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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