VERRUCOUS CARCINOMA IN ASSOCIATION WITH PROLIFERATIVE VERRUCOUS LEUKOPLAKIA: A CASE RARE ENTITY

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ABSTRACT

Verrucous carcinoma (VC) also known as Ackerman's tumour is a low grade variant of oral squamous cell carcinoma (OSCC) commonly affecting the buccal mucosa of oral cavity. Clinically VC has a proliferative cauliflower-like appearance. Smokeless tobacco is found to be the most etiological factor for VC although other potentially malignant lesions like proliferative verrucous leukoplakia (PVL)may act as a predisposing factor.PVL is characterized by malignant transformation in nearly 74% of the cases and seen mainly in older women without any habits. It begins as a simple slow growing persistent leukoplakia that tends to spread, become multifocal and affects the gingiva frequently. We report a rare case of VC associated with PVL in 59 year old woman without any habits.

Key words: Verrucous carcinoma;Proliferative Verrucous Leukoplakia;Ackerman's tumour; Oral Cavity.

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Introduction

Verrucous Carcinoma (VC), is a low grade variant of squamous cell carcinoma, was first described by Lauren V Ackermann in 1948 so it was known as Ackermann's Tumor".[1] It has pathognomonic clinical appearance, biological behavior and microscopic features. The neoplasm appears exophytic papillary in nature with a rough, pebbly surface. Majority of the patients give a history of chewing tobacco, may also have poorly fitting dentures, carious and jagged teeth.[2] A high-risk potentially malignant disorder, PVL beginsas a simple slow-growing, persistent hyperkeratosis thattends to spread and become multifocal and, in timedevelops exophytic, wart-like, or erythroplakic areasthat become carcinomas.[3] We herewith report a case of VC associated with PVL in 59 year old woman with no history of any habits or local etiological factors noted.

Case report

A 59 old female patient reported to the department of Oral medicine and Radiology with a chief complaint of white growth on the right aspect of cheek and tongue as well as white changes on the lower gum noticed in two months. Detailed history taking revealed that white lesions on right buccal mucosa appeared firstly which progressed to tongue and mandibular gingiva. It was slowly progressive without any discomfort reported other than mild xerostomia. Patient had consulted a dentist and was given candid gum paint and vitamins. Initially patient noticed some change, but the lesion was progressive. Medical history revealed that she was under antihypertensive and antigastritic medication. Uneventful extraction history was also reported. She never had any habit history.

On clinical examination right buccal mucosa near commissure and lateral border of posterior third of tongue were noted with white exophytic growth having warty appearanceof about 1x 2 cm in size. The buccal mucosal lesions are associated with nonscrappable white plaque lesions extend to lower labial gingiva till left lower canine. (Figure-1) A provisional diagnosis of PVL was given and differential diagnoses considered were VC, OSCC, verruca vulgaris and hyperplastic candidiasis.

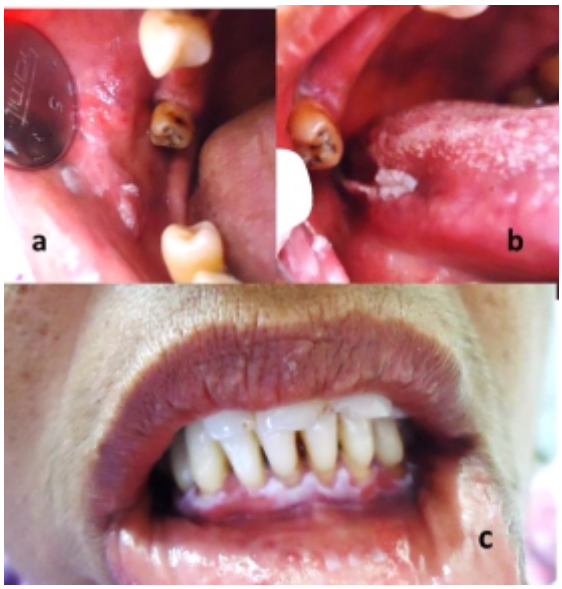

Figure1: Clinical photographs showing a.white exophytic growth having warty appearance on right buccal mucosa b. white exophytic growth having warty appearance on lateral border of posterior third of tongue c.white plaque lesions on lower labial gingiva .

