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International Dental Journal of Student's Research

Journal homepage: https://www.idjsronline.com/

Case Report

Non-surgical management of periodontal disease exacerbated by herpes virus: A case report

Zubeida Haleem[1]*, Shivanand Aspalli[1], Nagappa Guttiganur[1], Narayana Geethika[1]

[1]Dept. of Periodontics and Oral Implantology, AME's Dental College and Hospital, Raichur, Karnataka, India.

Abstract

Periodontal disease is a chronic inflammatory condition affecting the supporting structures of the teeth, primarily driven by bacterial pathogens and host immune responses. However, recent evidence suggests that viral infections, particularly Herpes Simplex Virus (HSV), may play a significant role in the pathogenesis and progression of periodontal disease. HSV, a neurotropic virus with lifelong latency, is known to modulate immune responses and contribute to tissue destruction in various oral conditions.

HSV-1, the predominant strain affecting the oral cavity, has been detected in periodontal pockets and is associated with increased inflammation, immune dysregulation, and enhanced bacterial virulence. The virus is thought to exacerbate periodontal disease through mechanisms such as cytokine upregulation, apoptosis of host cells, and interactions with periodontal pathogens. Furthermore, HSV has been linked to aggressive and necrotizing periodontal diseases, particularly in immunocompromised individuals.

Understanding the relationship between HSV and periodontal disease is crucial for improving treatment strategies. Investigating viral contributions to periodontal pathology may open new avenues for targeted antiviral therapies alongside conventional periodontal treatment. This case report highlights the relationship between HSV and periodontitis, emphasizing the importance of non-surgical management approaches in treating periodontal conditions exacerbated by the virus.

Keywords: Herpes simplex virus, Periodontal diseases, Gingival enlargement

Received: 03-02-2025; **Accepted:** 29-04-2025; **Available Online**:14-06-2025

1. Introduction

Periodontal disease is a complex inflammatory disorder that compromises the integrity of tooth-supporting structures. It results from an imbalance in the oral microbiome and immune system responses. While bacterial species such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* are well-recognized etiological agents, recent findings indicate that viral infections, notably Herpes Simplex Virus (HSV), may also contribute to disease initiation and progression.[1-2]

The interaction between herpesviruses and bacteria can be bidirectional with bacterial enzymes or inflammation induced factors, having the potential to activate periodontal herpesvirus. Periodontitis has a sequential infectious process that proceeds from bacteria to herpes virus to bacteria. Initially bacteria present in the dental biofilm induce gingivitis which then permits latent herpesvirus which is embedded in the DNA of macrophages, T lymphocytes and B lymphocytes to infiltrate the periodontium. Reactivation of the latent herpesvirus can occur spontaneously or during the period of decreased host defence seen because or drug induced immunosuppression, emotional stress, concurrent infection, hormonal changes, physical trauma. Herpes virus activation leads to increased inflammatory mediator response triggering a cytokine or chemokine of 1L-1β,TNF α,1L 6,prostaglandins, interferons, and other mediators which have potential to propagate bone resorption .Most of the herpes virus, associated cytokines and chemokines are prominent in periodontal lesions. In a vicious circle, the triggering of cytokine responses may activate latent herpes virus which will further aggravate periodontal disease. Moreover, most immune competent individuals experience episodes of oral herpes virus reactivation, but it will last only

*Corresponding author: Zubeida Haleem
Email: uroosahaleem@gmail.com

DOI: https://doi.org/10.18231/j.idjsr.2025.019

for few hours or day, which is very less time span to initiate periodontal disease. Association between systemic diseases like cardiac diseases and viruses is still a question. Vascular disease occur at a higher frequency as a result of combined infections with herpes simplex virus and Chlamydia pneumonia. HSV and periodontopathic bacteria in aggregate and herpesvirus 8 have also been associated with vascular diseases.[3]

2. Case Presentation

A 28-year-old male patient reported to the Department of Periodontics and Oral Implantology with a chief complaint of swollen gums in the mandibular anterior region persisting for 4 to 6 months. The swelling had gradually increased in size and bled upon brushing, leading the patient to cease oral hygiene practices for the past two weeks.

