Proliferative Verrucous Leukoplakia: A Case Report

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ABSTRACT:

Proliferative verrucous leukoplakia (PVL) was first described in 1985 by Hansen et al. It is a rare type of oral leukoplakia,. Female patients & elderly patients over 60 years of age are most commonly affected. We present herein a case of oral proliferative verrucous leukoplakia (OPVL) and discuss this relatively rare entity in light of current information. Proliferative verrucous leukoplakia (PVL), is a rare potentially malignant oral mucosal disorder. It has a high rate of transformation to oral squamous cell carcinoma (SCC) and verrucous carcinoma (VC). Most commonly affected sites are buccal mucosa, gingiva, alveolar ridges and tongue. Initially it appear as focal clinical hyperkeratosis that progresses to become a wide multifocal disease with gross exophytic features.

Keywords: Hyperkeratosis, Oral leukoplakia, Oral mucosal lesions, Oral proliferative leukoplakia, Potentially malignant disorder.

INTRODUCTION

Oral cancer is amongst the most commonly occurring cancer in the world.[1] Many etiological factors are responsible for oral squamous cell carcinoma (OSCC). Premalignant lesions, especially leukoplakia are associated with OSCC. Leukoplakia is defined as "a white plaque of questionable risk, having excluded(other) known diseases or disorders that carry no increased risk for cancer".[2] The World Health Organization has described leukoplakia as a "precancerous lesion". However, more recently the term "potentially malignant,''is used for all "premalignant" and "precancerous" lesions.[3] Hansen et al in 1985 first described Proliferative verrucous leukoplakia (PVL)[3] It is a a long term progressive condition, which develops initially as a white plaque of hyperkeratosis that eventually becomes a multifocal disease. The premalignant capacity of PVL can range between 40 and 100% in a follow-up period of 4.4 to 11.6 years.[3] Thus early diagnosis and long term follow up is important to prevent malignant transformation.

CASE REPORT

A male patient aged 64 years reported to Department of Oral Medicine & Radiology with chief complaint of white patch seen on left side of tongue since last two years. Initially it was small and gradually increased to the present size. There was no pain or burning sensation on the tongue due to the patch. No relevant past dental/medical or family history was reported. Personal history revealed patient consumed mixed diet and had deleterious habit of chewing tobacco 4-5 times a day since last 40 years He keeps tobacco inside right & left buccal vestibule and also below tongue for 5-10 minutes and then spits it out.

Intra-oral examination revealed the well formed maxillary ridge & atrophic mandibular ridge. Examination of tongue (Figure 1) shows a solitary, irregular in shape plaque,

which was white in colour with multiple projections were seen on lateral border and ventral surface of left side of the tongue. It was 4 x 3 cm in dimension. The surface shows minute (approximately 1 mm) tapered projections which were white in colour and gave a cauliflower like appearance. Small white flecks anterior to this lesion were also seen. Pigmentation seen on ventral surface of tongue. Surrounding area of tongue appears normal. On palpation all inspectory findings were confirmed. It was non tender, non scrapable and non stretchable and soft to firm in consistency. Induration was absent. Co-relating the history and clinical findings, the provisional diagnosis made was, Verrucous leukoplakia on left lateral border and ventral surface of tongue. Differential diagnosis considered were, verrucous squamous cell carcinoma, chronic hyperplastic candidiasis, Hairy leukoplakia.

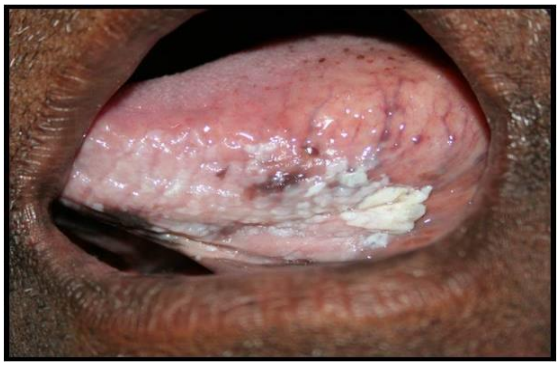

Figure 1: Intraoral image showing a solitary, diffuse plaque of white color on left lateral border & ventral surface of tongue

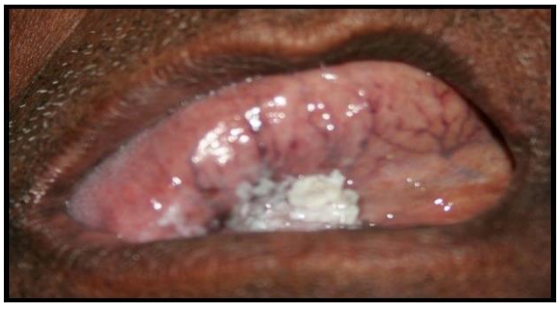

Figure 2: on second visit an intraoral image before biopsy shows small white flecks had reduced in size.

