

Objectives: Oral cancer etiology is multifactorial and the main risk factors are tobacco chewing and smoking along with alcohol consumption and viruses. In the current study from a South Indian population, we sought to determine the role of HPV 16 in the pathogenesis, its concordance with p16 over expression in OSCC. Preliminary examination on FFPE embedded OSCC (n=297) sample was observed by H & E staining, and cases showing cytological changes were selected.

Findings: HPV 16 DNA prevalence were assessed by Conventional PCR method which showed 128 out of 297 samples positive for HPV16 DNA. Further, the frequency distribution of HPV 16 E6/E7 in 128 tissues was evaluated by qPCR which showed 97 samples were positive for HPV16 E6 qPCR and 98 samples positive for HPV16 E7 qPCR. For the same 128 samples immunohistochemistry was conducted for p16 evaluation. Out of 128 tissue samples, 19 tissue samples were found to be positive for p16 overexpression (+++). Evaluation of mRNA expression of E6 and E7 in the 19 samples was estimated by flow-cytometry, which revealed only 7 samples positive for the mRNA expression. There was no correlation between p16 expressed (+++) samples and quantification of HPV16 mRNA expressions. From these investigations the role of HPV in the etiology, pathogenesis of OSCC was not established.

Conclusion: This led to the performance of meta-analysis in which studies pertaining to HPV-related OSCC evaluated by application of conventional and qPCR, flow cytometry and IHC for the detection of DNA, mRNA and p16 overexpression, respectively were included. The results obtained were indicative that HPV prevalence in the oral cavity is low and unlikely to play a major significant or decisive role in the etiology, pathogenesis of OSCC. Our results were in accordance with the meta analysis results.

WNT/B-CATENIN SIGNALING PATHWAY REGULATES TUMOR-INITIATING CELLS IN HEAD AND NECK SQUAMOUS CELL CARCINOMA.

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Objectives: Head and neck squamous cell carcinoma (HNSCC) is one of the leading cancers, with a 40% 5-year survival rate in advanced cases. HNSCC is notorious for its high recurrence rate and frequent occurrence of synchronous/metachronous primary tumors. Tumor initiating cells (TICs) model was proposed to explain its tumor heterogeneity and frequent recurrences. Lineage tracing is a genetic approach that allows identification of TICs in their native habitats and characterization of their in vivo behavior.

Findings: Our re-analysis of TCGA data revealed that high expression of AXIN2, a downstream target of Wnt signaling, was significantly correlated with low survival rate of HNSCC. To characterize the Wnt-responsive cell population, we established a carcinogen-induced model using Axin2-CreER reporter mice. After tamoxifen induction, clonal expansion of fluorescent reporter-positive cells (Wnt-responsive tumor cells) was visualized in the basal cell layer of epithelial dysplasia and HNSCC, suggesting the critical role of Wnt in regulating TICs in early

stages of carcinogenesis. β -catenin and LEF1 immunofluorescent staining were performed to confirm Wnt activation. Lineage tracing was also accomplished in 3D organoid culture. The fluorescent reporter-positive cells were capable of forming organoids. Furthermore, application of cisplatin enriched AIXN2 cell population.

Conclusions: Activation of Wnt/ β -catenin signaling pathway is an early event in HNSCC carcinogenesis. Lineage tracing using the Axin2-CreER reporter may link TICs properties with a fundamental signaling pathway in normal development. Further research is required to clarify the role of Wnt-responsive TICs in recurrence and therapy resistance of HNSCC.

ADENOID AMELOBLASTOMA: A SERIES OF 5 TUMORS. DR. ELIZABETH ANN BILO-DEAU, DR. RAJA SEETHALA. UNIVERSITY OF PITTSBURGH

Background: First described in 1959 by Waldron and more fully characterized by Loyola et. al. in 2014, adenoid ameloblastoma (with dentinoid) is a rare odontogenic tumor variant with less than 20 reported cases in the literature.

Objectives: Adenoid ameloblastomas were identified after review of ameloblastomas from the University of Pittsburgh Medical Center Department of Pathology archives from 1990 to 2018.

Findings: A review of our archives yielded 6 cases of adenoid ameloblastoma in 5 patients. Of these, 2 tumors were obtained from the "in-house" pathology files with one tumor being a recurrent adenoid ameloblastoma, whereas the remaining were consultations. The 4 cases (in 3 patients) received in consultation had a differential diagnosis of a salivary neoplasm. All cases were in the maxilla (5/5, 100%), 60% were in males, with a mean age of 52.6 years (range 29-65), and mean size of 5.8 cm (range 4.0 to 7.8 cm). Increased mitotic activity was present in all cases, (mean 7 mitoses/10hpf, range 3-11 mitoses/10hpf). All tumors exhibited pseudoglandular spaces. Other common histologic features included whorls or morules, clear cells, ghost cells, and matrix production with hard tissue formation. Immunohistochemically, the tumors were positive for p63 (3/3, 100%), beta catenin within the morules (2/3, 66.6%), AE1/3 (1/1, 100%), CK5/6 (1/1, 100%), vimentin (1/1, 100%), CEA (2/2, 100%), p40 (1/1, 100%) and negative for BRAF V600E (0/3, 0%), calretinin (0/2, 0%), CK7 (0/1, 0%), SMA (0/4, 0%), S100 (0/3, 0%), Sox-10 (0/1, 0%), TTF-1(0/1, 0%), Pax-8 (0/1, 0%), p16 (0/1, 0%).

Conclusions: Adenoid ameloblastoma (with dentinoid) is a rare, aggressive odontogenic tumor variant with morphologic overlap with salivary neoplasms. Given the histologic similarities to other tumors and the high rate of recurrence, further characterization of this entity is needed

PRIMARY INTRAOSSEOUS SOFT TISSUE MYOEPIHELIOOMA OF THE MANDIBLE: CASE REPORT AND LITERATURE REVIEW.

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Introduction: Soft tissue myoepithelioma (STM) is a benign tumor composed of spindle and epithelioid cells arranged in various patterns within chondromyxoid stroma. While salivary gland myoepithelioma has been well-recognized as a variant of pleomorphic adenoma, the absence of normal myoepithelial cells within STM may be why STM was only recently-recognized. The occurrence of STM within the bone is