Case Report

VERRUCOUS HYPERPLASIA – A RARE CASE REPORT

Raju MS, Mehrotra V, Mishra G, Sajid Z

ABSTRACT: Oral verrucous hyperplasia is a rare clinico-pathological entity, which is soft slow growing, long-term progressive soft tissue lesion, but remains an enigmatic and difficult to define. The etiology of Oral verrucous hyperplasia remains still unclear. Tobacco use does not seem to have a significant influence on the appearance of Oral verrucous hyperplasia. These lesions may occur both in smokers and non-smokers. It is observed more frequently in women and elderly patients over 60 years at the time of diagnosis. The buccal mucosa and tongue are the most frequently involved sites. It develops initially as a white plaque of hyperkeratosis that eventually becomes a multifocal disease with confluent, exophytic and proliferative features. Various published case series have presented Oral verrucous hyperplasia as a disease with aggressive biological behaviour due to its high probability of recurrence and a high rate of malignant transformation. Prognosis is poor for this seemingly harmless-appearing white lesion of the oral mucosa. This article describes the clinical aspects and histologic features of an Oral verrucous hyperplasia case that demonstrated the typical behaviour pattern in a long-standing, persistent lesion and discusses this relatively rare entity in light of current information.

Key words*:* Premalignant lesions, verrucous hyperplasia, verrucous carcinoma

Introduction

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)[1] and differentiated from verrucous carcinoma. They separated these entities based on lack of invasive growth in the verrucous hyperplasia that is entirely superficial to adjacent normal epithelium. Verrucous hyperplasia is not only encountered in oral mucosa but also in sinonasal and laryngeal mucosa. Hansen *et al.* (1985)[2]described proliferative verrucous leukoplakia a type of non-homogeneous leukoplakia which is irreversible, slow growing with highest potential of malignant transformation and resistant to treatment. It is a clinical term and histopathologically it may show simple hyperkeratotic lesion to verrucous hyperplasia, verrucous carcinoma or well differentiated squamous cell carcinoma. However enlarged lymphnode in this case may not be correlated with the same lesion.

CASE REPORT

A 65-year-old male patient [Figure 1] reported to the Department of Oral Medicine and Radiology with the chief complaint of loosening of teeth in lower left jaw since 2 years and wanted removal of mobile teeth, his past medical history, including his family history was unremarkable. Patient's dental history revealed extraction of multiple mobile teeth 2 years back. Patient gave a history of tobacco chewing since 20 years 2-3 times/day and also having the habit of bidi smoking 2-4 bidi/day but has quit the habit completely since last 5 years.

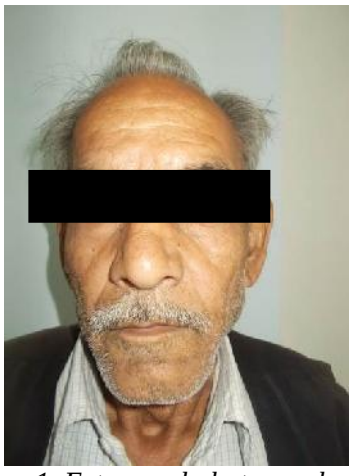

Figure 1: Extra-oral photograph of the patient

CLINICAL EXAMINATION

Extra-oral examination revealed left submandibular lymph node enlarged, mobile & non tender on palpation. Intra-oral examination [Figure 2] revealed multifocal tumefactive (three in number) is seen on left buccal mucosa i.r.t 36,37,38

anteroposteriorly it extends from approx 0.5 cm away from left commisural area of mouth upto 38 region & superoinferiorly 1 cm below the occlusal line of lower jaw and at the level of occlusal line of upper jaw of left side with cauliflower like appearance pebbled surface which is irregular in shape & the overlying mucosal color looks the same as normal adjacent mucosa.

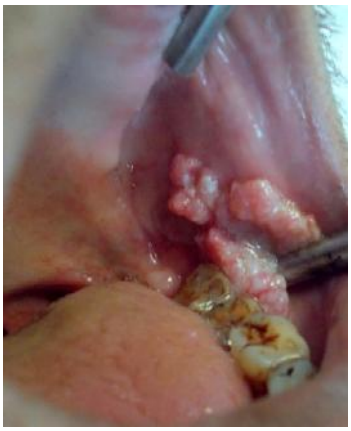

Figure 2: Intra - oral photograph of patient

On the basis of history & clinical examination a provisional diagnosis of verrucous carcinoma was given along with the differential diagnosis of irritational fibroma, verruca vulgaris, verrucous leukoplakia, papillary squamous carcinoma, verrucous hyperplasia & squamous papillomatous hyperplasia. The patient was subjected to following investigations to reach to a final diagnosis: OPG, Complete hemogram, Incisional biopsy. Orthopantomograph reveals severe bone loss in relation to 36, 37 & 38 and also showing multiple missing teeth [Figure 3]. In complete hemogram all the values were in the normal range. An incisional biopsy was performed from left buccal mucosa and the tissue was sent for histopathological analysis [Figure 4].

On histopathological examination the sections revealed the presence of hyperplastic, hyperparakeratinized stratified squamous epithelium showing epithelium showing acanthosis. Retepegs were broad and did not show pushing borders. There were no features of parakeratin plugging which is characteristic of verrucous carcinoma. These were no features of dyspalsia.

