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Case Report

Verrucous hyperplasia with moderate oral epithelial dysplasia: A case report

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Abstract

Objective: To present a case of verrucous hyperplasia (VH) with moderate epithelial dysplasia in a 52-year-old male, detailing clinical features, histopathological findings, and treatment.

Case Presentation: A 52-year-old male reported pain and a gradually enlarging lesion on the left buccal mucosa for six months. He had a prior history of bidi smoking and gutka consumption, though discontinued 3–4 years ago. Clinical examination revealed a corrugated, tender, whitish-pink mass on the left buccal mucosa with associated oral submucous fibrosis. Histopathology of the excised lesion confirmed VH with moderate dysplasia.

Results: The lesion was managed surgically with adjuvant topical therapy, including corticosteroids and Immunomodulator drugs. Follow-up indicated clinical improvement with no evidence of recurrence.

Conclusion: Early diagnosis and intervention in VH are essential to prevent progression to verrucous carcinoma. Histopathology remains crucial for differentiation from other potentially malignant lesions.

Keywords: Verrucous hyperplasia, Oral epithelial dysplasia, Oral potentially malignant disorder, Buccal mucosa.

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1. Introduction

Verrucous hyperplasia (VH) is a well-recognized premalignant exophytic epithelial lesion of the oral mucosa, most commonly affecting the buccal mucosa and gingiva, particularly in individuals with longstanding habits such as tobacco chewing and smoking.[1] Clinically, it presents as a whitish or pink, verrucous or papillary surface lesion, which may resemble other oral proliferative lesions, including verrucous carcinoma (VC) and proliferative verrucous leukoplakia (PVL).[2]

VH was first clearly defined by Shear and Pindborg in 1980, who described it as a lesion characterized by epithelial hyperplasia without invasion into connective tissue, distinguishing it from VC.[2] However, due to overlapping clinical and histological features, accurate differentiation is critical for appropriate management and prognosis. VH exhibits a low but definite risk of malignant transformation (~3–5%), necessitating histopathological confirmation and long-term follow-up.[3,4]

Recent histological classifications by Wang et al. divide VH into plaque-type and mass-type variants. The mass-type variant is associated with a higher risk of transformation and may mimic early verrucous carcinoma, especially in the presence of epithelial dysplasia.[3] Moreover, molecular alterations, such as p53 mutations, iNOS overexpression, and allelic loss, may indicate early genetic instability and support the concept of VH as a potentially malignant disorder.

2. Case Presentation

A 52-year-old male presented to the Department of Oral Medicine and Radiology at ITS Dental College, Murad Nagar, Ghaziabad, India, with pain while chewing food for six months. Initial symptoms included burning with spicy

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food, later developing into a painful lesion on the left buccal mucosa, exacerbated during mastication. The patient had a restricted mouth opening (34 mm), a history of tuberculosis (2004–2007), and extractions of teeth #27 and #28. He reported cessation of bidi smoking and gutka use 3–4 years ago but had a 14-year history of both.

2.1. Clinical examination

A 1 x 1 cm tender, sessile, whitish-pink mass with a corrugated surface was noted on the left buccal mucosa. Bilateral blanching and a 3 x 4 cm keratotic plaque with a cracked mud appearance were observed. No palpable lymph nodes were present. TMJ evaluation showed bilateral clicking, deviation to the left, and restricted mouth opening. (**Figure 1**)

2.2. Histopathological findings

Excisional biopsy revealed exophytic papillary projections with hyperplastic stratified squamous epithelium overlying fibrovascular connective tissue. Koilocytes and hyperparakeratinization were present. Rete ridges had broad "elephant's foot" morphology. Basal epithelial cells showed dysplastic changes. Dense chronic inflammatory infiltrates were noted juxta-epithelially.(**Figure 2**)

2.3. Treatment

The patient underwent surgical excision of the lesion under local anesthesia, ensuring adequate clearance of margins to minimize the risk of recurrence or malignant transformation.

Postoperatively, the patient was prescribed a short course of systemic medications for inflammation control, infection prophylaxis, and symptomatic relief, including: Pantoprazole 40 mg once daily (proton pump inhibitor for gastric protection), Chymoral Forte (trypsin-chymotrypsin) twice daily (anti-inflammatory enzyme therapy), Amoxicillin 500 mg three times daily for 5 days (antibiotic), Zerodol-SP (aceclofenac + serratiopeptidase + paracetamol) twice daily (analgesic and anti-inflammatory), Betadine® gargles for local antisepsis, three times daily.

In the subsequent healing phase, a topical therapeutic regimen was initiated to promote mucosal repair and control inflammation: Rexidine M Forte gel (chlorhexidine + metronidazole), Kenacort oral paste (triamcinolone acetonide 0.1%), Betnesol mouthwash (betamethasone), SM Fibro tablets (containing micronutrients and anti-fibrotic agents) Candid B paint (clotrimazole + beclomethasone) for antifungal and corticosteroid action.

