INTRODUCTION:

PVL is an entity with a high tendency to develop OSCCs, the frequency ranging from 60% to 100%. Its rate of malignant transformation is extremely high. The characteristics of its clinical and pathological progress are considered vital bases for the diagnosis of PVL because there are no particular differences between the pathological changes of PVL and those of oral verrucous leukoplakia.

CASE REPORT:

A 35 year old male patient name Sanjeev kumar reported to our department with the chief complaint of a painless rough white patch on right side of inner cheek region since 6 months and also burning sensation associated with the patch since 6 months.

History of present illness revealed that 6 months back patient felt some rough area on right side of inner cheek region which was around 4x4 cm in size. Patient went to see a local dentist who suggested biopsy and it was reported as verrucous hyperplasia on biopsy. Patient was given some topical medicine to apply but he didn't get any relief.

Patient had habit of gutka chewing since 10 years, 3 pouches of gutka/day. He had a habit of keeping gutka in right buccal vestibule for 1 hour and then spitting out. He had habit of tobacco chewing with lime since 5 years once daily. He also had habit of paan chewing with lime infrequently.

On general physical examination right submandibular lymph node was palpable, single in number, mobile, soft in consistency & non tender.

On Intraoral soft tissue examination a white patch was noted on right buccal mucosa. On hard tissue examination stains & calculus was present. On local examination of the lesion non scrapable white in color, ill defined exophytic proliferative patch measuring about 4x4cm was present on right buccal mucosa extending antero-posteriorly 1 cm away from right commissural area till retromolar pad area. The lesion was present at level of occusal plane in relation with teeth 43, 44 45, 46, 47. On palpation the patches were rough in texture, leathery in consistency & was non tender.

A provisional diagnosis of proliferative verrucous leukoplakia was given. Differential Diagnosis of Verrucous carcinoma and Squamous papilloma were given.

Toluidine blue staining along with lugol's iodine was performed and was suggestive of dysplastic changes. In investigations Hematologic findings were normal. Orthopantomogram was done to rule out bone involvement. Excisional biopsy was done and on histopathology parakeratinised stratified squamous epithelium & underlying connective tissue was seen. Increased layers of stratum spinosum & few areas of parakeratin plugging were seen. Chronic inflammatory cell infiltrate in connective tissue, some muscle fibres & areas of mucous salivary gland acini suggestive of verrucous hyperplasia. A final diagnosis of proliferative verrucous leukoplakia was given.

DISCUSSION:

In 1985 term proliferative verrucous leukoplakia was given by Hansen et al. It is a type of non homogenous leukoplakia. It manifests in form of white lesions distributed over one or more locations (multifocal) within the oral cavity which are clinically and histologically distinguishable from typical leukoplakia lesions. The term PVL is used for lesions initially presenting homogenous white appearances with change in clinical & microscopic aspects during their natural history. The term proliferative means persistent, diffuse, progressive, and multifocal.
Verrucous is used for warty, verrucal, exophytic, keratotic lesion. Leukoplakia which arises in flat white keratotic patches. It most commonly occurs in 5th to 6th decade. Mean age of diagnosis is over 60 years. Female predilection (4:1). Most common site is buccal mucosa in women and tongue in men. Most cases occur in western population.

Etiology
Hansen reported 62% of patients in his study chewed tobacco. Zakrewaka reported 70%. Silverman et al said 31%. Bagan et al found least i.e 23.3% Association with HPV strain 16 & 18 has also been reported. This was reported by Palefsky 1995, Gopalakrishnan 1997, Eversole 2000. Candida was found in 68% of the patients. This was reported by Silverman et al. Recently Bagan et al reported EBV associated with PVL. Gender & age related effects on immune competence have also been reported.

Histopathology
PVL is a clinical diagnosis. Hansen et al reported that PVL on histopathologic continuum goes through 4 stages.
1) Hyperkeratosis
2) Verrucous hyperplasia
3) Verrucous carcinoma
4) Papillary squamous cell carcinoma

Murrah & Batsakis also reported that it goes through 4 stages
1) Hyperkeratosis without epithelial dysplasia
2) Verrucous hyperplasia
3) Verrucous carcinoma
4) Squamous cell carcinoma

Absence of epithelial dysplasia in initial stage of histopathologic spectrum of PVL prevents it from being recognized as potentially malignant.

Immunohistochemistry
Increased tumor growth factor α was observed in PVL. Alterations in cell cycle regulatory genes like p16 INK4α & P14ARF. These are genes involved in cell cycle regulation. p16 INK4α is a tumour suppressor gene located on chromosome 9 in region of 9p21. It is either deleted or mutated in a wide range of malignancies.

Treatment
PVL is resistant to all treatments and recurs. Fettig et al suggested aggressive surgery like block resection, but total resection is rarely possible due to widespread disposition of lesion in oral cavity

Malignant Transformation
There is 100% rate of malignant transformation. This is due to successive appearance of multiple primary tumors (field cancerization)

Recurrence
Recurrence after treatment is the rule. Soon after first treatment lesion appears at previous site as well as at new site, gingiva most commonly

REFERENCES:
8. Shear M, Pindborg, J J ; Verrucous hyperplasia of the oral mucosa, Cancer (cancer), 980-; 46 , 8 : pp 1855-62.

Corresponding Address:
Dr. Nupur Agarwal
rupun48@gmail.com
LIST OF PHOTOGRAPHS

Fig 1:- Pre - Operative Intra Photograph
Fig 2:- After Toluidine Blue Staining
Fig 3:- After Lugol’s Iodine Staining
Fig 1:- Post - Operative Intra Photograph