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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