Int. J. Sci. R. Tech., 2026 3(4)
A Multidisciplinary peer-reviewed Journal
www.ijsrtjournal.com [ISSN: 2394-7063]

Original Article

Verrucous Hyperplasia: More Than Just A Benign Overgrowth- A Review

Abiksha C.*, Mathumala Subramanian, G. Keerthana, S. Marytresa Jeyapriya, M. Sathish Kumar

Department of Oral and Maxillofacial Pathology Karpaga Vinayaga Institute of Dental Sciences Chengalpattu, India

ABSTRACT

Verrucous papillary lesions (VPLs) of oral cavity are diagnostically challenging as they include a spectrum of benign, potentially malignant, and frankly malignant lesions. A majority of the benign VPLs have viral aetiology and include commonly occurring squamous papilloma along with verrucavulgaris, focal epithelial hyperplasia, and condyloma. Current understanding of potentially malignant. VPLs is perplexing and is primarily attributed to the use of confusing and unsatisfactory terminology. Clinically and histologically oral verrucous hyperplasia, a potentially malignant disorder, resembles oral verrucous carcinoma and may be indistinguishable from one another. Verrucous hyperplasia (VH) is a rare and potentially malignant exophytic oral mucosal lesion with a verrucous or papillary surface. VH has a tendency to transform into its malignant counterpart, verrucous carcinoma, which is clinically indistinguishable, yet histologically variable. The most common site of predilection is the buccal mucosa and VH more prevalent in patients with habits of areca nut chewing or smoking. A biopsy of the lesion with the surrounding normal mucosa and adequate depth should be performed to histopathologically to diagnose the condition and provide treatment management. The most reliable way to separate these entities on routine haematoxylin-eosin-stained tissue sections is to recognize the exophytic growth patterns of oral verrucous hyperplasia from the combined exophytic and endophytic growth patterns associated with verrucous carcinoma. A brief overview of the treatment modalities adopted is also discussed.

Keywords: Hyperplasia; Verrucous Carcinoma; Squamous Cell Carcinoma; Oral epithelial dysplasia

INTRODUCTION

According to dental literature, the term verrucous describes as a wart-like surface morphology seen in some oral lesions. In oral pathology, the word verrucous literally means wart-like or resembling a verruca and is used to describe lesions with rough, papillary or cauliflower-like projections. (1) Verrucous papillary lesions (VPLs) of the oral cavity are diagnostically challenging as they include a spectrum of benign, potentially malignant, and frankly malignant lesions.1 VPL clinically presents themselves as a grey white, exophytic growth in gingiva, buccal mucosa or any other part of the oral cavity and histopathologically they may range from a simple hyperkeratotic lesion to verrucous hyperplasia, verrucous carcinoma or even frank squamous cell carcinoma. (1) Verrucous hyperplasia is a histopathological entity, which shows considerable clinical and histological resemblance to verrucous carcinoma. This was first described by Shear and Pindborg (1980) and differentiated from verrucous carcinoma (2). OVH (Oral Verrucous Hyperplasia) is an elementary step towards OVC (Oral Verrucous Carcinoma), which then transforms to SCC (Squamous Cell Carcinoma) in a later stage (3). Shear et al. first differentiated VH from VC based on the absence of endophytic growth in the former entity, wherein the verrucous and hyperplastic epithelium was completely superficial to the adjacent normal epithelium (4). Verrucous hyperplasia (VH) is a potentially malignant disorder presenting as a white or pink exophytic mucosal lesion with a typical verrucous or papillary surface. The first-ever verrucous papillary lesion reported was by Fridell and Rosenthal in 1941. Ackermann observed 31 similar cases and formulated the term ‘verrucous carcinoma’ for such a condition. However, a complete clinical and

