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Proliferative Verrucous Leukoplakia: A Need for Its Diagnostic Acumen.

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Abstract: *In day today practice we see multiple numbers of cases of white lesions in the oral cavity; oral leukoplakia is one of prevalent white lesion.Proliferative verrucousleukoplakia(PVL) is exceptional and aggressive type of oral leukoplakia.A multifocal or diffuse extension makes the lesion inscrutable. In case of proliferative verrucous leukoplakia it is important to give a "clinicopathological" correlation. Because histopathologically it varies from an epithelial hyperplasia with hyperkeratosis to oral squamous cell carcinoma(OSCC) and variants of OSCC, whereas clinically it might vary from a simple homogenous white patch to an aggressive appearance of carcinoma. This leads to a diagnostic dilemma. One of the aspects elaborated in this paper is relation between PVL and field cancerization. With the detailed review of literature the present paper focuses on a case which came to the department.*
Key words: *Proliferative verrucous leukoplakia, field cancerization, white patch.*

I. Introduction

Hansen et al in 1985 studied a different type of leukoplakia in 30 patients and termed it as "Proliferative verrucous leukoplakia" .Proliferative Verrucous Leukoplakia (PVL) is described by the World Health Oraganisation (WHO) as a rare, but distinctive, high risk clinical form of oral precancerous lesions, without a single pathognomonic criterion but with characteristic combined histologic and clinical features and behavior [1] Sapp has defined PVL as "Diffuse white and/or papillary("warty") areas of oral mucosa resulting from varying degrees of epithelial hyperplasia; has potential to develop into verrucous carcinoma or well differentiated oral squamous cell carcinoma".[2] Silverman and Gorsky studied 54 patients and 17 of which were from the original case reported by Hansen et al showed 70 % of transformation into carcinoma(mean, 7.7 years from initial diagnosis ; range, 1-27 years) a second malignancy developed in another PVL site in 31.5% of the cases. In the final report by Hansen et al 87% patients transformed to Squamous cell carcinoma in the follow up period which was extended in 20 years in some patients.

II. Case Report

A 45 year female patient reported to the department of Oral Pathology and Microbiology with the chief complaint of white coloured patches at front region of jaw which were painless. There was no past dental medical or family history. Patient was having history of mishri application thrice daily since 20 years and she was having history of chewing betal nut twice daily after meals twice daily.

Clinical examination:

Clinical examination revealed that there were few homogenous white patches on labial mucosa, gingival from first quadrant to second quadrant and anterior gingival of mandible(Fig 1). Patient was advised for biopsy and other routine investigation but she refused. Again after a month patient came back this time lesions were transformed to verrucous pattern (Fig 2) and were involving right and left buccalmucosa.On palpation the growth was firm, non tender, non compressible an non fluctuant. Based on first visit and second visit clinical history provisional diagnosis was given as oral proliferative verrucous leukoplakia.

Patient was subjected for following investigation

1) Toludine blue staining was done and positive area was excised and sent for histopathological investigation.
2) Total blood hemogram was done and all counts were within normal limits.
3) ELISA was done and it was negative

Histopathological features:

Histopathology revealed hyperkeratotic epithelium and at few places keratin plugging was seen. Dysplastic features were seen such as basilar hyperplasia, hyperchromatic nuclei, cellular and nuclear pleomorphism and loss of stratification. Few focus were showing whirling of epithelial cells suggestive of

epithelial pearl. The connective tissue showed thick collagen bundles which were interspersed with fibroblasts. Few dialated, engorged blood vessels were also present. Inflammatory cell infiltrate was seen. According to Hansen et al 1-4 criteria histopathology report was given as hyperkeratosis with moderate dysplasia.
The clinical and histopathological report were suggestive of Oral Proliferative verrucous leukoplakia.(Fig 3
Treatment:Patient was too reluctant that after continuous motivation for treatment and guiding her about the consequences, unfortunately she refused for treatment.

