tumor overall. Among the malignant tumors, mucoepidermoid carcinoma was the most common type, followed by secretory carcinoma and acinic cell carcinoma.

KI67 IS AN INDEPENDENT PROGNOSTIC MARKER FOR RECURRENCE AND RELAPSE IN ORAL SQUAMOUS CELL CARCINOMA PATIENTS.

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Objectives: Ki67 expression was associated with the prognosis of several tumors and played a key role in the choice of medical treatments. However, the diagnostic value of Ki67 in oral squamous cell carcinoma (OSCC) has not been fully-evaluated. In this study, we aimed to elucidate the prognosis value of Ki67 in large number of OSCC patients.

Findings: Ki67 expression was detected by immune-histochemical staining methods in 298 OSCC samples and 98 none-tumor oral mucosa samples (62 dysplasia mucosa and 26 normal mucosa), which were acquired from Nanjing Stomatological Hospital, Medical School of Nanjing University. Expression of Ki67 was assessed independently by two professional pathologists. Expression of Ki67 in normal mucosa, mucosa with dysplasia and OSCC tissue was compared. Correlations between Ki67 expression and clinicopathological parameters were analyzed by Chi-square test. Kaplan-Meier survival curves and cox progression analysis were used to assess the diagnostic value of Ki67 for OSCC. We found that Ki67 expression was higher in OSCC tissues than in non-tumor tissues, and it increases with the progression of dysplasia in oral mucosa tissues. In addition, high Ki67 expression in OSCC patients was associated with poorer tumor differentiation (P=0.001), more lymph node metastasis (P=0.006), and inferior worst pattern of invasion type (WPOI) (P<0.0001). Kaplan-Meier survival analysis demonstrated that patients with higher Ki67 expression was correlated with poorer OS (P=0.0333), RFS(P=0.003), MFS (P=0.0032) and DFS (P=0.003). Further, multivariate analysis also demonstrated Ki67 expression remained an independent negative prognostic factor for survival for OS, DFS, RFS and MFS.

Conclusions: Ki67 overexpression is associated with the progression of OSCC and can serve as an independent prognostic factor for OSCC patients

METASTASIS TO THE MANDIBLE FROM AN UNDIAGNOSED PULMONARY ADENOCARCINOMA: A REPORT AND REVIEW OF THE LITERATURE.

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Objective: Metastatic lesions account for 1% of all oral and maxillofacial malignancies. A quarter of gnathic metastases are discovered before the primary tumor is known. We present a case of adenocarcinoma of the mandible, as first evidence of advanced lung cancer.

Findings: A 65-year-old male presented to the oral surgeon with a 6-month history of lower left jaw pain. Panoramic radiograph showed an ill-defined radiolucency inferior to the mandibular canal. A biopsy revealed a scattered glandular proliferation, with a few areas consisting of cribriform architecture and foci of back to back glandular lumens. No features of mucoepidermoid carcinoma were identified. A subsequent PET CT scan showed an ill-defined nodule in the left upper lobe of the lung measuring up to 2.5 cm in greatest dimension. Multiple hilar, subcarinal, and paratracheal nodules were also identified, concerning for nodal metastasis. Immunohistochemical stains were then performed on the original biopsy from the mandible and the tumor cells stained positive for TTF1, Cytokeratin 7, and Napsin A, suggestive of adenocarcinoma of pulmonary origin. Consequently, MRI of the brain identified lesions in the parietal and frontal lobes, measuring up to 3.4 cm. Treatment for the patient included chemotherapy with Pemetrexed (Alimta) and carboplatin, immunotherapy with Keytruda, and once tapered off, palliative radiotherapy.

Conclusion: Primary adenocarcinoma of the jaw is extremely rare, except for 2−3% central mucoepidermoid carcinomas. The possibility of a metastatic tumor should be a consideration when encountering unusual histomorphology of an adenocarcinoma in the jaw bone.

FASCIN EXPRESSION IN AMELOBLASTOMA, ODONTOGENIC KERATOCYST AND DENTIGEROUS CYST.

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Objectives: The purpose of this study was to assess and compare fascin expression in 4 lesions which differ in aggressiveness: odontogenic keratocyst (OKC), dentigerous cyst (DC) and two types of solid and unicystic ameloblastoma, and to find out whether fascin expression is associated with aggressiveness of these lesions or not.

Methods: 9 solid ameloblastomas (SA), 12 unicystic ameloblastomas (UA), 13 OKC and 12 DC were assessed in this study. The slides were examined at x400 magnification. Finally the lesions were divided into two groups based on microscopic examination, “low expression” and “high expression”.

Findings: There were no significant differences between the lesions, except that fascin expression was slightly higher in UA in comparison to other groups in intensity and count of the immunostaining cells.

Conclusions: The results of this study suggest that fascin might be more involved in cell invasion and migration (as in carcinomas) than local aggressiveness. We suggest more studies

with more samples, assessing expression of different proteins be done in the future.

