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Minha Majeed Kak
Post Graduate Student, Department of Oral and Maxillofacial Pathology and Microbiology, Kothiwal Dental College and Research centre Moradabad, Uttar Pradesh, India

Jharna Bharali
Post Graduate Student, Department of Periodontology, Kothiwal Dental College and Research Centre Moradabad, Uttar Pradesh, India

Piranha Rastogi
Professor and Head, Department of Oral & Maxillofacial Pathology and Microbiology, Kothiwal Dental College and Research Centre Moradabad, Uttar Pradesh, India

Krishna Kumar Chaubey
Professor and Head, Department of Periodontology, Kothiwal Dental College and Research Centre Moradabad, Uttar Pradesh, India

Corresponding Author:
Minha Majeed Kak
Post Graduate Student, Department of Oral and Maxillofacial Pathology and Microbiology, Kothiwal Dental College and Research centre Moradabad, Uttar Pradesh, India

Role of mast cells in periodontal health and disease: A comparative study

Minha Majeed Kak, Jharna Bharali, Priyanka Rastogi and Krishna Kumar Chaubey

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Abstract
Background: Mast cells (MC) are mobile secretory cells containing granules which are distributed around the microvascular endothelium in the oral mucosa and dental pulp having diverse biological functions which include phagocytosis, antigen processing and production of cytokines and release of a variety of preformed and newly formed physiological mediators. Mast cells are also involved in tissue injury and repair, allergic inflammation and host defense due to the presence of these mediators. The significant contribution of mast cell mediators to tissue damage and propagation of inflammatory responses make the control of mast cell activity vital to the management of many inflammatory diseases. As Mast cells have been shown to be capable of eliciting immune and inflammatory responses in Periodontal Diseases, this study was conducted to quantify the Mast cells in healthy and diseased gingiva and also whether they correlate the degree of inflammation and clinical features of Periodontium.
Materials and Methods: A total of 30 soft tissue samples 10 each of Normal healthy Gingiva, Dental Plaque induced Gingivitis (DPIG) and Chronic Periodontitis (CP) were selected. Samples were obtained from patients undergoing periodontal surgery in CP and DPIG. In healthy (Control) group patients undergoing crown lengthening procedure were selected. Samples were fixed in 10% buffered formalin and stained with 1% Toludine blue stain and observed under light microscope for the presence of Mast Cells and Mean Mast Cell Index was calculated. Results obtained were statistically analyzed and interpreted
Results: The Mean Mast Cell index was found to be highest in Chronic Periodontitis Group followed by Dental Plaque induced Gingivitis Group than Normal healthy Gingiva. Quantitative analysis of Mast cells with Toludine blue staining revealed statistically significant difference among the three groups examined.
Conclusion: Increase in the number of Mast Cells in Human Periodontal Diseases indicates the importance of these cells in the progression to Chronic Periodontitis.

Keywords: Mast cells, mast cell index, gingivitis, chronic periodontitis, dental plaque

Introduction
The primary etiological factor in inflammatory periodontal disease has been attributed to the Bacterial plaque, but recently studies have focused on and revealed the role of the immune system in the evolution of periodontal disease, indicating that bacterial antigens trigger an immunopathological reaction and that the immune susceptibility of the patient determines the utmost outcome of the disease process [1]. Mast cells have been detected in both healthy and diseased gingiva, in different numbers at various sites among the other cells found in the periodontal tissues [2]. Mast cells (MC) are mobile secretory cells containing granules which are distributed around the micro vascular endothelium in the oral mucosa and dental pulp [3]. They have diverse biological functions which include phagocytosis, antigen processing and production of cytokines and release of a variety of preformed mediators (e.g., Histamine, Proteoglycans and Proteases) and newly formed physiological mediators (e.g., Leukotrienes and Prostaglandins) [4, 5]. Mast cells are also involved in tissue injury and repair, allergic inflammation and host defense due to the presence of these mediators.

The significant contribution of mast cell mediators to tissue damage and propagation of inflammatory responses make the control of mast cell activity vital to the management of many inflammatory diseases [6]. The aim of this study was to quantify the mast cells in healthy and diseased gingiva also, whether they correlate the degree of inflammation and clinical features of Periodontium.