III. Discussion

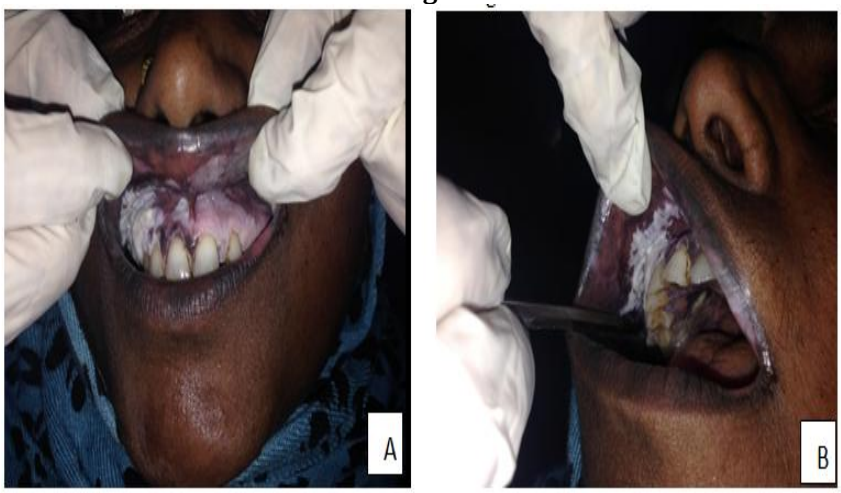
Hansen et al in 1985 studied a different type of leukoplakia in 30 patients and termed it as "Proliferative verrucous leukoplakia" .Proliferative Verrucous Leukoplakia (PVL) is described by the World Health Oraganisation (WHO) as a rare, but distinctive, high risk clinical form of oral precancerous lesions, without a single pathognomonic criterion but with characteristic combined histologic and clinical features and behavior [1] Sapp has defined PVL as "Diffuse white and/or papillary("warty") areas of oral mucosa resulting from varying degrees of epithelial hyperplasia; has potential to develop into verrucous carcinoma or well differentiated oral squamous cell carcinoma".[2] Silverman and Gorsky studied 54 patients and 17 of which were from the original case reported by Hansen et al showed 70 % of transformation into carcinoma(mean, 7.7 years from initial diagnosis ; range, 1-27 years) a second malignancy developed in another PVL site in 31.5% of the cases. In the final report by Hansen et al 87% patients transformed to Squamous cell carcinoma in the follow up period which was extended in 20 years in some patients[3]

Proliferative verrucous leukoplakia is considered to be a "preneoplastic process" of the oral mucosa.[4] Our case fulfilled criteria of Hansen et al. Improbable like a leukoplakia PVL has tendency for females, female to male ratio is about 4:1 with verrucous hyperplasia with or without dysplastic features and which leads to the malignancy. PVL has 100% rate of malignant transformation.[3]Urizar suggested a better term "Proliferative multiple leukoplakia".[5] The etiology for PVL is unclear however the role of tobacco and alcohol has been documented as a potential causes of these lesions. [6]Jose Bagan et al studied the association between the PVL and Human papilloma virus but they found that there is no association between both.[7]
There are various grading systems of PVL and they are enlisted in the (table 1).[8]
Proliferative verrucous leukoplakia is an aggressive lesion which may misdiagnose by a pathologist on only initial histopathological appearance. Thus it's important to understand that it's a fatal lesion where there is 100 % risk of malignant transformation and treatment is not substance. So reporting should be clinicopathological and patient should be kept under observation.

IV. Conclusion

Proliferative verrucous leukoplakia is an aggressive lesion which may misdiagnose by a pathologist on only initial histopathological appearance. Thus it's important to understand that it's a fatal lesion where there is 100 % risk of malignant transformation and treatment is not substance. So reporting should be clinicopathological and patient should be kept under observation.