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

histological analysis of such oral mucosal lesions was performed by Shear and Pindborg to differentiate them from VC and verrucous leukoplakia (VL). Buccal mucosa (57.89%) is found to be most commonly involved, followed by tongue, gingiva, alveolar mucosa, soft palate. VH generally occurs in individuals in the 4th to 6th decade of life. There is a striking association between habits of tobacco chewing, areca nut chewing and smoking with the occurrence of VH and its malignant transformation into VC or oral squamous cell carcinoma. The rate of malignant transformation of VH reported is 5.1% in ~54.6 months. The transformation rate of VL is 4 15% in 5-8 years. VH can be differentiated from VC only by histopathological analysis. In VC, the hyperplastic epithelium invades the connective tissue, whereas in VH, the hyperplastic epithelium remains superficial to the normal epithelium (5). Verrucous hyperplasia involving the anterior mandible is very rare. One such rarity is Verrucous proliferation arising from pre cystic lining (6). Early-stage lesions appear clinically as either homogenous or non-homogenous leukoplakias and are diagnosed microscopically as either hyperkeratosis or hyperkeratosis with lichenoid features. The early-stage lesions are indistinguishable from the more common unifocal leukoplakias which often causes a delay in diagnosis where it becomes more difficult to manage therapeutically. The intermediate stage represents the premalignant stage and present clinically as verrucous lesions that are diagnosed microscopically as verrucous leukoplakia or verrucous hyperplasia (VH), terms that are used interchangeably. The late stage represents the malignant stage which appears as an exophytic mass with a verrucous surface contour, are clinically larger and more extensive than VH and are diagnosed microscopically as verrucous carcinoma (VC) (7).

CLINICAL FEATURES

Oral verrucous hyperplasia (OVH) is reclassified into “plaque” and “mass” variants while further stating that clinically both the variants can be called OVH, but histopathologically only “mass” variant fits the bill to be called the same and “plaque” type lesions can be called an oral verruciform leukoplakia.3 Over some time, it has been observed that clinicians have found it difficult to distinguish verrucous hyperplasia from verrucous carcinoma due to its marked similarity in clinical appearance(1). This again can be correlated to early acquisition of habits which is more in Indian males than females (2). The clinical appearance of these lesions has not by and large been well described as much accentuation has been laid on the verrucous/ exophytic nature of these lesions with little consideration being given to colour variation. In addition, these lesions are viewed as clinically indistinct from verrucous carcinomas which are commonly white or greyish white in colour (3). Hazarey et al. reported that placement of tobacco-betel-lime quid (i.e. a mixture of slaked lime, chewing tobacco and betel leaf pieces) in the buccal vestibule was the most predominant habit associated with VH growth (4). The most common site reported for the occurrence of VH was the gingiva and alveolar mucosa. However, a previous study revealed buccal mucosa (41%) to be more frequently involved. Other sites with predilection for the occurrence of VH are the tongue, palate, labial mucosa, sino-nasal mucosa, larynx and perianal region. VH generally occurs in individuals in the 4 to 6th decade of life. Hansen et al (1985) suggested six histological stages ranging from normal mucosa, hyperkeratosis, verrucous hyperplasia, verrucous carcinoma, papillary squamous cell carcinoma and less well differentiated squamous cell carcinoma. Males predominated females (2:1) which is contradictory to other studies. Shear and Pindborg classified VH into a blunt and sharp variety. The blunt variety comprises of broader, f latter and less keratinized verrucous processes, whereas sharp processes are long, narrow and heavily keratinized, leading to a white appearance (5). The occurrence of verrucous proliferation in an intraosseous cyst causes one to speculate on the supposed cause. Various etiologies for verrucous growth in odontogenic lesions proposed thus far include the presence of HPV (human papillomavirus), candidal infection, and the habit of tobacco consumption as these factors have correlated well with oral mucosal verrucous lesions (6). Like the early-stage lesions, most tumours were located on either the gingiva, palate or buccal mucosa with a minority of cases on the tongue or labial mucosa (7). 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. 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 (8). Ackerman and McGavran introduced the term "verrucous hyperplasia" to describe a condition that closely resembles verrucous carcinoma clinically and histologically. A subsequent review by Adkins and Monsour concluded that an entity described as verrucous leukoplakia by many authors may actually correspond to some forms of verrucous hyperplasia (9). Epithelial dysplasia is considered to be a critical factor in determining malignant potential. The presence of moderate or severe dysplasia has been accepted to have the greatest likelihood for malignant transformation, although it is also known that progression to cancer is not inevitable (10).

