

## **Immunohistochemical Study of Notch Signaling Proteins in the Calcifying Epithelial Odontogenic Tumor (Pindborg Tumor)**

Type:

Article

Abstract:

Notch signaling pathway mediates diverse biological processes including cell fate decisions during odontogenesis. Dysregulation of Notch has been implicated in the tumorigenesis of some odontogenic neoplasms but its role in the calcifying epithelial odontogenic tumor (CEOT) remains unclarified. The aim here was to investigate Notch expression in CEOT and to speculate on its significance. Receptors Notch1-4 and their ligands (Jagged1, Jagged2 and Delta1) were examined immunohistochemically in six CEOT cases. Expression levels were quantified according to the percentage of positive tumor cells, amyloid-like proteins and calcifications: (-), negative staining; (+), mild and focal positivity <25%; (++) , moderate positivity in significant areas 25-50%; (+++), strong positivity in predominant areas >50%. CEOT epithelium demonstrated variable expression levels for Notch1, 3, 4, Jagged1 and Delta1 suggesting upregulation of these molecules at sites of tumor differentiation. Distribution patterns were distinct with some overlap. Their localizations were largely membranous and/or cytoplasmic. Notch2 and Jagged2 were absent. Amyloid-like materials strongly expressed Jagged1 but variably Notch1, 3 and Delta1 implicating that these signaling proteins maybe competitive substrates with CEOT amyloid-like proteins for proteolysis. Notch2, 4 and Jagged2 were absent. Mineralized substances including Liesegang rings were negative for Notch receptors and ligands suggesting that calcification process is associated with downregulation of these molecules. Stromal endothelium and fibroblasts were stained variably positive. Taken together, current data suggest that Notch receptors and their ligands may play differing roles in the acquisition of cell fates in CEOT. Notch accumulations within amyloid-like protein suggest impaired proteolysis.

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