

Original Article

Expression of Podoplanin in Odontogenic Keratocyst: An Immunohistochemical Study

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ABSTRACT:

Background: The odontogenic keratocyst (OKC) is known to have a more aggressive biologic behaviour than other cysts of jaw. Podoplanin which is frequently used as a lymphatic endothelial marker in OSCCs has recently been found to play a possible role in odontogenic tumorigenesis also. Hence; we planned the present study to assess the role of podoplanin in OKC. **Materials & methods:** The present study included evaluation a total of 20 OKC specimens and 20 dental follicle specimens (Control). The tissue sections were stained; one with hematoxylin and eosin (H & E) stain and another with Podoplanin Antihuman antibody by immunohistochemical methods. All the results were analyzed by SPSS software. **Results:** Significant results were obtained while comparing the mean staining final score in between OKCs specimens and dental follicle specimens. **Conclusion:** Podoplanin influences the proliferative activity of basal and suprabasal cells in the epithelial lining of OKCs. However; future studies are recommended.

Key words: Dental follicle, Odontogenic keratocyst, Podoplanin

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INTRODUCTION

Odontogenic cysts & tumors comprises of unusually diverse group of lesions. These lesions reflects the multiformity in the complex development pattern of dental structures since these lesions originate through some alteration from the normal pattern of odontogenesis.¹⁻³

The odontogenic keratocyst (OKC) is known to have a more aggressive biologic behavior than other cysts of jaw.⁴ Podoplanin which is frequently used as a lymphatic endothelial marker in OSCCs has recently been found to play a possible role in odontogenic tumorigenesis also.⁵⁻⁷

Podoplanin may be involved in the process of local expansion of developmental, inflammatory and neoplastic odontogenic lesions. The pattern of staining for podoplanin in OKC could be related to its neoplastic nature, and may suggest a role of this protein in tumor invasiveness.⁸ Hence; we planned the present study to assess the role of podoplanin in OKC.

MATERIALS & METHODS

The present study was conducted in Jammu region and included evaluation of expression of podoplanin in OKC specimens. For the present study, we evaluated a total of 20

OKC specimens and 20 dental follicle specimens (Control). The inclusion criteria were as follows:

- Cases with microscopically confirmed diagnosis of odontogenic keratocyst

Data on patient age, gender, and lesion site were obtained from information submitted with the surgery request forms. The tissue sections were stained; one with hematoxylin and eosin (H & E) stain and another with Podoplanin Antihuman antibody by immunohistochemical methods.

For the evaluation of cytoplasmic and / or membranous podoplanin expression by epithelial odontogenic cells, 4 fields in OKC & dental follicles (magnification 400 X) were studied under the microscope. Mean of the four fields was estimated for each sample and considered as the final score for that sample.

Scoring was based on:

Intensity of the podoplanin expression in the epithelial odontogenic

Cells (A)

0 = absent, 1 = weak, 2 = moderate, 3 = strong, and 4 = very strong.

Percentage of podoplanin positive odontogenic cells (B)
 0 = 0% positive cells, 1 = <25% positive cells, 2 = 25–50% positive cells, 3 = 50–75% positive cells, 4 = >75% positive cells.

Total scores A+B

0 = absent,
 1–4 = weak, and
 5–8 = strong.

Final scores ranged from 0 to 8

0 = absent,
 1–4 = weak,
 5–8 = strong.

All the results were analyzed by SPSS software. Chi- square test and one way ANOVA were used for the evaluation of level of significance. P- value of less than 0.05 was taken as significant.

RESULTS

Mean final score of the OKCs specimens is 5.80 while mean score of dental follicle specimens is 2.40. Significant results were obtained while comparing the mean final score in between OKCs specimens and dental follicle specimens.

