus and stains .Patient was advised for Routine Hematological investigations which were under normal limit.Based on history and clinical findings the condition was diagnosed with Herpetic ulcer in right posterior hard palate. Aphthous ulcer was considered as a differential diagnosis.

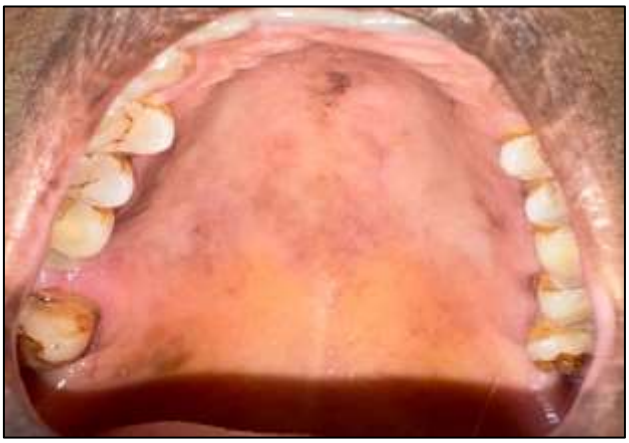
PRE -OPERATIVE IMAGE

Fig :1 shows Presence of multiple small crusted ulcers approximately 1 × 2 mm in size, with well defined margin, , with erythematous border surrounding central whitish pseudomembranous slough located on the right posterior palatal region in relation .

Treatment done : The patient was prescribed with antiviral drug topical acyclovir 5% cream to be applied two to three times daily for 1 week and the patient was instructed regarding medication adherence. also he was advised to maintain good oral hygiene , brush twice daily and avoid spicy food as conservative therapy and was given with analgesic and antiviral mouth wash as supportive therapy. The patient was scheduled for a follow-up after one week for review after one week follow up ,Patient reviewed, signs and symptoms were reduced. Healing was satisfactory .Patient was kept under follow-up for recurrence .

POST OPERATIVE IMAGE

Figure 2: signs and symptoms were reduced. Healing was satisfactory



Discussion:

Herpetic ulceration is a clinically significant mucocutaneous lesion caused by Herpes Simplex Virus (HSV), a double-stranded DNA virus of the Herpesviridae family. HSV infection are are among the most common viral infections globally ,with HSV-1 being the primary type associated with oral lesion .Pathophysiologically, the virus infects epithelial cells and then travels via sensory nerves to remain latent in the trigeminal ganglion, with later reactivation causing recurrent lesion[4,5].The condition does not eshibit a significant gender preference with a male to female ratio of approximately 1:1, and morbidity is generally low in healthy individuals but can be significant in children or immunocompromised patients due to pain, dehydration, and difficulty in eating[4,5]. HSV-1 mainly occurs in children aged between 6 months to 5 years, presenting as oral lesions[4], whereas HSV-2 is more prevalent in adolescents and adults and is typically associated with genital lesions rather than oral involvement[5].Herpetic ulcers are broadly classified into primary and secondary types based on the stage of infection and clinical presentation[3,5]. Primary herpetic ulcer, also reffered to as primary herpetic gingivostomatitis, develop during the initial exposure to HSV-1 and are more commonly seen in children and young adults[4,5]. these lesions are often accompined by systemic symptoms such as fever, malaise, headache, and regional lymphadenopathy[4]. In immunocompetent individuals, the ulcer typically resolves within 10–14 days without scarring, after which the virus become latent in the trigeminal ganglion[3,4].Secondary herpetic ulcer, also referred to as recurrent herpes simplex infection, results from reactivation of the latent virus[3,4]. Reactivation may be triggered by stress, fever, trauma, sunlight exposure, immunosuppression, or hormonal changes[5,6]. The lesions usually present as a clusters of vesicles that rupture to form small, shallow ulcers and typically heal within 7–10 days[6]. A common clinical presentation of secondary infection is herpes labialis[6].Primary infection often presents as acute herpetic gingivostomatitis, characterized clinically by multiple painful vesicles that rapidly rupture to into shallow ulcers affecting the gingiva, oral mucosa, and perioral tissues,

often associated with systemic symptoms like fever and malaise[7,8]Although patient history may vary, the clinical morphology alone can strongly suggest herpes infection, particularly when lesions occur on keratinized tissues such as the palate[7]. This site predilection is diagnostically important because most other common oral ulcers, such as aphthous ulcers, predominantly occur on non-keratinized mucosa[7].

Diagnosis of intraoral herpes is primarily clinical, especially when lesions exhibit classical morphology and distribution[3,4]. Laboratory confirmation through cytology, viral culture, or PCR testing is typically reserved for atypical cases, immunocompromised patients, or research purposes[5]. Investigations may include Tzanck smear (which reveals multinucleated giant cells), viral culture (shows multinucleated giant cells and intranuclear inclusion bodies), PCR, or serology[5]. Early recognition is essential because antiviral therapy is most effective when initiated during the prodromal or early ulcerative stage[1,2]. Timely treatment reduces lesion duration, viral shedding, and symptom intensity[5,6].The differential diagnosis for palatal ulcerations includes traumatic ulcer, thermal burn, necrotizing sialometaplasia, aphthous stomatitis, erythema multiforme, and vesiculobullous disorders[7,8]. Traumatic ulcers are usually solitary and associated with an identifiable mechanical cause, whereas necrotizing sialometaplasia presents as a deep crater-like ulcer[7]. Aphthous ulcers rarely occur on keratinized mucosa, and immune-mediated vesiculobullous diseases typically present with widespread mucosal involvement rather than localized clustered lesions[7,8]. In contrast, the grouped appearance, superficial ulceration, and erythematous base are strongly suggestive of herpetic origin[7]

Management includes definitive, preventive, supportive, and conservative approaches[3,7]. Definitive treatment involves systemic antiviral therapy such as acyclovir and valacyclovir, which are most effective in reducing severity and duration of lesions[1,6]. Preventive management includes antiviral mouthwashes to reduce local viral activity[3]. Supportive treatment focuses on symptomatic relief using analgesics and topical anesthetics[3,7]. Conservative measures include stress reduction, maintenance of good oral hygiene, and adherence to a soft and less spicy diet to promote healing[3,7]. Early recognition of lesion pattern and site plays a key role in differentiating herpetic ulcers from other oral ulcerative disorders[7],

Conclusion:

This case emphasizes the importance of recognizing the distinctive clinical features of intraoral herpetic lesions, particularly their predilection for keratinized mucosa[7]. Herpetic ulcer is a painful vesiculo-ulcerative lesion of the oral mucosa caused by Herpes Simplex Virus (HSV-1)[2,5]. Accurate differentiation from other ulcerative lesions is essential to ensure appropriate management and to avoid unnecessary interventions[7,8]. Prompt antiviral therapy and patient education regarding recurrence triggers are key components of successful treatment[1,6]. Prevention is better than cure; lifestyle modifications, including stress management and avoidance of known triggering factors, may help reduce the frequency and severity of recurrent infections[5,6].

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