and myoepithelial cells, showing tubular, cribriform and solid subtypes. The solid subtype is considered as high-grade with more aggressiveness and poorer prognosis. However, the molecular mechanism remains unknown. The aim of this study was to identify the clinicopathological characteristics of high-grade SACC, to clarify the molecular mechanism underline its distinct characteristics, and hopefully to explore potential molecular targets for SACC therapy.

Study design: Activated Notch1 (NICD) and myoepithelial cell markers were used for immunohistochemistry in 119 SACCs including 59 cribriform-tubular and 60 solid subtypes. Notch1 mutations were analyzed by DNA sequencing in all the SACC cases. The effect of activating NOTCH pathway on the biological behavior of SACC cell lines was investigated with transfection and functional studies.

Results: Notch1 mutations in the negative regulatory region and Pro-Glu-Ser-Thr–rich domains were identified in 26 of 119 patients with SACC, and 24 (92%) of 26 Notch1 mutant cases were predicted to be activating with NICD positive, with 2 cases predicted to be inactivating with NICD negative. Most (23/24, 96%) cases with activating Notch1 mutations were high-grade solid SACCs. Meantime, only 17 (18%) of 93 NOTCH1 wild-type tumors stained positive, and 16 of 17 tumors with NICD positive were high-grade solid subtypes. Furthermore, high-grade solid SACCs showed dramatically decreased short-term survival, tended to suffer bone invasion and metastasis, and presented NICD positive and myoepithelial cell markers negative simultaneously. Transfection and functional studies showed forced NICD expression promoted high level of proliferation and migration in SACC cells.

Conclusions: Our findings showed activating Notch1 mutations were related to the loss of myoepithelial differentiation in high-grade SACCs and might contribute to higher proliferation and worse outcome in high-grade tumors. Targeting the Notch signaling pathway in high-grade SACCs may provide therapeutic benefits.

HISTOMORPHOLOGICAL COMPARISON OF SOLITARY KERATOCYST AND KERATOCYST ASSOCIATED WITH BASAL CELL NEVOID SYNDROME (NBCS).

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Introduction: The keratocyst of the jaws is an odontogenic entity of locally aggressive biological behavior, with potential for progressive growth, with infiltrative growth, despite being a completely benign cystic lesion. However when associated with NBCS, the behavior is more agressive, without changing its benign biological nature.

Objectives: compare the histopathological findings of solitary keratocyst v / s associated with Basal Cellular Nevoid Syndrome.

Material and method: The histology of 20 cases of solitary keratocyst is reviewed and compared with 20 keratocysts associated with SNBC. The characteristics of the epithelium and the connective tissue that make up the cystic capsule are evaluated; all stained with hematoxycillin eosin.

Conclusions: There are histomorphological differences in both tipes of keratocyst; the most outstanding difference is the presence of a remarkable number of satellite cysts in the cystic capsule, of those associated with the SNBC, which would explain the high recurrence rate of them.

SYNCHRONOUS EXPRESSION OF E-CADHERIN AND SYNDECAN-1 IN AMELOBLASTOMA: A PRELIMINARY STUDY.

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Objectives: This study aimed to evaluate the percentage of expression and the possibility of synchronicity between E-cadherin (Ecad) and Epithelial syndecan-1 (Syn-1E) in Unicystic ameloblastoma (UAM) and solid multicystic ameloblastoma (SMA) and the association between this synchronicity and stromal immunoexpression of Syn1 (Syn1S).

Findings: Immunohistochemical analysis of Ecad and Syn1 was performed for 30 (15 UAM, 15 SMA) cases of ameloblastoma, the percentage of expression was evaluated with the average value for expression, and intensity was evaluated with the Immunomebrane plug-in. (Image J, BioMediTech, Finland) The percentage expression of Ecad and Syn1E was high in UAM. ($p<0.05$ vs SMA) The stromal expression of Syn1 was high in SMA ($p<0.05$, vs UAM) and the intensity expression was similar in both types of ameloblastomas. ($p>0.05$)

Conclusions: We observed synchronicity in the expression of Ecad and Syn1E in both types of ameloblastomas. The adhesiveness of the tumoral cells is probably related to the regulation of expression of both proteins; thus, an increase or reduction of synchronicity is related to cell invasion and the capacity to migration to the stroma, which was reflected in the behavior of the ameloblastomas in the present cases. This assumption may be further supported by an increase in expression of Syn1S in the SMAs; therefore, it is possible that the low synchronicity between Ecad and Syn1E constitute an important factor for the aggressiveness of ameloblastomas.

SECRETORY CARCINOMA OF THE PAROTID GLAND IN A 9-YEAR-OLD FEMALE PATIENT: A CASE REPORT AND REVIEW OF THE LITERATURE.

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Introduction: Formerly known as mammary analogue carcinoma (MASC), secretory carcinoma of the salivary glands (SCSG) is a low-grade malignancy harboring the ETV6-NTRK3 translocation. SCSG shares morphological and genetic features with secretory carcinoma of the breast. It shows a characteristic protean histological phenotype reminiscent of acinic cell carcinoma, mucoepidermoid carcinoma, and adenocarcinoma, NOS. We report a case of SCSG in a pediatric patient showing a pure zymogen-poor acinic cell carcinoma morphology.