Figure 1: Enlargement at baseline

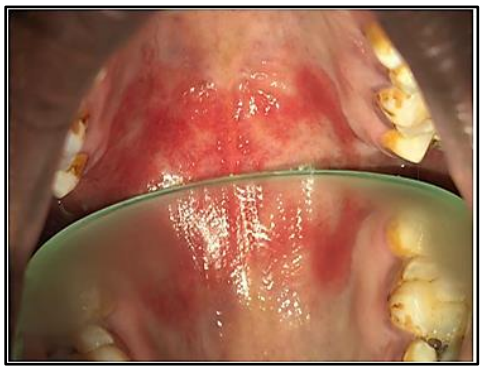

Figure 2: Palatal view at baseline showing vesicles

Figure 3: Enlargement post antiviral therapy of 2 weeks

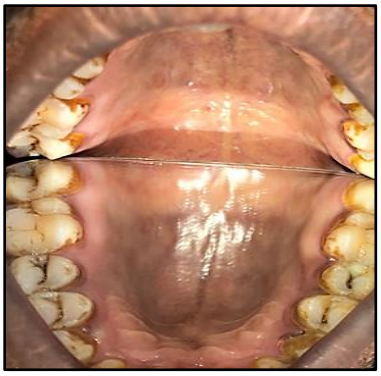

Figure 4: Palatal view post antiviral therapy of 2 weeks

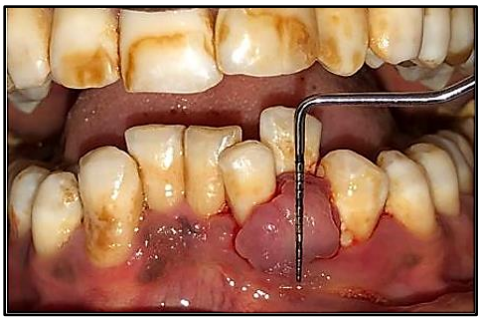

Figure 5: Post ultrasonic scaling

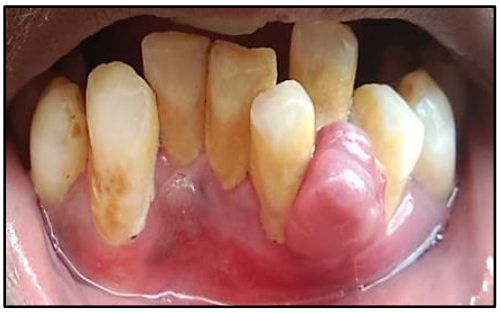

Figure 6: Relapsed in enlargement after 6 months of missed follow up

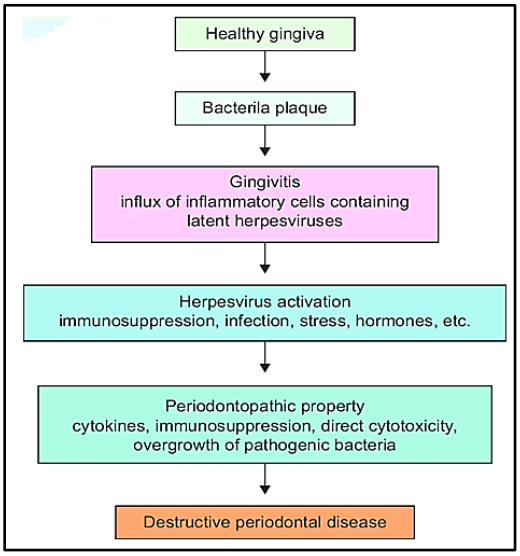

Figure 7: Relationship of HSV with periodontics

3. Medical and Personal History

The patient's medical history was non-contributory. He followed a mixed diet and had no adverse oral habits.

