Immunolocalization Of Notch Signaling Protein Molecules In A Maxillary Chondrosarcoma And Its Recurrent Tumor

Type:

Article

Abstract:
Background: Notch receptors are critical determinants of cell fate in a variety of organisms. Notch signaling is involved in the chondrogenic specification of neural crest cells. Aberrant Notch activity has been implicated in numerous human diseases including cancers; however its role in chondrogenic tumors has not been clarified. Method: Tissue samples from a case of primary chondrosarcoma of the maxilla and its recurrent tumor were examined immunohistochemically for Notch1-4 and their ligands (Jagged1, Jagged2 and Delta1) expression. Results: Both primary and recurrent tumors were histopathologically diagnosed as conventional hyaline chondrosarcoma (WHO Grade I). Hypercellular tumor areas strongly expressed Notch3 and Jagged1 in spindle and pleomorphic cells suggesting up-regulation of these protein molecules at sites of tumor proliferation. Expression patterns were distinct with some overlap. Differentiated malignant and atypical chondrocytes demonstrated variable expression levels of Jagged1, and weak to absent staining for Notch1, 4 and Delta1. Protein immunolocalization was largely membranous and cytoplasmic, sometimes outlining the lacunae of malignant chondrocytes. Hyaline cartilage demonstrated a diffuse or granular precipitation of Jagged1 suggesting presence of soluble Jagged1 activity at sites of abnormal chondrogenesis. No immunoreactivity for the other Notch members was observed. Calcified cartilage was consistently Notch-negative indicating down-regulation of Notch with cartilage maturation. Stromal components namely endothelial cells and fibroblasts variably expressed Notch1, 3 and Jagged1 but were mildly or non-reactive for the other members. Conclusions: Results indicate that Notch signaling pathway may participate in cellular differentiation and proliferation in chondrosarcoma. Findings implicate Notch3 and Jagged1 as key molecules that influence the differentiation and maturation of cells of chondrogenic lineage.

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