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Verrucous Leukoplakia - A Diagnostic Dilemma

Shaik Ali Hassan[1], Sumit Bhateja[1*], Neha Aggarwal[1] and Geetika Arora[2]

*[*1]Department of Oral Medicine & Radiology, Manav Rachna Dental College, Faridabad, Haryana, India.*

[2]Dept of Public health Dentistry, Inderprastha Dental College Ghaziabad, India.

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ABSTRACT

White lesions both physiologic as well as pathologic are relatively frequent in the oral cavity, the most common pathologic entity being oral leukoplakia (OL). There are many variants of OL, one of which is oral proliferative verrucous leukoplakia (OPVL). OPVL is a rare clinico-pathological entity, which is slow growing, long-term progressive lesion, but remains an enigmatic and difficult to define. The etiology of OPVL remains still unclear. This article describes the clinical aspects and histologic features of an OPVL case that demonstrated the typical behavior pattern in a long-standing, persistent lesion.

Keywords: Leukoplakia, Oral proliferative leukoplakia, Malignant transformation

INTRODUCTION

The term Leukoplakia originates from two Greek words: *leuko*, i.e., white and *plakia*, i.e., patch. As described by WHO in 1940, oral leukoplakia represents "a predominantly white lesion of oral mucosa that cannot be characterized as any other definable lesion clinically or pathologically, often associated with tobacco products, some of which may transform into cancer" [1].

In India OPVL prevalence is 1.5 to 4.3% [2,3]. It is seen in localized area in distribution, hyperkeratotic in nature and white in appearance due to wetting of keratotic patch [4].

We present a rare case of Verrucous Leukoplakia presenting as Verrucous Carcinoma, which can put a clinical dilemma due its clinical picture (**Figure 1**).

CASE REPORT

A 56 years old male patient (**Figure 1**), reported to Department of Oral Medicine and Radiology with a chief compliant of missing teeth in left upper back tooth region since 1 year Anamnesis was non-contributory. Patient reported a habit history of bidi smoking, about 5-6 times daily since 15-20 years and claims to be an occasional drinker since about 20 years. General Examination revealed patient was well built, well nourished, conscious, alert, oriented to time, place and person. No signs of pallor, icterus, cyanosis, clubbing, lymphadenopathy and edema are seen. Extraorally patient shows right and left submandibular lymphadenopathy, bilateral whitish lesions on the commissures of the lips that extend intraorally and have a triangular appearance. On intraoral inspection grayish white slightly raised lesion having a verrucous or papillary texture measuring 4 × 5 cm in size seen on the left buccal mucosa.

Figure 1. Extraoral photograph of patient.

Corresponding author: Dr. Sumit Bhateja, Reader (MDS), Department of Oral Medicine & Radiology, Manav Rachna Dental College, Faridabad, Haryana, India, Tel: 91-9891353960; E-mail: bhateja.sumit@gmail.com

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On right side a whitish plaque measuring 2 × 3 cm seen extending from upper buccal vestibule to the lower buccal vestibule is seen. Both lesions are extensions of white, triangular, raised, plaques present on the commissures of the lips extraorally. The lesions have an irregular surface with papillary like extensions. On palpation the lesion is non-tender, non-fluctuant, non-compressible, there is presence of network of white plaque which resembles verrucae (**Figures 2 and 3**). Based on history and clinical examination a provisional diagnosis of proliferative verrucous leukoplakia was made which was later confirmed histopathologica.

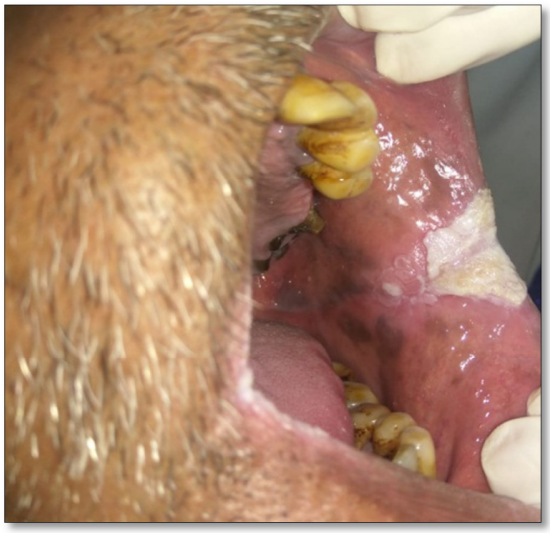

Figure 2. Intraoral picture showing white plaque on left vermilion border.