RADIOGRAPHIC FINDINGS

It has been showed that the possibility of verrucous hyperplasia from a prexisting cyst. Surgeons must be aware of such lesion and it is mandatory to correlate the clinical and histopathological finding to establish a diagnosis and need regular long term follow up. Verrucous hyperplasia involving the anterior mandible is very rare. Verrucal proliferation in odontogenic lesions is an extremely rare occurrence with only a handful of cases reported in the literature till date. One such rarity is Verrucous proliferation arising from pre cystic lining. To the best of our knowledge only nine cases of odontogenic cysts with verrucous proliferation have been reported in English literature of which six were reported as verrucous carcinomas arising in odontogenic cysts (6).

HISTOPLATHOLOGICAL FEATURES

OVH begins as a white plaque of hyperkeratosis known as plaque type variant which can get further transformed into mass type with less keratinization, exophytic growth and proliferative features thus leading to malignant transformation and poorer prognosis (1). As proposed by Shear and Pindborg considerable acanthosis with broadened rete ridges causes deprivation of distant epithelial cells from blood supply and becomes oedematous and swollen. These necrotic cells undergo desquamation, leaving cleft in the surface of the epithelium. Verrucous projections are formed in this way in between clefts. This also explains the presence of the papillary projections of lamina propria, which support the verrucous projections. At a later stage both the verrucous projections and the clefts between them undergo keratinization. Dysplasia is commonly seen in verrucous hyperplasia which is consistent with finding by Shear and Pindborg (1980) (2). Exophytic Para keratinized stratified squamous epithelium proliferating in the form of papillary projections showing keratin plugging. The underlying connective tissue stroma showed moderate chronic inflammatory cell infiltrate, adipose tissue and blood vessels (3) The epithelium exhibited hyper Para keratinisation with a few koilocytes seen in the superficial layers. The rete ridges had a broad 'elephant's foot' shape and were at the same level as that of the adjoining normal epithelium. Some of the cells in the basal layer of the epithelium exhibited dysplastic features. In addition, the underlying connective tissue revealed dense chronic inflammatory cell infiltrates, chiefly concentrated in the juxta-epithelial areas. (4) A histological examination depicted a Para keratinized epithelium with long, broad pushing rete pegs and a para keratin layer exhibiting a typical chevron pattern with scanty connective tissue. The epithelium exhibited basal cell hyperplasia, a loss of stratification, acanthosis, individual cell keratinization, cellular and nuclear pleomorphism at places and minimal abnormal mitotic figures (5). The proposed histopathologic criteria for diagnosis of oral verrucous hyperplasia are as follows:

(a) long and narrow heavily keratinized verrucous pro cesses or broad and flat verrucous processes that are less keratinized;

(b) absence of invasion of the hyperplastic epithelium into the lamina propria as compared with the adjacent normal mucosal epithelium;

(c) presence of cytologic/architectural features of dysplasia (9).

TREATMENT

Total surgical excision has been the conventional mode of treatment for oral verrucous hyperplasia. Transformation and recurrence to either squamous cell carcinoma or verrucous carcinoma have been reported after surgical intervention, but this has been due to non-adherence to strict guidelines and criteria

for surgery (1). The treatment modality executed in the present case was wide surgical excision of the lesion with adequate soft tissue margins to avoid recurrence. Photodynamic Therapy (PDT) is another mode of treatment option because it is well tolerated by patients and can be used repeatedly without cumulative side effects and also contributes to lesser scar formation (3). Surgery alone is the most common method of management for both VC and VH cases, due to their overlapping clinico pathological features. However, it is important to ensure wide surgical excision of the lesion with adequate soft tissue margins so as to avoid recurrence. Although sporadic cases of cervical and distant metastasis have been reported, the overall rate of metastasis is insignificant. (4) Different procedures that have been used in the treatment of VH are surgical excision, chemotherapy, radiotherapy, cryotherapy or a combination of these. Surgical excision is the most conventional and reliable treatment. A wide surgical excision with adequate soft tissue margin and depth is paramount to preventing recurrence of the lesion. Some studies have demonstrated the use of cryogun (Brymill Corp.) cryotherapy and topical 5-aminolevulinic acid-mediated photodynamic therapy as an effective treatment modality for VH and leukoplakic lesions (5). Verrucal proliferation in odontogenic lesions is an extremely rare occurrence with only a handful of cases reported in the literature till date. Surgical treatment considerations include segmental resection, partial mandibulectomy, total excision of lesion with adequate clearance will prevent recurrence. Surgical resection may be the intuitive approach to eliminate the lesion, but the biologic behaviour of verrucous hyperplasia, verrucous leukoplakia and verrucous carcinoma requires addressing the possible underlying viral infection if it exists, in addition to the excisional procedure it requires (6). 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 (8). Poswillo is of the opinion that oral verrucous hyperplasia and verrucous carcinoma should be managed similarly because of the significant overlap in their clinicopathologic features. Recurrence and/or transformation of oral verrucous hyperplasia to either verrucous carcinoma or conventional SCC have been reported after surgical intervention (9).