Incisional biopsy was done from exophytic growth of right buccal mucosa and microscopically section showed hyperparakeratotic stratified squamous epithelium with parakeratin plugging and broad elongated rete ridges with pushing borders. Minimal dysplastic change could be noted. Underlying minimal connective tissue exhibited with intense chronic inflammatory cell infiltrate. (Figure-2)Correlating clinical and histopathological features the given

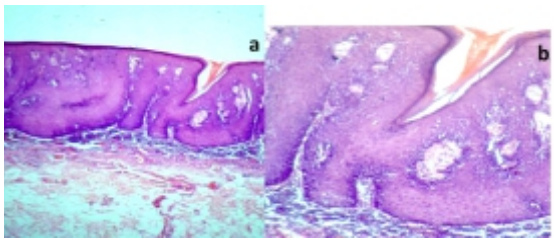

Figure-2: Photomicrograph showing section with hyperparakeratotic hyperplastic stratified squamous epithelium with parakeratin plugging, broad elongated rete ridges with pushing borders, minimal dysplastic changes and intense chronic inflammatory cell infiltrate, a(4x) & b(10x)

specimen was confirmed the diagnosis of VC. Patient underwent laser surgery later.

Discussion

VC is a less common tumour, represents 4.5-9% of oral squamous-cell carcinomas and its aetiologyis not yet well defined.[1] Smokeless tobacco users are at a higher risk of developing VC than non-tobacco users. Human papilloma virus has been identified in the cells of this tumour but is still not considered as a causative factor.[1] The most common sites reported are the buccal mucosa, gingiva and the alveolar ridge; other sites of involvement include the palate, floor of the mouth and lip. VC is characterized by well circumscribed cauliflower-like exophytic growth with a cleft, pale, warty or fungating appearance, attached by a broad base. It is clearly demarcated from the adjacent mucosa with a pebbly mamillated surface. VC is locally aggressive and with no metastatic potential.[1] The most important differential diagnoses of VC includes: (i) OSCC showing verrucoid features, (ii) PVL (iii) verrucous hyperplasia (iv) pseudo-epitheliomatous hyperplasia, (v) verruca vulgaris, and (vi) keratoacanthoma. [2]

The histopathological features of VC includes hyperkeratotic hyperplastic epithelium with parakeratin plugging and bulbous "elephant feet" likerete ridges showing endophytic growth pattern with pushing borders, typically showing minimal or absent cytological atypia and intact basement membrane.[2, 4] If at all focal atypia or dysplasia is evident, it must be limited to the basal layer of epithelium. Lympho-plasmacytic inflammatory reaction is marked especially, in cases, where keratin has plunged deep into the connective tissue inducing foreign body granuloma formations.[2] All these feature were evident in our case.

Diagnosis of verrucous carcinoma is difficult and its reporting needs experience.[5] Verrucous hyperplasia or leukoplakia is theinitial pathologic diagnosis in 60% ofcases. Deepincisional biopsy or wide excision is needed correct diagnosis of VC. The prognosis of verrucous carcinoma is better than conventional malignant tumours. Various treatment modalities include surgery, chemotherapy, or combination of these with photodynamic therapy. [2]

PVL is a rare condition first described by Hansen et al. in 1985 and it's an aggressive form of oral idiopathic leukoplakia.[6] An association with human papilloma virus infection has been suggested. PVL appears to resist to all attempts at therapy and often recurs. The World Health Organization also described the PVL with a high rate of malignant transformation, 74%.[3] PVL is a distinct clinical form of oral leukoplakia which in turn is defined by its progressive clinical course, changing clinical and histopathologic features, and potential to develop into cancer, defined by Cabay et al, 2007. [7]

PVL commences as one or more homogeneous leukoplakic areas and later, the lesions enlarge and affect other locations, especially the gingivae. The buccal mucosa, gingiva, and alveolarridges were most often affected.[3] The diagnosis of PVL based on clinical data is often late due to its progressive evolution fro mhomogeneous leukoplakic areas spreading to many different locations and the appearance of verrucous forms takes time. Histopathology may help, but it depends on the site biopsied, the stage of the disease, and presumably by other factors. Murrah and Batsakis (1994) and Batsakis et al (1999) proposed four stages of histopathological development with PVL: hyperkeratos is without epithelial dysplasia, verrucous hyperplasia, VC and OSCC.[3] Lesions are managed with surgery, carbon dioxide laser, and photodynamic therapy.[6]

Conclusion

Cases of VC, PVL, verrucous hyperplasia, and verrucous keratosis are clinically indistinguishable; biopsy should be advised as early as possible for the sake of early diagnosis and prompt treatment. VC associated with PVL may be an indication of "field cancerization" and can lead to multiple recurrences, so such patients should to be kept under regular follow-up.

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