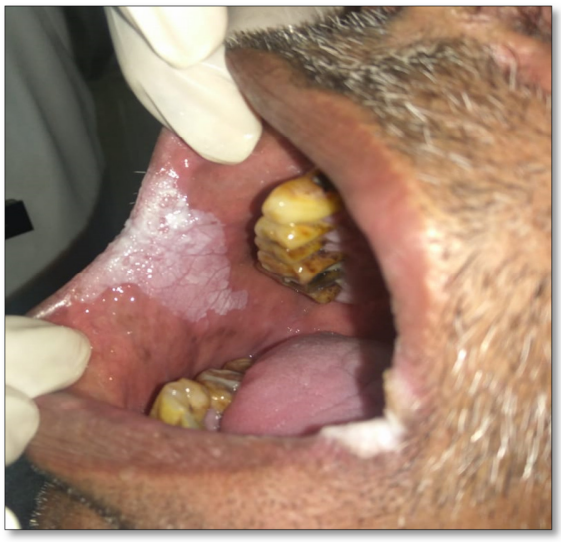

Figure 3. Intraoral picture shows whitish plaque on right buccal mucosa.

Tobacco Cessation Counseling was done and the patient was instructed to quit the habit. Patient was prescribed Cur Q Plus tablets (Curcumin 300 mg + Piperine 1.8 mg), three times daily for three months and the patient is placed on a fortnightly recall.

DISCUSSION

White lesions are a commonly seen in the oral cavity. Among those leukoplakia is most common. It becomes more exophytic with the development of the multiple keratotic plaques with roughened surface projection. It has got predilection for female than males and is present with the patients who don't smoke. As the disease progresses it can change clinically and microscopically identical to the verrucous carcinoma or squamous cell carcinoma.

Etiopathogenesis

Risk factors: The risk factors that contribute to the development of OPVL are:

Local factors:

- **Tobacco:** Tobacco is widely used in two forms:

Smokeless tobacco (chewable tobacco and oral use of snuff)

Smoking tobacco (cigar, cigarette, bidi and pipe)

a) Smokeless tobacco: Chewing of tobacco leads to leaching of several materials like tobacco tars and resins. These extracts of tobacco contain chemicals like nitrosonornicotine, nicotine, pyridine and picoline. These chemical constituents and an alkaline pH act as irritants and lead to alterations of mucosa in the form of sub lethal injury within the deeper layers of oral epithelium. This induces concomitant epithelial hyperplasia.

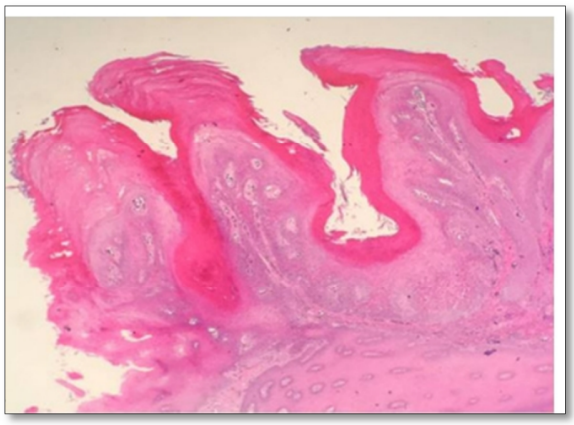

Figure 4. Histological staining shows mild hyperplasia.

b) Smoking tobacco: The smoke along with its constituents like polycyclic hydrocarbons, beta-napthylamine, nitrosamines, etc., and the heat generated in the oral cavity cause irritation in the oral mucosa, frequently seen as reddening and stripping of the mucosa. With continued smoking minute red and white striations are formed and the surface appears swollen.

- **Alcohol:** Prevalence of OPVL is higher in drinkers than in non-drinkers. This is mainly due to the action of alcohol to facilitate the entry of carcinogen into exposed cells and thus altering the oral epithelium and its metabolism.
- **Chronic irritation:** Continuous trauma is a factor that leads to development of OPVL. The source of irritation or trauma may be due malocclusion, ill-fitting dentures, sharp broken teeth, hot and spicy food, etc. The chronic irritation must be intense enough to induce surface epithelium to produce and retain keratin.
- **Candidiasis:** *Candida albicans* is commonly seen in association with leukoplakia. Tobacco smoking may lead to candidal colonization because of increased keratinization, reduced salivary immunoglobulin-A concentration or depressed PMN leucocyte function.

Regional and systemic factors:

- **Nutritional deficiency:** Sideropenic anemia and other nutritional deficiencies predispose to the condition.
- **Xerostomia:** These factors are salivary gland disease, radiation, etc.
- **Drugs:** Anticholinergic, anti-metabolic drug, etc.
- Syphilis [5].