In the final phase of management, topical Imiquimod 5% cream was applied to the surgical site on alternate days for immunomodulatory support and to reduce the risk of dysplastic progression, particularly due to the presence of mild epithelial dysplasia in histopathology.

The patient demonstrated progressive clinical improvement with no evidence of recurrence at the time of writing. Regular follow-up has been scheduled at monthly intervals for the first 6 months and biannually thereafter, with emphasis on habit cessation counseling and long-term surveillance.

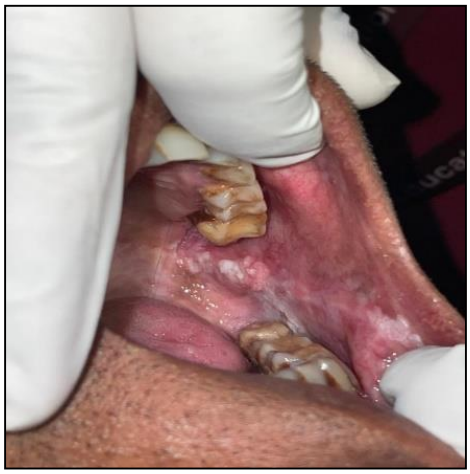

Figure 1:

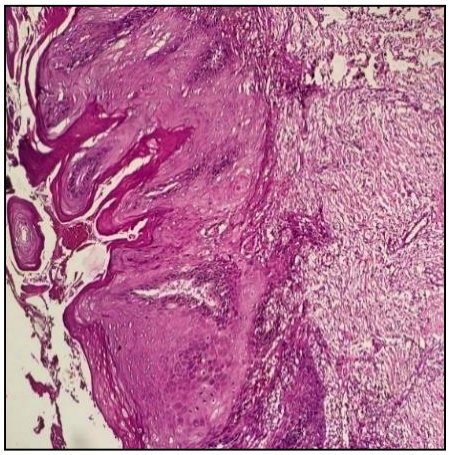

Figure 2:

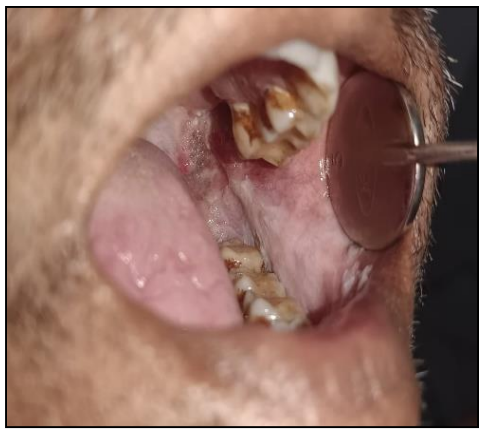

Figure 3:

3. Discussion

VH remains a diagnostic challenge due to its clinicopathological overlap with verrucous carcinoma (VC) and proliferative verrucous leukoplakia (PVL). While both VH and VC show exophytic, verrucous growths, VC is characterized histologically by endophytic proliferation and blunt rete pegs infiltrating the underlying connective tissue—features absent in VH.[2,4] Therefore, deep biopsy and multiple sections are crucial to rule out VC.

The classification by Wang et al. into plaque-type and mass-type VH has significant clinical implications. The mass-type may represent a more advanced stage and show higher incidence of epithelial dysplasia and malignant transformation, suggesting the need for more aggressive treatment.[3]

Differentiation from PVL is equally important, as PVL is a multifocal, progressive lesion with a notoriously high rate of malignant transformation, often resistant to conventional therapies.[6] While PVL is typically multifocal and evolves slowly, VH is usually localized and shows a verrucous architecture without progressive spread, unless associated with dysplasia.

Another differential is papillary squamous cell carcinoma (PSCC), which presents with papillary architecture but shows marked cytological atypia and deeper connective tissue invasion, distinguishing it from VH.[4]

Molecular studies have provided insight into the pathogenesis and transformation risk of VH. Chen et al. reported increased inducible nitric oxide synthase (iNOS) expression in VH lesions, indicating an inflammatory–oncogenic link.[7] Similarly, Poh et al. demonstrated frequent allelic loss, suggesting early genetic events in verrucous lesions.[8] Overexpression of tumor suppressor genes (p53) and oncogenic markers (EGFR, cyclin D1) further implicates these pathways in malignant potential.[9]

The primary treatment modality is complete surgical excision with histologically clear margins.[10] Adjunctive therapies may include topical agents like Imiquimod, especially in cases with epithelial dysplasia or limited resection potential. More importantly, habit cessation (tobacco, areca nut) and regular monitoring are vital to prevent recurrence and progression.

4. Conclusion

VH presents diagnostic challenges due to overlap with other verrucous lesions. Histopathological evaluation and long-term monitoring are essential. Early intervention and patient education remain the cornerstone of management.

5. Source of Funding

None.

6. Conflict of Interest

None.

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