4. Clinical Examination

Intraoral examination revealed:

1. Reddish-pink gingiva with round, rolled-out margins
2. Soft, edematous consistency with an absence of stippling.
3. Gingival enlargement measuring approximately 15mm (W) x 14mm (B) x 14mm (L) in relation to teeth 31, 32, and 33 (classified according to Bokenhamp and Bohnhorst, 1994) (**Figure 1**).
4. Positive bleeding on probing
5. Poor oral hygiene index (OHI-S by Greene and Vermillion: 3.16)
6. Plaque index (Silness and Löe: 2.0, indicating poor oral hygiene)
7. Gingival index (Löe and Silness: 2.70, indicating severe gingivitis)
8. Presence of small vesicles on the soft palate and anterior & posterior faucial pillars (**Figure 2**)

5. Provisional Diagnosis and Investigations

Based on clinical findings, a provisional diagnosis of localized inflammatory gingival enlargement was made. Considering the presence of vesicular lesions, a viral etiology was suspected, and the patient was referred to Raichur Institute of Medical Sciences, Government Hospital, Raichur, for further evaluation. Laboratory investigations revealed HSV-1 IgM: Positive (8.5 AU/ml) and HSV-1 IgG: Moderate (50 AU/ml).

5.1. Management and follow-up

The patient was prescribed antiviral therapy with Acyclovir 400 mg BID for two weeks. Post-therapy, laboratory investigations revealed - HSV-1 IgM: Negative (<1.0 AU/ml) and HSV-1 IgG: Increased (100.514 AU/ml). Clinically, a significant reduction in gingival enlargement measuring 10mm x 9mm x 12mm and vesicular lesions on palate was observed (**Figure 3** & **Figure 4**). The final diagnosis of localized inflammatory gingival enlargement exacerbated by HSV-I was established.

Following the antiviral therapy, non-surgical periodontal therapy, including scaling and root planing, was performed. The patient was prescribed 0.12% chlorhexidine mouthwash and Metrogyl DG gel for two weeks. At the subsequent follow-up, the gingival enlargement had further reduced to 9mm x 7mm x 8mm (**Figure 5**).

The patient was advised regular follow-ups but did not return until six months later, presenting with a relapse in gingival overgrowth to 12mm x 10mm x 12mm (**Figure 6**). This highlights the critical role of patient compliance in achieving successful periodontal outcomes. Failure to adhere to follow-up visits and recommended investigations may lead to disease recurrence and progression, complicating management.

6. Discussion

Emerging evidence suggests a bidirectional relationship between Herpes Simplex Virus (HSV) infection and periodontal disease, where HSV may contribute to disease progression, while chronic periodontal inflammation may facilitate viral reactivation. In the present case, the detection of HSV-1 IgM positivity in a patient with gingival enlargement aligns with studies demonstrating the presence of HSV-1 and HSV-2 in periodontal pockets, suggesting a potential role in disease pathogenesis.[1] HSV infection is known to trigger a strong inflammatory response, upregulating proinflammatory cytokines such as IL-1β and TNF-α, which promote periodontal tissue destruction.[4] This inflammatory mechanism explains the persistent gingival inflammation seen in our patient. Additionally, HSV has been shown to facilitate the colonization and virulence of periodontal pathogens such as *Porphyromonas gingivalis* and *Tannerella forsythia*, worsening periodontal disease.[2] This correlation is evident in our case, where the patient exhibited persistent gingival overgrowth despite undergoing non-surgical therapy. Furthermore, a study investigating the transcriptomes of human gingival fibroblasts following HSV-1 infection revealed the upregulation of inflammatory pathways, suggesting that HSV-1 may directly contribute to gingival overgrowth and tissue destruction.[5] This mechanistic insight further supports our findings, as the patient's gingival enlargement did not fully resolve with conventional periodontal therapy alone.

Conversely, periodontal disease itself may create an environment that promotes HSV reactivation. Chronic periodontal inflammation weakens local immune defenses, increasing susceptibility to HSV recurrence.[6] The presence of chronic inflammation in our patient likely facilitated persistent HSV activity, contributing to gingival enlargement. Studies suggest that periodontal pockets serve as latent reservoirs for HSV, allowing for recurrent infections under conditions of stress or immunosuppression.[7] This hypothesis aligns with our case, where recurrent gingival enlargement was observed despite initial improvement.

