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Role of Mast Cell in Oral Pathology

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ABSTRACT

In oral tissues,the mast cell releases various pro-inflammatory cytokine tumor necrosis factor alpha (TNF-Ü) which stimulate leukocyte infiltration in various inflammatory condition of oral cavity such as oral lichen planus (OLP), periapical lesions, gingivitis & periodontitis. Mast cells are the local residents of the connective tissue and plays an important role in immunopathology and producing mitogenic cytokines.These cells plays a role in pathogenesis of oral diseases.

Keywords: Oral Pathology, Inflammatory.

INTRODUCTION

Mast cells are granulated cells and appears as a large spherical cells which is present in all of the connective tissue elements including skin, submucosa or connective tissue of various organs and mucosal epithelial tissues & also in dental pulp[1,2]. Life span of weeks to months.It is derived from bonemarrow and it contains granules granules are rich in heparin, chondroitin sulphate, proteoglycan and numerous enzymes including collagenase.These granules are metachromatic in nature and it is confirmed with staining such as Toludine blue.

Ultrastructure of Mast Cell

Three types of mast cell

1. In deeper connective tissue,the cells (except that in close vicinity to blood vessels) appears as round /oval in shape & dark purple in colour. The borders of cell are well defined & nucleus is not visible due to granules called as intact cells[3]
2. In the superficial connective tissue, the mast cells appear flattened / irregular and the cytoplasm appears granular immediately below the infiltrate area and near the blood vessels . The cell borders are not defined and the nucleus is only partially appreciable; these are called spreading cells[4].
3. The third type called degranulated cells found within the infiltrate & appeared paler the staining has reverted from metachromatic violet to light pink, the nucleus blue in color and well defined[5]

Role of mast cell released cytokines

IL-3 – induce basophil recruitment & activation
IL-5-eosinophil recruitment & activation
IL-13 – induction of IgE synthesis

Mast cell bears receptors for IgE and degranulates this cytophilic antibody is cross-linked by antigen . Mast cell RANTES degranulation can be caused by other factors such as mechanical trauma, complement C5a, eosinophil –derived cationic protein, and bacterial products . In the

absence of IgE mediated activation the mast cell can produce inflammation and its events under many conditions[6].

Thus, mast cell release proinflammatory mediators, promotes inflammation and angiogenesis, extracellar matrix degeneration and tissue remodeling.

Role of MC in Oral Pathologies

1. Role of MC in OLP:

Mechanism:

RANTES activation

↓

Mast Cells chemotaxis and degranulation

TNF- Ü

Endothelium cell adhesion molecule expression (Cd62 E, CD54, CD106)

Chymase activation

MMP- 9 activation

Disruption of basement membrane

Keratinocyte apoptosis

Intra epithelial CD 8
T-cell migration through BM

Survival of inflammatory cell

Non-specific T-cell recruitment[1]

Action of mast cell mediators in oral lichen planus leading to the following clinical and histopathological changes[7]

2. Action of mast cell mediators in oral submucous fi brosis leading to the following clinical and histological changes[7]

3. Role of MC in angiogenesis in oral squamous cell carcinoma:

Action of mast cell mediators in oral squamous cell carcinoma leading to the following clinical and histological changes[7]

4. Role of MC in oral leukoplakia:

Action mast cell mediators in oral leukoplakia leading to the following clinical and histological changes[7]

5. Role of MC in Periapical lesions:

Mechanism:

MC releases

Histamine

Increase vascular permeability of small blood vessel

6. Role of MC in Pyogenic Granuloma:

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MC + neuropeptides neural immune network with LC in mucosal tissue

Degranulation of MC

Cytokine, vasoactive amine and enzyme

Inflammatory and vascular changes

Pyogenic granuloma

7. Role of MC in wound healing:

Wound healing is a dynamic process consisting of four phases:

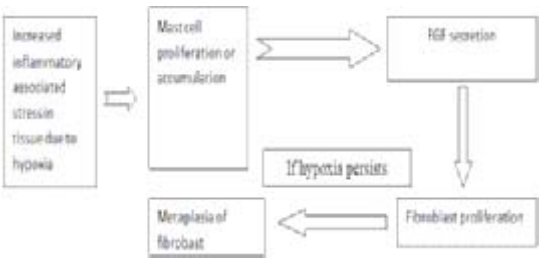

Table 1:

Mediators	Clinical features	Histopathologic features
Histamine. Induces vasopermeability... Antigen induced T-cell proliferation Inhibits the neutrophil.. Induces increased expression of E-selectin, ICAM and ICAM, whichcauses leukocytic margination	Vesicles, bullae and erosive lesionsChronic persistence of the lesion.	Submucosal edema. Traffi cking of T-lymphocytes..
TNF-alpha. Increased production of matrix metalloproteinases like stromyelsin, collagenase. Destruction of basement membrane	Vesicles, bullae and erosive lesions	Necrosis and liquifactive degeneration of basal cell layer.

Table 2:

Mediators	Clinical features	Histopathologic features
Prostaglandins and leukotreines Increase the mucous gland secretion Increased venous permeability	- Excessive salivation.	Submucosal edema
Histamine Causes vasodilatation and vasopermeability.		Submucosal edema
Heparin. Causes vasoproliferation. -	Petechiae	
Interleukin-5. Causes growth and differentiation of eosinophils	Itching sensation.	In early stages of oral submucous fibrosis consists of Inflammatory infiltrate eosinophils
Eosinophilic chemotactic factor (ECF) Causing eosinophilic migration		
Interleukin-1. Stimulates fibroblastic proliferation.	Decreased mouthopening	Increased collagen fiber bundles.

Table 3 :

Mediators	Clinical features	Histopathologic features
IL-1 AND TNF-alpha. Causes increased epithelial cell proliferation..	Exophytic growth or a plaque	Increased thickness of the epithelium
Heparin. Causes angiogenesis and type-VIII	Tumour angiogenesis	Increased vascularity of the stroma.

Table 4 :

Mediators	Clinical features	Histopathologic features
Histamine. Enhances permeability across the epithelial surface. Antigen induced T-cell proliferation.	Chronicity of the lesion	Increased mucosal permeability despite hyperkeratosis
Heparin. Causes endothelial cell proliferation and migration	Erosive leukoplakia.	Increased vascularity of the stroma and ulceration.
Interleukin-1 and TNF. Increased epithelial cell proliferation.	White patch or a plaque	Increased thickness of the epithelium.

a. Homeostasis
b. Inflammation
c. Proliferation
d. Tissue remodeling and resolution

I. MC activate fibroblast enzyme tryptase

synthesis collagen + hyaluronic acid

fibrous tissue formation

II. MC + fibroblast13

fibronectin integrin

receptor

Wound healing

Wound healing involves degradation, cell migration, synthesis of fibronectin, fibrin and high amount of collagen type II and matrix remodeling to return the tissue to normality

III. MMP – 9 from mast cell Wound healing[1]

8.Role of mast cell in peripheral ossifying fibroma[9]:

CONCLUSION

This article mainly focus that all oral reactive lesions have MCs , thus having a possible role in pathogenesis of these lesions.In recent years. Mast cells have gained a lot of importance owing to the vast number of chemical mediators released by them with wide range of actions in various disease processes. Once confirmed, it makes easier for us to target the therapeutic modalities against mast cells and the granules it contains to alter the course of disease/lesion.

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