Clinical classification:

- **Homogenous:** It is a completely whitish lesion
  - Flat: It is smooth surface.
  - Corrugated: Like a beach at ebbing tide.
  - Pumice like: With a pattern of fine lines.
  - Wrinkled: Like dry, cracked mud surface.
- **Non-homogenous**:
  - Nodular or speckled: Characterized by white spikes or nodules on erythematous base.
  - Verrucous: Slow growing, papillary proliferations above mucosal surface that may be heavily keratinized.
  - Ulcerated: Lesion exhibits red area at the periphery of white patches.
  - Erythroleukoplakia: Leukoplakia present in association with erythroplakia.

Clinical features:

- **Sex and age distribution:** Males are more affected. The mean age of diagnosis is 70 years [6].
- **Common sites:** Leukoplakia mostly occurs bilaterally mainly affecting the buccal mucosa, gingiva and vermilion border of lip. Lips, palate, maxillary mucosa, retromolar area, floor of mouth and tongue.
- **Color:** The lesion appears white or yellowish white due to hyper keratinized areas. As the lesion progresses, it become thicker and whiter, sometimes developing a leathery appearance with surface fissure.
- **Appearance:** Surface of the lesion shows a pappilomatous surface that feels rough on palpation.

Proliferative verrucous leukoplakia:

- It is an aggressive type of leukoplakia with very high rate of malignant transformation, but oral carcinoma can develop from any leukoplakia.
- It is seen more in elderly women. Women to men ratio is 4:1.
- Proliferative verrucous leukoplakia appears as a single or multifocal growth involving several oral sites. Most commonly buccal mucosa affected in women and tongue in men.
- Development of proliferative verrucous leukoplakia starts as a simple hyperkeratosis without epithelial dysplasia, verrucous carcinoma and then carcinoma.
- Proliferative verrucous leukoplakia has association with human papilloma virus infection. HPV-16 infection may play an important role in these lesions.
- Among different forms of leukoplakia, proliferative verrucous leukoplakia has high malignant transformation. In a study of 54 cases of proliferative verrucous leukoplakia, Silverman and Gorsky found out that 70.3% subsequently developed squamous cell carcinoma [7].

Differential diagnosis of oral proliferative verrucous leukoplakia [8]

- Lichen Planus: Differentiated by Wickham's Striae and multiple lesions.
- Syphilitic mucus patches: Features like split papillae and condyloma latum may be present.
- White sponge nevus: Present soon after birth and is widely distributed all over the mucus membrane.
- Discoid lupus erythematosus: Central atrophic area with small white dot and radiating white straie.
- Candidiasis: Differentiated as the candidal lesions are scrapable.
- Verrucous carcinoma: Lesions are elevated (exophytic).

Histopathology

PVL shows a variable microscopic appearance, depending on the stage of the lesions. Early PVL appears as a benign hyperkeratosis that is indistinguishable from other simple leukoplakic lesions. With time, the condition progresses to a papillary, exophytic proliferation that is similar to localized lesions of verrucous leukoplakia. In later stages this papillary proliferation exhibits down growth of well-differentiated squamous epithelium with broad, blunt rete ridges. This epithelium demonstrates invasion into the underlying lamina propria, in this stage it is indistinguishable from verrucous carcinoma. In the final stage the invading epithelium becomes less differentiated, transforming into the condition called squamous cell carcinoma. Because of the variable clinical and histopathologic appearance of PVL, careful correlation of the clinical and microscopic findings is required for diagnosis [9,10].

Treatment

- Prohibition of smoking: Patient should be asked to cease smoking with immediate effect.
- Removal of chronic irritant: Sharp points from the dentures should be removed along with enameloplasty of sharp teeth.
- Elimination of other etiological factors: Factors like syphilis, alcohol, dissimilar metal restoration, etc., should be eliminated.
- Carbon dioxide laser, radiation, topical bleomycin solution, oral retinoids, beta-carotene and systemic chemotherapy have all failed to achieve permanent cure.
- Laser ablation reportedly has been successful in a very small group of patients followed for 6-178 months.
- Topical photodynamic therapy also may prove useful; it causes relatively low morbidity and no scarring and multiple mucosal sites can be treated simultaneously.
- The use of antioxidant nutrients and vitamins has not been reproducibly effective in management. Programs have included single and combination dosages of vitamins A, C and E; beta carotene; analogues of vitamin A; and diets that are high in antioxidants and cell growth suppressor proteins [11,12].

CONCLUSION

Oral Proliferative verrucous leukoplakia is a rare and aggressive form of disease which requires special attention. The aim/intention of this case report is to report the clinical case so as to sensitize the oral physicians.

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