A significant challenge in our case was the recurrence of gingival overgrowth following a six-month lapse in follow-up, emphasizing the impact of non-compliance on disease relapse. Similar patterns have been observed in other studies where inadequate adherence to periodontal maintenance led to treatment failure and recurrence. Herrera et al.(2021) documented a case of drug-influenced gingival enlargement in a young patient undergoing non-surgical periodontal therapy, where poor compliance over four years resulted in recurrent gingival overgrowth.[8] Likewise, Ardila and Bedoya-García (2019) reported persistent gingival hyperplasia in a patient with anticonvulsant-induced

overgrowth due to irregular compliance with systemic and local interventions.[9] Our case reinforces the necessity for strict periodontal maintenance, particularly in viral-associated cases, where chronic inflammation can drive disease recurrence if left unchecked.

While surgical intervention such as gingivectomy is often considered for persistent gingival enlargement, we opted against it in this case for two primary reasons. Firstly, the patient declined surgical excision, preferring a non-invasive approach. Secondly, given the viral etiology of the gingival enlargement, non-surgical management with antiviral therapy was prioritized to assess the response and reduce the risk of unnecessary surgical trauma. Studies have suggested that surgical excision in viral-induced gingival enlargements may not address the underlying cause and could lead to recurrence if the viral component remains active.[5] In our patient, the enlargement initially reduced following antiviral and periodontal therapy, supporting the decision to avoid immediate surgical intervention. However, due to non-compliance with follow-up care, relapse was observed, reinforcing the need for strict long-term maintenance.

HSV infection has been associated with an increased prevalence of severe periodontitis, with evidence suggesting that HSV can replicate within gingival fibroblasts, keratinocytes, endothelial cells, and inflammatory cells, leading to direct cytopathic effects and immune dysregulation.10 Additionally, HSV may impair host immune responses, increasing susceptibility to bacterial invasion and further disease progression.[11] Previous research has demonstrated that viral replication within monocytes, lymphocytes, and macrophages allows HSV to alter immune mechanisms, directly or indirectly influencing host responses.[12-13] This aligns with our case, where persistent gingival enlargement and inflammation were observed despite traditional periodontal therapy.

Furthermore, herpesviruses have been implicated in viral-bacterial co-infections, where viral activation enhances bacterial virulence while simultaneously reducing the ability of periodontal tissues to resist bacterial invasion by infecting or altering structural and host defense cells of the periodontium, ultimately worsening periodontal disease outcomes.[14-16] This has led to growing interest in the potential role of antiviral therapy in managing refractory periodontitis. Some studies have reported positive responses to antiviral treatment in severe periodontal cases, suggesting that targeted antiviral interventions may be beneficial for HSV-associated gingival enlargement.[17] Our case highlights the necessity for integrating antiviral strategies with periodontal treatment protocols to improve long-term outcomes in patients with HSV-associated periodontal disease.

7. Conclusion

This case highlights the potential role of viral infections in exacerbating inflammatory gingival conditions and emphasizes the necessity of early diagnosis, appropriate therapeutic intervention, and stringent follow-up to prevent recurrence. Additionally, patient education and adherence to follow-up schedules are essential to ensure optimal treatment outcomes and prevent disease relapse.

In conclusion, the notion that herpesviruses play a key role in severe periodontitis has significant therapeutic implications. A new direction in periodontal disease prevention and treatment may focus on controlling disease-initiating herpes viruses. Research has shown that reducing gingivitis through antiplaque measures can diminish the herpes viral load in periodontal sites, potentially mitigating disease progression.

Understanding these associations reinforces the importance of routine periodontal assessment in individuals with recurrent HSV infections. Recent advancements in studying herpes viral - bacterial interactions and host inflammatory mechanisms hold great promise in developing new preventive and curative strategies.

8. Future Directions

Future research should focus on longitudinal and interventional studies to establish causality and identify optimal therapeutic strategies. A multidisciplinary collaboration between periodontists, virologists, and immunologists can significantly improve patient outcomes, ultimately leading to innovative ways to prevent and treat periodontitis.

9. Source of Funding

None.

10. Conflict of Interest

None.

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How to cite: Haleem Z, Aspalli S, Guttiganur N, Geethika N, Non-surgical management of periodontal disease exacerbated by herpes virus: A case report, *Int Dent J Stud Res*. 2025;13(2):102-106.