Table 1: Distribution of final score in OKC and dental follicle specimens

GROUP	No. of cases	Final Score(A+B)	
		Mean	SD
OKCs	20	5.80	0.98
Dental Follicles	20	2.40	0.80

Table 2: Comparison of staining in between OKC specimens and Dental follicle specimens

GROUP	TOTAL (A+B)	
	t-value	p-value
OKCs vs Dental Follicles	5.842	<0.05

DISCUSSION

Cysts & tumors derived from the odontogenic tissues constitute an unusually diverse group of lesions. Although different types of odontogenic cysts and tumours arise from the derivatives of embryologic dental lamina, the potential for further proliferation of these epithelial remnants during formation of a cyst and tumour is different.⁹⁻¹¹ In our study, we also evaluated the expression pattern of podoplanin in OKCs. We found that expression of podoplanin was significantly higher in OKCs than in dental follicle.

Tsuneki M et al determined immunolocalization modes of podoplanin among odontogenic tumors to discuss possible

roles of podoplanin in their characteristic tissue architecture formation.

Figure 1: Staining of OKC specimen

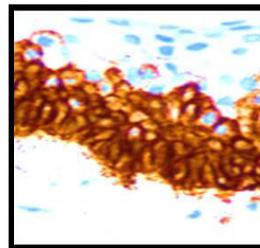
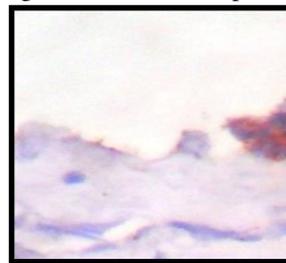


Figure 2: Staining of dental follicle specimen



Immunohistochemical profiles of podoplanin were investigated in 40 surgical specimens from ameloblastoma, adenomatoid odontogenic tumor, calcifying-cystic odontogenic tumor- and KCOT in comparison with those of proliferating cell nuclear antigen, integrin β1, fibronectin, and matrix metalloproteinase 9. Podoplanin was localized in the basal cell layer or in the peripheral zone of AM foci. It was found in spindle-shaped tumor cells of AOT, in both the basal and polyhedral cells of CCOT, and in the basal and parabasal cells of KCOT linings. They concluded that podoplanin positive cells and areas in odontogenic tumors are in close associations with extracellular matrix signalings as well as cell proliferation.¹² Friedrich RE et al analysed six keratocystic odontogenic tumour (KCOTs) from patients with known nevoid basal cell carcinoma syndrome (NBCCCS) immunohistochemically with antibodies to podoplanin (D2-40) and p63. They observed a continuous linear immunoreactivity of basal epithelial cells for podoplanin in all cases. Strong nuclear P63 expression was detected in basal cell layers and diminished in suprabasal layers. They concluded that KCOTs exhibited enhanced podoplanin expression in a clinical setting of NBCCS & the overexpression of this protein is capable of promoting the formation of elongated cell extensions, and increasing adhesion and migration of inflammatory cells. Podoplanin expression in KCOT is possibly associated with slow invasion of the adjacent structures and the well-known frequent local tumour recurrences of this odontogenic tumour.¹³

Kentaro Kikuchi et al examined the expression of podoplanin in calcifying cystic odontogenic tumor (CCOT)

in comparison with that in other so-called hard α -keratin-expressing tumors such as craniopharyngioma (CP) and pilomatricoma (PM). They observed positivity for hard α -keratin in ghost, shadow and transitional cells in all of these tumors. The podoplanin expression in CCOTs was evident in the periphery of ameloblastoma-like epithelium and the epithelial cells adjacent to ghost cells. On the other hand, in adamantinomatous-type CPs, podoplanin expression was observed in epithelial components corresponding to the stratum intermedium, but not in the periphery of ameloblastoma-like epithelium. In squamous type CPs podoplanin was expressed in basal cells but all of the PMs were podoplanin-negative. In the periphery of the ameloblastoma-like epithelium or basophilic cell layer, podoplanin was expressed more strongly in CCOTs than in CPs or PMs. Their findings suggested that the expression of podoplanin in CCOTs may reflect rapid turnover of cytoskeletal filaments and local invasiveness.¹⁴

CONCLUSION

From the above results, the authors concluded that podoplanin influences the proliferative activity of basal and suprabasal cells in the epithelial lining of OKCs. However; future studies are recommended.

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