Clinical presentation: A 9-year-old female patient presented to the ENT clinic with 8 months history of left facial swelling. Imaging studies showed a 2.7 cm lesion of the left parotid gland with no lymphadenopathy. Left lateral lobe parotidectomy with facial nerve dissection and preservation was performed. Gross examination of the tumor revealed a tan, hemorrhagic ill-defined mass, measuring $2.5 \times 2.4 \times 2$ cm. Microscopic examination showed lobulated growth pattern consisting of papillary architecture and microcystic spaces. Lobules of tumor cells were identified in the intraparotid lymph node. The tumor was negative for DOG-1 and strongly positive for Mammaglobin, S-100, GATA-3, CK 7, and Pan cytokeratin. The ETV6 gene translocation was subsequently detected by FISH.

Literature review: A total of 17 cases of pediatric SCSGs (age range: 8-18 years) have been reported in the literature. The parotid gland (88%) is the most common location. The mainstay of treatment is surgical resection. Based on the limited number of documented cases, SCSG appears to show an indolent biological behavior rarely occurring in pediatric patients.

Conclusion: Because of the spectrum of histological patterns of SCSG, immunohistochemical markers and genetic documentation of the ETV6-NTRK3 gene fusion are essential to correctly diagnose this tumor.

IMMUNOHISTOCHEMICAL DISTRIBUTION OF THE SIBLINGS IN AMELOBLASTOMA AND ODONTOGENIC KERATOCYST.

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Objective: Ameloblastomas are benign but aggressive neoplasms of the jaw. The odontogenic keratocysts (OKC) are aggressive jaw cysts of odontogenic origin. A recent WHO classification designated OKC as a neoplasm but a current edition redesignated it as a cyst. The small integrin-binding ligand n-linked glycoproteins (SIBLINGs) are a family of molecules that some epithelial neoplasms use in furthering their progression. Members of the SIBLING family are bone sialoprotein (BSP), dentin matrix protein1 (DMP1), dentin sialophosphoprotein (DSPP), matrix extracellular phosphoglycoprotein (MEPE), and osteopontin (OPN). The objective of this study was to compare the expression of the SIBLINGs in ameloblastomas and odontogenic keratocysts.

Findings: Using immunohistochemistry, with appropriate controls, 49 cases of ameloblastomas and 35 cases of OKCs were screened for the expression of all five SIBLINGs in a retrospective study on archived paraffin sections. Immunoreactivity was scored as positive if > 10% of tumor/cyst cells stained for a SIBLING and negative if <10% of cells failed to stain for a SIBLING. For ameloblastomas 46 (94%) were immunopositive for BSP, 49 (100%) for OPN, 38 (76%) for DSPP, 16 (33%) for DMP1, 32 (65%) for MEPE, 47 (96%). For OKCs 32 (91%) were positive for BSP, 27 (77%) for DMP1, 35 (100%) for DSPP, and 14 (40%) for MEPE. The expression of DSPP and MEPE in ameloblastomas and OKCs respectively, were significant ($p<0.05$) compared with controls. MEPE ($\chi^2=6.15$, $p<0.05$), and BSPP ($\chi^2=26.06$, $p<0.05$) had positive predictive values greater than chance for ameloblastoma and odontogenic keratocyst, respectively.

Conclusion: While all members of the SIBLINGs are expressed in ameloblastomas and OKCs, DSPP expression in OKCs is significantly higher than in ameloblsatomas, whereas, MEPE expression in OKCs is considerably lower than in ameloblastomas. The differences in the expression of DSPP and MEPE between ameloblastomas and OKCs may indicate differences in the degree of aggressive behaviors.

GALECTIN-1 INHIBITION OF ORAL CANCER IN VITRO.

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Galectin-1 is a carbohydrate-binding molecule that has been shown to be over-expressed in many types of cancer, including oral squamous cell carcinoma (OSCC). The higher the level of expression of galectin-1 by OSCC cells the greater the likelihood of invasion, distant metastasis and a poor survival rate.

Objectives: Investigation of the effect of galectin-1 in OSCC invasion, migration and epidermal-mesenchymal transition in vitro,and the effect of inhibition of galectin-1 using a small-molecule inhibitor (OTX008).

Results: One normal oral keratinocyte (NOK) cell line and three OSCC cell lines were cultured and the expression of galectin-1 protein in each quantified using an ELISA. All cell lines were found to express galectin-1, and one of the OSCC lines produced significantly more galectin-1 than the NOK cell line at 6, 24 and 48 hours.

All four cell lines were cultured with three concentrations of galectin-1 (50, 100 and 150 ng/mL) and four concentrations of OTX008 (12.5, 25, 50 and 100 μg/mL), and cell viability was assayed at 24, 48, 72 and 96 hours. Galectin-1 decreased cell viability at 24 hours in two of the OSCC lines, had no effect on the third, and increased cell viability in the NOK cells at 72 hours. OTX008 reduced cell viability in a dose-dependent manner in all cell lines, and this effect increased at each time point during a 96 hour culture period. OTX008 had the least effect on cell viability of the OSCC line with the highest galectin-1 levels compared to the other cell lines.

Conclusions: Galectin-1 is expressed by NOK and OSCC cell lines in vitro. OTX008 decreases the cell viability of OSCC and NOK cells in a dose-dependent manner, however this effect is reduced by higher endogenous levels of galectin-1.

DOWN-REGULATED YAP INHIBITS PROLIFERATION AND MIGRATION OF ORAL SQUAMOUS CELL CARCINOMA CELLS.

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