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Original Article

Mast Cells Density in Hyperkeratosis, Dysplastic Oral Mucosa and Oral Squamous Cell Carcinoma

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Abstract

Background: Mast cells normally present in small numbers in the connective tissue of all organs and release a variety of potent mediators like histamine, leukotrienes, cytokines, chymase, basic fibroblast growth factor (bFGF), tryptase and heparin through degranulation.

Objectives: The aim of the present study was the assessment of mast cell density in hyperkeratosis, dysplastic oral mucosa and oral squamous cell carcinoma (OSCC).

Methods: In this retrospective analytical study, paraffinized specimens from 15 cases of normal mucosa and 23 cases of well-differentiated OSCC, cases of hyperkeratosis and dysplasia were selected. Sections were stained with toluidine blue and then were counted at 400× magnification in hotspot areas under a light microscope. The results were analyzed using ANOVA. *P* values less than 0.05 were considered significant.

Results: Mast cells density (MCD) increased in hyperkeratosis, dysplasia, and OSCC. There was not any significant correlation between mast cell density and hyperkeratosis, dysplastic mucosa and normal mucosa. There were significant differences between mast cell counts in SCC and normal mucosa.

Conclusions: MCD is higher in hyperkeratosis and dysplastic oral mucosa than in normal mucosa. There were significant differences between mast cell counts in SCC and normal mucosa.

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Background

Mast cells normally present in small numbers in the connective tissue of all organs and more particularly, around blood vessels and nerves in the dermal layer of skin and surface of lung mucosa, and digestive system (1-3). Their sizes are ranging from 5 to 15 µm in diameter, and in histologic sections appear ovoid or spindle-shaped cells with cytoplasmic granules. They release a variety of potent mediators like histamine, leukotrienes, and cytokines, chymase, basic fibroblast growth factor (bFGF), tryptase, heparin through degranulation (1,4). It is considered that mast cells have conflicting dual roles from defending against tumors to causing tumor progression. Of course, some of their products induce angiogenesis in tumors (5-7). Oral squamous cell carcinoma (OSCC) remains a serious problem of oral health worldwide and has a complex biological behavior and despite the advances in the treatment modalities, the 5-year survival rates of the patients with OSCC have improved only slightly (8). Sharma et al showed that mast cells have a role in up-regulation of tumor angiogenesis in OSCC (9). The contribution of mast cells to angiogenesis during the progression from oral hyperkeratosis without dysplasia to oral hyperkeratosis with dysplasia or OSCC is not clear yet due to conflicting results within the literature (5).

Highlights

- Mast cells density increased in hyperkeratosis, dyspkasia, and OSCC.

Mohtasham et al found significant differences between the numbers of mast cells in oral dysplastic mucosa and OSCC (10).

Bivji showed an increase in the number of mast cells/unit microscopic field in oral leukoplakia compared to normal mucosa (11). Pazouki et al observed an increase in vascularization during the transformation from normal oral mucosa, through dysplasia, to in-situ and infiltrating carcinoma supporting the pivotal role of angiogenesis in malignancy progression (12). Flynn et al (13) demonstrated a direct correlation between sequential mast cell infiltration, activation and distinct stages of hyperkeratosis, dysplasia, carcinoma in-situ in the oral cavity and implicated the role of mast cells in configuring the angiogenic phenotype in premalignant lesions. Similarly, Iamaroon et al also observed a linear increase from normal oral mucosa, hyperkeratosis, premalignant dysplasia to squamous cell carcinoma suggesting the role of mast cells in upregulation of angiogenic process (14).

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