Findings: Lesions from two of the patients were negative for the MAML2 gene rearrangement while lesions from the other two patients were positive for the MAML2 gene rearrangement.

Conclusion: Although it can be concluded that the two patients with positive translocation for MAML2 had a diagnosis of IMEC, the same conclusion could not be drawn for the two patients with negative translocation. Whether the cases that were negative for the translocation are GOCs with MEC-like islands or MAML2 negative IMEC could not be ascertained. Therefore, MAML2 gene rearrangement is not always dependable in differentiating IMECs and GOCs that share similar histologic overlap. The limited nature of the study due to small sample size precludes a more definitive conclusion. Collaboration vis-à-vis cases and data exchanges between oral and maxillofacial pathology centers may help achieve a better understanding of two uncommon, but clinically impactful, entities.

TUMOR ASSOCIATED MACROPHAGES: TAGGING AGGRESSIVENESS IN ORAL SQUAMOUS CELL CARCINOMA.

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Introduction: A tumor cannot progress independently of its micro-environment-the stromal cells, tumor associated inflammation, metabolic alterations and extracellular matrix remodeling is significant in disease progression, evolution and metastasis. Bi-directional interactions between the tumor cells and stromal elements determine individual tumor behavior as reflected in the prognostic variability of cases within the same histological grade.
However, pivotal findings relating to the tumor micro-environment (TME) in Oral Squamous Cell Carcinoma (OSCC) still remain unaccounted for in the standard grading and staging systems. Thus evaluation of the TME could provide a more robust and accurate predictive assessment of OSCC.
Tumor Associated Macrophages (TAM) constitute the major inflammatory cell population of the TME, with a prominent role in stromal modulation and tumor progression. TAMs have also been regarded as suitable demonstrators of the "seed and soil theory" of metastasis.

Objective: To correlate the presence and role of TAMs in OSCC with the STNMP staging system.

Findings: Immunohistochemical evaluation revealed a definitive presence of TAMs at the advancing front of the tumor. The density of cells escalated from STNMP stage-1 to stage-4. A statistically significant, strong positive correlation was noted between-TAMs, tumor stage, tumor size and nodal status. A poor correlation between TAMs and tumor grade was noted.

Conclusion: In India, of the 77,003 new cases of OSCC registered annually, 67.7% of the patients are lost to the disease. While tumor grade is indicative of the degree of differentiation of OSCC, it is inadequate as a sole predictor of tumor behavior and prognosis. A holistic evaluation of tumors and their TME may be the remedy. Thus, it has emerged that TAMs being dynamic cells of the TME, could be utilized as indicators of tumor behavior and aggressiveness.

PLAQUE-TYPE LICHEN PLANUS OR LEUKOPLAKIA WITH LYMPHOCYTIC HOST RESPONSE?.

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Introduction: Oral epithelial dysplasia (OED) and oral squamous cell carcinomas (SCCAs) often exhibit a lymphocytic host response (LHR) present as a band at the epithelium-connective tissue interface. Because these are often diagnosed as dysplasia with lichenoid mucositis or lichenoid dysplasia, clinicians assume that such lesions represent dysplasia or SCCA arising within lesions of oral lichen planus (OLP). If the clinical lesion is a solitary plaque, the diagnosis of plaque-type OLP may be made. Lichenoid lymphocytic reactions are not specific to OLP and may be seen in drug-induced, contact hypersensitivity reactions and other conditions. The objective of this study is to review cases of leukoplakia with a lichenoid LHR.

Materials and Methods: Cases diagnosed as OED with lichenoid features or lichenoid mucositis that represented biopsies from solitary white lesions were identified from the files of one laboratory from January 2013 to December 2018.

Results: There were 13 males and 11 females (1.2:1 male to female ratio), and the median age was 61 (range 37–90). All lesions were unilateral and the two most common locations were the tongue (12 cases, 50.0%) and the gingiva (5 cases, 20.8%). Hyperkeratosis and/or parakeratosis and epithelial atrophy was present in 23 (95.8 %) and 10 cases (41.6%) respectively while degeneration of the basal cells was present in 7 cases (29.1%) only. OED was present in 13 (54.1%) of the cases (5 mild, 5 moderate, 2 severe, and 1 carcinoma-in-situ); 36.3% of the cases that showed epithelial atrophy also showed OED. A lymphocytic band was present in 24 cases (100%).

Conclusion: These lichenoid lesions were solitary plaques located most commonly on the tongue and gingiva, common sites for leukoplakia with 54.1% exhibiting OED. As such, these lesions more likely represent leukoplakia with a LHR rather than OLP. Clinicopathologic correlation is essential for accurate diagnosis.

COMPARATIVE ASSESSMENT OF P16 PROTEIN EXPRESSION IN NORMAL AND DYSPLASTIC ORAL MUCOSAL EPITHELIUM.

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Objective: Expression of the protein marker p 16INK4a is used as a surrogate for human papillomavirus (HPV) infection in biopsies of oral and tonsillar mucosa. While HPV infection accounts for <5% of oral cavity cancers, its association with oral epithelial dysplasia (OED) is unclear, with prevalence estimates ranging from zero to greater than 90%. In this study, the expression of p 16INK4a was examined in archived biopsy specimens by immunohistochemistry within three groups: control mucosa (CM), low-grade dysplasia (LGD) and high-grade dysplasia (HGD). Tissue samples were age-, sex-and site-matched with 24 cases in each group. Grading of p16 expression was performed according to the criteria of intensity and proportion of cells as described by Grobe et al.

Findings: Fifteen of the 24 HGDs (62.5%), fourteen of the 24 LGDs (58.3%) and four of the 24 CMs (16.6%) were positive for p 16INK4a expression. The difference in p16 expression between HGD versus CM and LGD versus CM were analyzed by Wilcoxon signed rank test and statistically significantly different at level with p-values of 0.0001 and 0.0009, respectively. Greater p16 expression was noted in HGDs compared to LGDs (p-value = 0.01960, which was significant at level). A step-down