Title:	Podoplanin, E-cadherin, beta-catenin, and CD44v6 in recurrent ameloblastoma: their distribution patterns and relevance
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Abstract:	BackgroundAmeloblastoma is a benign but locally infiltrative odontogenic epithelial neoplasm with a high risk for recurrence. Podoplanin, a lymphatic endothelium marker, putatively promotes collective cell migration and invasiveness in this neoplasm. However, its role in the recurrent ameloblastoma (RA) remains unclear. As morphological, signaling, and genetic differences may exist between primary and recurrent tumors, clarification of their distribution patterns is of relevance. Materials and methodsPodoplanin was examined immunohistochemically in conjunction with E-cadherin, -catenin, and CD44v6 in 25 RA. Immunostaining according to tumor area, cellular type, and location, and relationship of these proteins were analyzed. Findings were compared with 25 unrelated primary ameloblastomas (UPA). Results: All four proteins were detected in RA and UPA samples. Expression rates for each protein were not significantly different between these two

	groups. RA demonstrated significant upregulation of podoplanin at the invasive front (P<0.05), whereas upregulation of -catenin and CD44v6 and downregulation of E-cadherin at this site were not statistically significant (P>0.05). Immunolocalization for all four proteins was predominantly membranous and less frequently cytoplasmic. Pre-ameloblast-like cells were podoplanin(+)/CD44v6(-), while stellate reticulum-like cells were podoplanin(-)/CD44v6(+). Acanthomatous, granular cell, and desmoplastic variants in both RA and UPA were podoplanin(-/low) but stained weak-to-moderate for E-cadherin, -catenin, and CD44v6. Stromal fibroblasts and lymph channels were variably podoplanin-positive. Conclusions: Podoplanin, -catenin, and CD44v6 upregulation at the tumor invasive fronts in RA and UPA supports a differential regulatory role by these molecules in mediating collective cell migration and local invasiveness. E-cadherin downregulation suggests altered cell adhesion function during tumor progression.
Keyword:	cd44v6; e-cadherin; podoplanin; recurrent ameloblastoma; beta-catenin squamous-cell carcinoma; immunohistochemical expression; odontogenic-tumors; tooth germs; osteopontin; receptors; lesions; marker; timp-2; basal
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