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LETTER TO THE EDITOR

Tumor Associated Tissue Eosinophilia in Ameloblastoma

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Tumor-associated tissue eosinophilia (TATE) is characterized by the presence of eosinophils as a component of peri- and intratumoral inflammatory infiltrate. It has been reported in various malignancies like colorectal, breast, ovarian, cervical, naso-pharynx, lungs, gastrointestinal, skin, genitourinary, Hodgkin's and prostate cancer [1]. In oral cavity, tissue eosinophilia is reported in oral squamous cell carcinoma [2], odontogenic cysts [3] and mucoepidermoid carcinoma [4]. However, TATE has not been reported in any odontogenic tumors till date.

In our histopathology practice, out of 45 diagnosed ameloblastomas (including peripheral, solid/multicystic and unicystic ameloblastomas), we came across 6 (13.33%) follicular ameloblastomas showing features of TATE. The demographic details of these 6 patients are shown in Table 1. Eosinophils are easily detected in hematoxylin and eosin stained section due to their peculiar morphological feature and moreover, no special stain or marker is required for their identification. In each section, 10 areas showing maximum density of eosinophils were selected as hot spots. In each area, eosinophils were counted using a light microscope under 400X magnification and were expressed as count per high power field. TATE was graded into low (< 50 eosinophils / HPF), moderate (50 to 120 eosinophils / HPF) and severe (> 120 eosinophils / HPF) [2].

Three cases showed moderate grade of TATE while two cases showed low TATE. One case showed severe TATE grading. (Figure 1) TATE was mainly seen in the connective tissue stroma. At majority of places connective tissue stroma associated with TATE showed features of degenerative changes. Occasionally tissue eosinophils were observed in the epithelial components of ameloblastoma. Surgical excision was performed in all the tumors with no evidence of recurrences in 5 cases till date.

Differentiation and migration of eosinophils can be induced by tumor products like IL-5, IL-3, eotaxin-1, and thymus and activation-regulated chemokines [5]. In oral squamous cell carcinoma, eosinophils are the main source of eotaxin [2], showing an autocrine-like mechanism for tissue eosinophilia. It is quite conceivable that ameloblast like cells or stellate reticulum like cells of ameloblastoma might be producing some of the aforementioned factors causing tissue eosinophlilia.

Pro-tumorigenic or anti-tumorigenic role of TATE is still a controversy. The host-favorable attributes of TATE are cytotoxic protein release, assisting penetration of tumor-killing cytokines and modulation of extracellular matrix formation. Pro-tumorigenic function is mainly associated with release of angiogenic factors for promotion of tumor angiogenesis [6]. TATE is considered unfavorable in Hodgkin's lymphoma while considered favorable in colorectal, breast and prostate cancers [1]. In oral squamous cell carcinoma, TATE is described as an independent favorable prognostic indicator [2]. Literature has also revealed TATE in benign tumors of epithelial and connective tissue origin [7]. This is the first time TATE has been reported in ameloblastoma. But due to limited number of cases, it will be inappropriate to draw any conclusion about the pro-tumorigenic or anti-tumorigenic role of TATE in ameloblastoma.

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Table 1 Demographic details of the ameloblastoma cases showing tumor associated tissue eosinophilia

Sr. No	Age	Sex	Jaw	Site	Size (cm)	Duration	Type	Eosinophil per HPF	Radiology	Treatment	Follow-up
1	25	M	Mandible	Body and angle	7 × 3	4	Follicular	30	Multi-locular radiolucency	Surgical excision	NR (24 months)
2	18	F	Mandible	Angle	4 × 3.5	8	Follicular	80	Multi-locular radiolucency	Surgical excision	NR (12 months)
3	36	M	Mandible	Body	5.5 × 4	4	Follicular	60	Multi-locular radiolucency	Surgical excision	NR (36 months)
4	14	M	Mandible	Body	8 × 5	3	Follicular	25	Multi-locular radiolucency	Surgical excision	Follow-up lost
5	27	M	Mandible	Angle and ramus	7 × 3	8	Follicular	125	Multi-locular radiolucency	Surgical excision	NR (24 months)
6	38	F	Mandible	Body	4 × 2.5	6	Follicular	65	Multi-locular radiolucency	Surgical excision	NR (40 months)

HPF High power field, *NR* No recurrence

In odontogenic cysts, mast cells have been known for promoting collagenolytic activities which helps in cyst growth and expansion [8]. Similarly, mast cells have been also reported in many odontogenic neoplasms including ameloblastomas [9]. Interestingly, mast cells liberate eosinophil chemoattractant factor and histamine, which attract eosinophils in the tissue [10]. In ameloblastoma, mast cells could have a role in recruiting eosinophils at the site of tumorigenesis. In the present case series, we observed degeneration of stroma (collagenolytic activity) in ameloblastoma at the site of tissue eosinophilia. Thus, presence of stromal degeneration and tissue eosinophilia suggests a possible role of mast cells in growth and expansion. However, it will be premature to draw any conclusion from the present findings. We recommend future studies on evaluating the role of mast cells and eosinophils in progression of ameloblastoma and its correlation with the prognosis of the patient. Moreover, study of expression of eotaxin, a powerful and selective eosinophil chemoattractant [2], will reveal interesting facts about the role of TATE in pathogenesis of ameloblastoma.

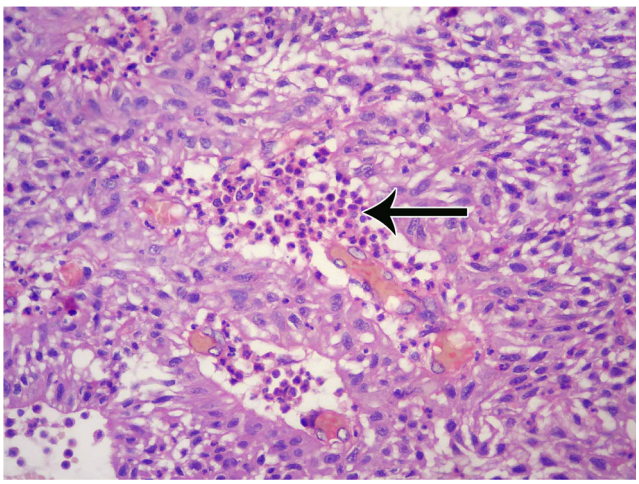

Fig. 1 Photomicrograph showing severe tissue eosinophilia in the stroma of follicular ameloblastoma. (Magnification X400; Hematoxylin and Eosin stain)

Compliance with Ethical Standards

Conflict of Interest None declared.

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