TUNICAMYCIN-INDUCED ENDOPLASMIC RETICULUM STRESS UP-REGULATES TUMOUR-PROMOTING CYTOKINES IN ORAL SQUAMOUS CELL CARCINOMA.

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Objectives: Signal transducer and activator of transcription (STAT)-3 lies at the convergence point of key pathways involved in many malignancies including oral squamous cell carcinoma (OSCC). Endoplasmic reticulum stress (ERS) and the unfolded protein response promote either survival or apoptosis in different cancers. We investigated the expression of STAT3 pathway-related genes and proteins under ERS in OSCC.

Three normal oral keratinocyte (NOK) and three OSCC cell lines were subjected to tunicamycin to induce ERS for 24 hours or to the vehicle medium as control. A pathway-focussed array was used to analyse the modulation of STAT3 pathway gene expression under ERS using qPCR. The expression of key regulated proteins was investigated in the cell lines using immunocytochemistry and in 76 OSCC and 9 normal oral mucosa (NOM) tissue samples using tissue microarray technology and immunohistochemistry.

Findings: ERS resulted in up-regulation of interleukin-6 receptor (IL6R) gene in NOK cell lines ($p = 0.001$) and IL5 ($p = 0.005$) and IL22 ($p = 0.024$) in OSCC cell lines. Greater STAT3 ($p = 0.019$) and leukaemia inhibitory factor receptor ($p = 0.042$) protein expression was observed in treated than untreated NOK cell lines.

Conclusions: The gene and protein regulation patterns show that ERS plays a role in modifying the tumour microenvironment in OSCC by up-regulating tumour-promoting cytokines.

CLINICO-PATHOLOGICAL SIGNIFICANCE OF B-CATENIN AND E-CADHERIN EXPRESSION IN SALIVARY GLAND TUMOR AT UCH IBADAN.

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Objectives: β-catenin (B-Cat) is a cell adhesion molecule associated with the invasion and metastasis of carcinomas of the head and neck, esophagus while reduced expression of E-cadherin (E-cad), a transmembrane glycoprotein, is associated with loss of differentiation, acquisition of an invasive phenotype, and an unfavorable prognosis in carcinomas from several sites. B-Cat & E-Cad complex are involved in cell adhesion, signal transduction & motility. Aim is to identify the clinical / pathological significance of B-Cat & E-Cad expressions in salivary gland tumors (SGTs) presenting at the University College Hospital, Ibadan.

Findings: The expressions of β-cat & E-Cad were analyzed in 46 SGTs (10 pleomorphic adenomas PSA, 3 basal cell adenoma, 12 adenoid cystic carcinoma ADCC, 10 mucoepidermoid carcinoma MEC, 5 acinic cell carcinoma ACC, 4 polymorphous low grade adenocarcinoma PLGA & 2 papillary cystadenocarcinoma PCADCA) by immunohistochemistry in formalin-fixed, paraffin embedded specimens (Rabbit monoclonal; Satacruz biotechnology). Result shows immunostaining of B-cat & E-Cad were membranous & cytoplasmic without nuclear involvement, staining was more severe in the ductal areas especially in PSA, there was significant loss of membranous stain in ACC on multivariate analysis. E-Cad staining loss was significantly associated with tumour stage in ACC & MEC.

Conclusion: loss of β-catenin adhesion molecule may be involved in the development of ACC. E-cad expression is an independent indicator of clinical aggressiveness in patients with ACC.& MEC.

KRAS MUTATIONS DRIVE ADENOMATOID ODONTOGENIC TUMOR.

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Objective: KRAS is the most frequently mutated oncogene in human neoplasms and we have previously reported KRAS p.G12V mutations in adenomatoid odontogenic tumors (AOT). We aimed to expand this cohort of samples and to test the association of KRAS mutations with clinical and histopathological parameters. A convenience sample of 30 AOT cases was included in the study. The hotpot KRAS p.G12V mutation was assessed by TaqMan allele-specific qPCR and codon 12 was direct sequenced. Clinical information obtained included patients age, tumor site, association of the lesion with impacted teeth and clinical tumor size. In addition, tumor capsule thickness was evaluated by morphometric analysis. Statistical analysis was carried out to test the association of KRAS codon 12 mutations with clinico-pathological parameters.

Findings: Molecular results confirmed KRAS p.G12V mutation in 14/23 cases, and p.G12R in 1/23. Eight cases were wild-type and samples from 7 cases failed amplification. Codon 12 mutations were not associated with any of the clinicopathological parameters tested ($p>0.05$).

Conclusion: AOT show high frequency of KRAS codon 12 mutations (15/23, 65%), which occur irrespectively of patients' age, tumor location, association with impacted teeth, tumor clinical size or histopathological capsule thickness. Supported by FAPEMIG, CAPES and CNPq/Brazil.

CORRELATION OF HPV16 DETECTION AND P16 EXPRESSION IN ORAL SQUAMOUS CELL CARCINOMA.

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