CONCLUSION

OVH is a premalignant lesion which occurs rarely and requires an early intervention to increase the chances of favourable outcome. Apart from the absence of an endophytic growth pattern in OVH, the presence of dysplasia in OVH was noted in our case report. Thus, biopsies of verrucous lesions should include the adjacent nor mal epithelium in order to ensure correct diagnosis. Follow-up after the treatment acts as a desideratum to assess recurrence potential and clinical behaviour (3) Distinction between VC and VH lesions can only be made histologically, by comparing the level of the rete ridges of the epithelium of the lesion with that of the adjacent normal epithelium. In addition, VH cases may also be confused with verrucous leukoplakia. Thus, biopsies of verrucous lesions should include the adjacent normal epithelium in order to ensure correct diagnosis. As VH has the potential for malignant transformation, patients should be treated in a similar manner to those with VC (4).

REFERENCE

1. Patel S, Pathak J, Dekate K, Shirke KJ, Patel T, Jain MN. Oral Verrucous Hyperplasia: A Case Series. Journal of Contemporary Dentistry. 2018;8(3):163–7.
2. Bodhade A, Hazarey V, Ganvir S. Verrucous hyperplasia: A clinico-pathological study. Journal of Oral and Maxillofacial Pathology. 2011;15(2):187
3. Anjali AK, Pereira T, Shetty S, Babu C. Oral verrucous hyperplasia with dysplasia – A Case Report. Oral Maxillofac Pathol J 2022; 13(1): page no. 64-66

4. Grover S, Jha M, Sharma B, Kapoor S, Mittal K, Parakkat NK, et al. Verrucous Hyperplasia: Case report and differential diagnosis. Sultan Qaboos University Medical Journal. 2017 Mar 30;17(1): e98-102
5. Jain A, Taneja S, Rai A, Yadav A. Management of verrucous hyperplasia involving the buccal mucosa using the buccal fat pad: A case report. World Academy of Sciences Journal. 2022 Mar 14;4(3)
6. Nathiya B, Durairaj D, James Antony Bhagat M, Suresh Kumar G, Raj Prakash B. Orthokeratinizing odontogenic keratocyst transformation in to verrucous hyperplasia- A case report of rare entity. Advances in Oral and SMaxillofacial Surgery. 2022 Apr; 6:100218.
7. Sharon Akrish, Lana Eskander-Hashoul, Adi Rachmiel, Ofer Ben-Izhak, Clinicopathologic analysis of verrucous hyperplasia, verrucous carcinoma and squamous cell carcinoma as part of the clinicopathologic spectrum of oral proliferative verrucous leukoplakia: A literature review and analysis, Pathology - Research and Practice, Volume 215, Issue 12,20198.
8. Saba Q, Rathore A, Uma A, Waikhom S. Verrucous hyperplasia with moderate oral epithelial dysplasia: A case report. Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology. 2025 Sep 29;11(3):109–11
9. Thomas George Kallarakkal, Ramanathan A, Rosnah Binti Zain. Verrucous Papillary Lesions: Dilemmas in Diagnosis and Terminology. International Journal of Dentistry. 2013 Jan 1; 2013:1–6.
10. Hsue SS, Wang WC, Chen CH, Lin CC, Chen YK, Lin LM. Malignant transformation in 1458 patients with potentially malignant oral mucosal disorders: a follow-up study based in a Taiwanese hospital. Journal of Oral Pathology & Medicine. 2006 Dec 20;36(1):25–9.

HOW TO CITE: Abiksha C.*, Mathumala Subramanian, G. Keerthana, S. Marytresa Jeyapriya, M. Sathish Kumar, Verrucous Hyperplasia: More Than Just A Benign Overgrowth- A Review, Int. J. Sci. R. Tech., 2026, 3 (4), 575-579. https://doi.org/10.5281/zenodo.19615105