V. Figures



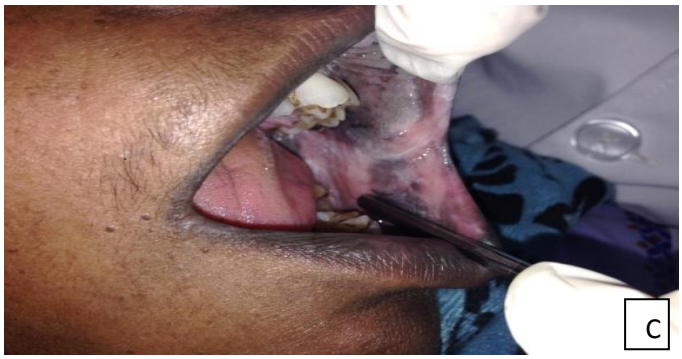


Fig 1. A. First visit a)Homogenous white patch on labial and buccal mucosa.b) White patch on buccal mucosa.c) white striations on buccal mucosa.

Fig 2. B) Second Visit a) Heterogenous verrucous, whart, shiny and proliferative white patches on labial and buccal mucosa b) Homogenous white patches on left buccal mucosa.

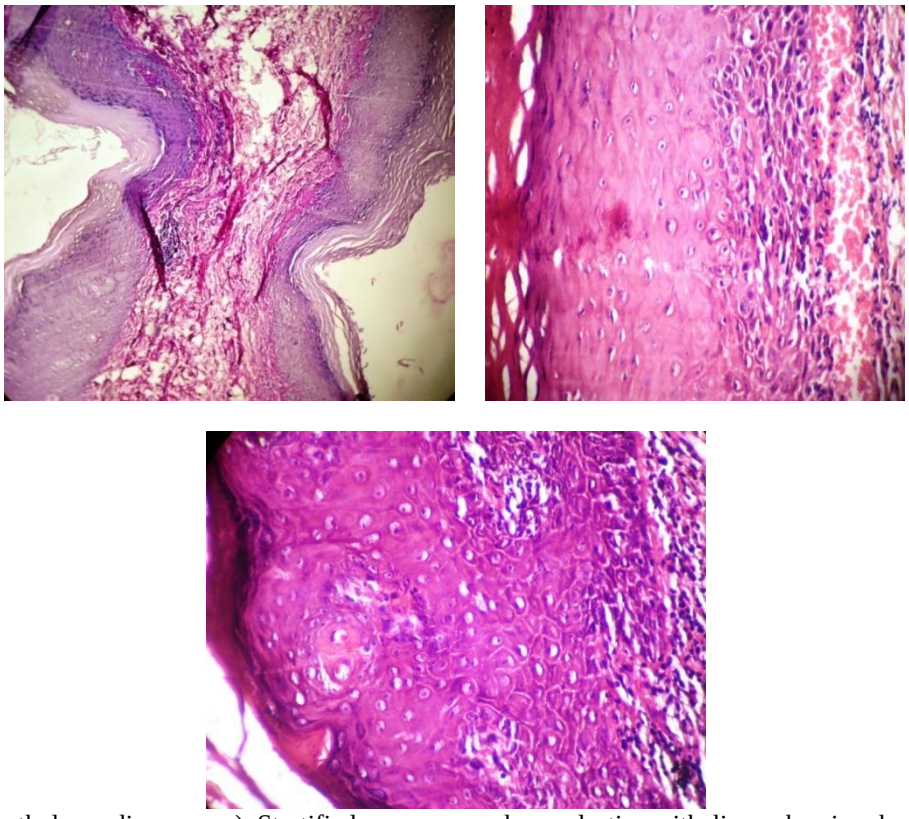


Fig 3. Histopathology diagrams a) Stratified squamous hyperplastic epithelium showing keratin pluggin, hyperkeratosis and subepithelial inflammatory infiltrate.(Hematoxylin and Eosin stained scanner view) b) Stratified eithelium with dysplastic features and subepithelial inflammatory infiltrate. c) Loss of stratification of cells (b and c Hematoxylin and Eosin stained. 40x magnification)

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