The treatment plan proceeded as follows: 1) Patient was counseled and educated about the deleterious effects of tobacco and motivated to quit chewing tobacco. 2) The patient was put on antioxidant Lycopene 5 mg/day for 3 months 3) Candid mouth paint topical application three times in a day for 8 days 4) Oral multivitamins were given 5) The patient was subjected to routine blood investigations 5) A week later excisional biopsy of the lesion (Figure 2) was done under local anaesthesia inclusive of the surrounding normal tissue and the patient was prescribed systemic antibiotics and antiseptic mouth wash. 6) The biopsy specimen was sent for histopathological examination. The patient was recalled after a week for reevaluation. Re-evaluation after 1 week showed uneventful wound healing.

HISTOPATHOLOGY

(Fig 3 & Fig 4) Histopathological examination revealed stratified squamous epithelium which was hyperkeratotic in nature. Whirling of epithelial cells present in few areas. The epithelium shows dysplastic features like basilar hyperplasia, increased nuclear cytoplasmic ratio, nuclear pleomorphism, abnormal mitotic activity, and prominent intercellular bridges. Irregular stratification of epithelial cells is seen. Rete ridges are broad and bulbous. The basement membrane is intact. The underlying connective tissue shows collagen fibers interspersed with fibroblasts. The inflammatory cell infiltrate is present chiefly lymphocytes.

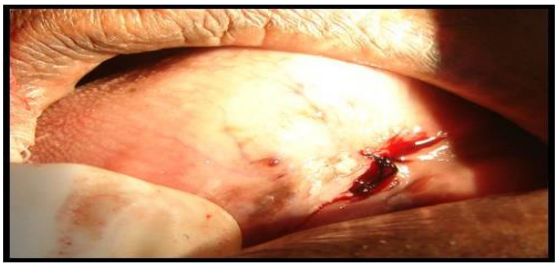

Figure 3: Intraoral image showing excisional biopsy

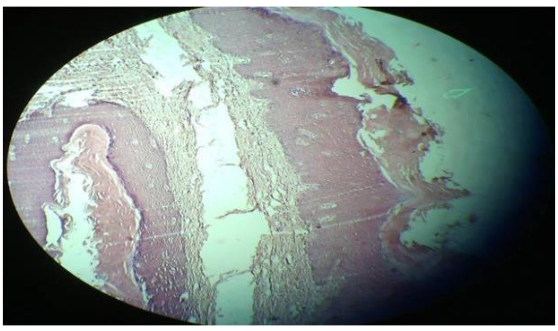

Figure 4: Histological photomicrograph showing parakeratinised stratifies squamous epithelium with broad & bulbous rete ridges (H & E stain, 10 X magnifications)

By combining the characteristics of the oral lesions and histopathological changes, a final diagnosis of Proliferative Verrucous Leukoplakia was made.

The patient was followed up at regular intervals with no recurrence for about a year beyond which the patient was lost to follow up.

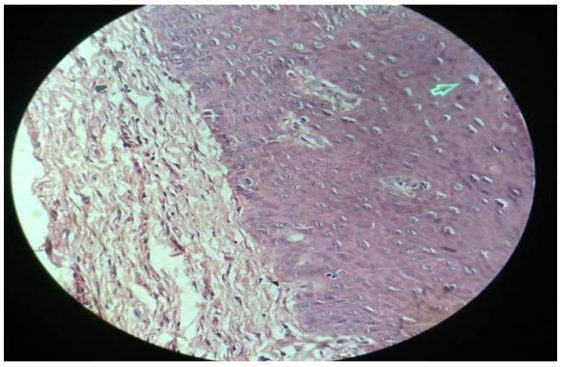

Figure 5: Histological photomicrograph showing dysplastic features like basilar hyperplasia, increased nuclear cytoplasmic ratio, nuclear pleomorphism, abnormal mitotic activity (H & E stain, 40X magnification)

DISCUSSION

The exact etiology of OPVL is not known. Tobacco is frequently absent as a known risk factor as OPVL occurs both in smokers and nonsmokers.[4] An association has been reported between human papilloma virus (HPV) and OPVL. Between 0% and 89% of OPVL are reported to be HPV positive[5] especially for HPV types 16 and 18. It has also been occurred in association with EpsteinBarr virus [5] or candida infection.[6] The proliferative effect of OPVL was explained on the basis of the high rate of field cancerization existing in OPVL patients.[7] However, none of these studies have given exact a etiopathogenesis of OPVL.

Hansen et al[3] suggested 10 histologic stages ranging from normal mucosa, hyperkeratosis, verrucous hyperplasia, verrucous carcinoma, papillary squamous cell carcinoma, and less well differentiated squamous cell carcinoma. Later, Batsakis et al[8] modified Hansen's proposal to four stages, eliminating the papillary squamous cell carcinoma, which he considered to be independent of PVL. Therefore, histopathologically, PVL starts as simple hyperkeratosis, which can progress to verrucous hyperplasia, verrucous carcinoma, and even OSCC, whether in situ or infiltrating.[3] Management of PVL has been described as difficult and the prognosis as poor.

Treatment regimens include surgery, radiation, chemotherapy and retinoids, carbon dioxide laser vaporization, and cryotherapy.[3,9,10]

CONCLUSIONS

OPVL is a rare, but highly aggressive form of OL. Therefore the clinician should be able do earliest possible diagnosis and total excision of this lesion. Even after surgical management the care should be taken to follow up these cases for a long time because recurrence rate of OPVL is high. They are also known to undergo malignant transformation.

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