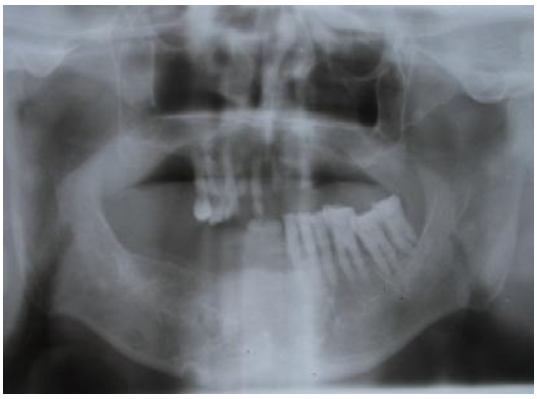

Figure 3: Photograph of Orthopantomogram

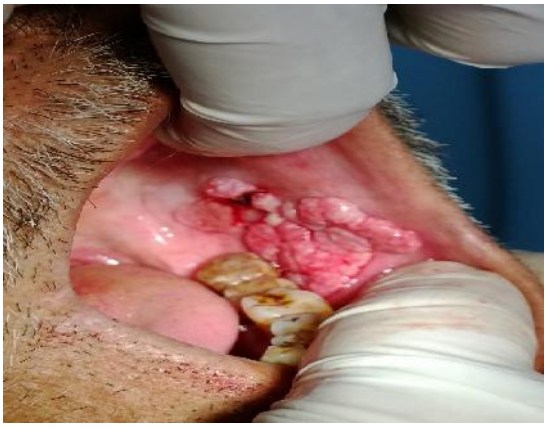

Figure 4: Post biopsy photograph.

Underlying connective tissue stroma showed infiltration of chronic inflammatory cells dilated & proliferated blood capillaries engorged with blood. Based on these features a diagnosis of Verrucous Hyperplasia was given [Figure 5].

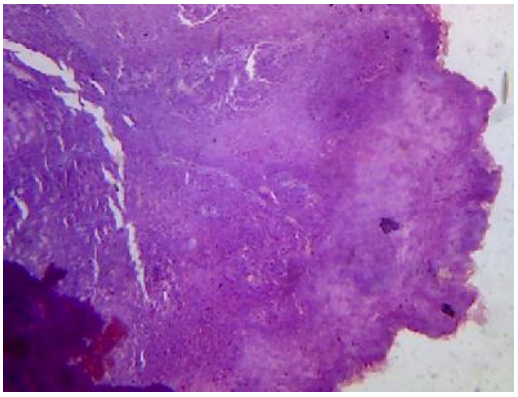

Figure 5: Photomicrograph of Verrucous Hyperplasia

TREATMENT AND FOLLOW-UP

The entire lesion was surgically excised using electrocautery and extraction of mobile teeth. The tissue excised was sent for histopathological re-evaluation, which confirmed the previous histopathological diagnosis. Later the follow-up was not possible as patient did not report back.

DISCUSSION

Verrucous hyperplasia probably represents a morphological variant of verrucous carcinoma by Slootwage and Muller (1983)[3]. Essential features in distinguishing verrucous hyperplasia from verrucous carcinoma, is the location of the thickened epithelium with respect to adjacent normal appearing epithelium. In verrucous hyperplasia most of the hyperplastic broadened rete ridges lay above the adjacent normal epithelium while verrucous carcinoma on contrary exhibits a downward growth pattern of otherwise similar rete ridges. Shear and Pindborg (1980)[1] stated that verrucous carcinoma often retract normal epithelium down with them into the underlying connective tissue.

For that distinction it is very necessary that biopsy should include adjacent normal epithelium. Proliferative verrucous leukoplakia (PVL) is clinically a non-homogeneous leukoplakia which often confused with verrucous hyperplasia.[4] Hansen *et al* (1985)[2] suggested six histological stages ranging from normal mucosa, hyperkeratosis, verrucous hyperplasia, verrucous carcinoma, papillary squamous cell carcinoma and less well differentiated squamous cell carcinoma. Malignant potential of PVL is more than other types of leukoplakia.

There are essentially two patterns. The first, which may be described as the "sharp" variety, comprises long, narrow, heavily keratinized verrucous processes. As a result of the heavy keratinization, the lesions are white. It is this variety that has probably been referred to as verrucous leukoplakia by a number of authors. Areas of homogeneous leukoplakia may be present adjacent to the lesion and elsewhere in the mouths of the same patients. The second variety, which we refer to as the "blunt" variety, consists of verrucous processes that are broader and flatter and not heavily keratinized, although a number of leukoplakic areas may also be seen in parts of the lesion. Leukoplakia is an important component of the lesion and of the mucosa elsewhere in the mouths of the same patients.

Histopathologically blunt variety predominates sharp variety as opposed to sharp variety reported by Shear and Pindborg .[1]This difference may be attributed to lesser sample size in present study. Hyperorthokeratinization was predominant in sharp variety and parakeratinization was predominant in blunt variety. Increase in thickness of stratum spinosum is responsible for verrucous type morphology of lesion. As proposed by Shear and Pindborg considerable acanthosis with broadened rete ridges causes deprivation of distant epithelial cells from blood supply and becomes edematous 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.[1]

Dysplasia is commonly seen in verrucous hyperplasia which is consistent with finding by Shear and Pindborg study (66%).[1] Greer et al, (1990)[5] suggested the association of Human papilloma virus (HPV) with development of verrucous hyperplasia.[5] Sakurai et al, (2000)[6] concluded that C-erb B-3 protein expression was an index of malignancy during progression from Verrucous Hyperplasia to Verrucous Carcinoma and Squamous cell carcinoma.[6] Wu *et al*, (2002)[7] stated that the expression of p53 and EGFR levels could be use as marker while differentiating verrucous hyperplasia from verrucous carcinoma and squamous cell carcinoma.[7]

CONCLUSION: Occasionally varices, vascular lesions like AV malformations and lymphangioma on oral mucosa mimics the above mentioned lesions however to rule out ultrasound with colour Doppler may need to be advised in some cases. Verrucous lesions of the oral cavity are distinct clinical entity with varied histopathology a surgical excision with wide margin is necessary for functional outcome.

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