Materials and Methods

This study was done in department of oral and maxillofacial pathology, Kothiwal dental college and research centre. A total of 30 cases 10 cases each of Chronic Periodontitis (CP) characterized by advanced loss of Periodontium, 10 cases of dental plaque induced gingivitis (DPIG) and 10 cases of clinically healthy gingival tissues as control were selected who reported to the Department of Periodontics Kothiwal Dental College and research centre. The American Academy of Periodontology guidelines were followed for classification of the periodontal disease and conditions [7]. The patients with no history of systemic disease were included in the study. In control group patients undergoing crown lengthening were included.

1. **Group I:** Ten tissue samples with periodontally healthy tissues (PD<3 mm with no bleeding on probing) (Fig 1)
2. **Group II:** Ten tissue samples with plaque- induced gingivitis (PD < 3 mm and CAL < 1 mm with bleeding on probing) (Fig 2)
3. **Group III:** Ten tissue samples with moderate to advanced CP (PD and CAL > 4 mm with bleeding on probing) (Fig 3).

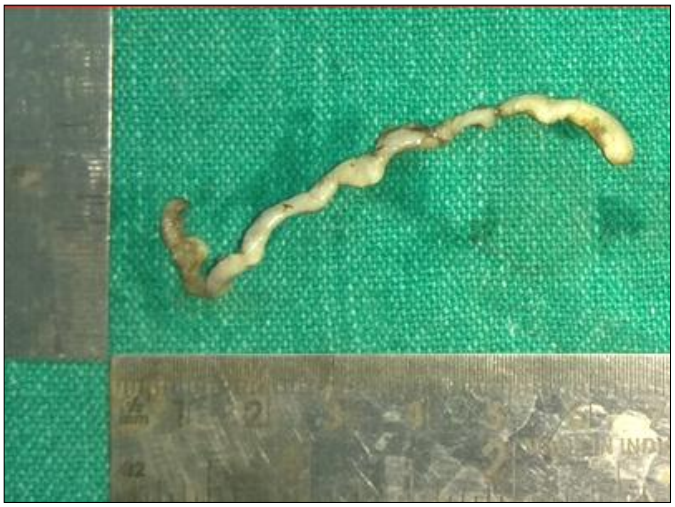


Fig 1: Normal gingiva

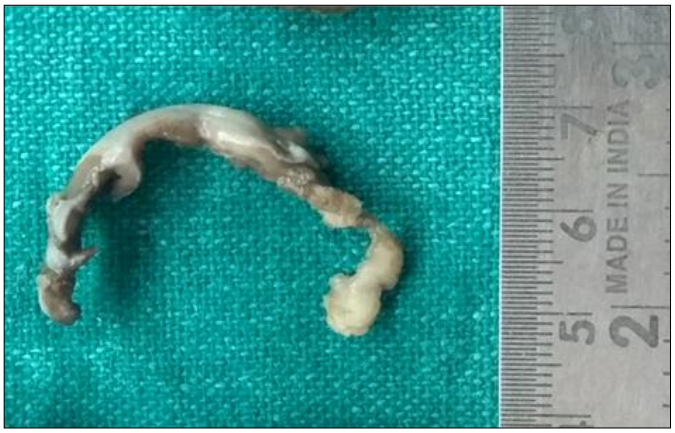


Fig 2: Plaque induced gingivitis

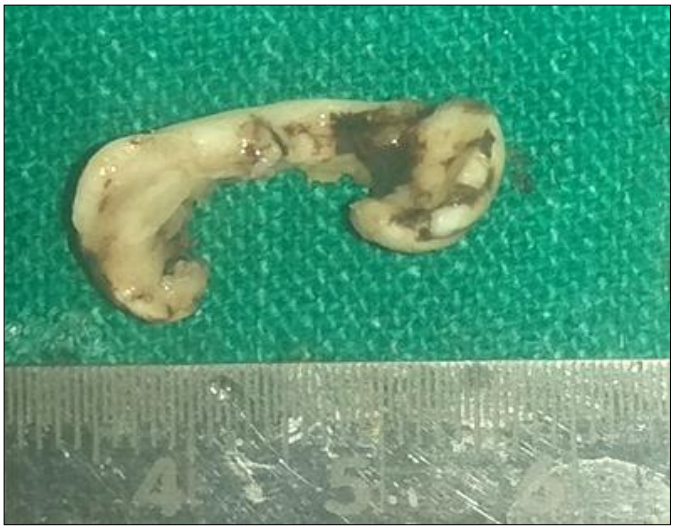


Fig 3: Chronic periodontitis

Tissue samples were obtained from patients' undergoing periodontal surgery in CP and DPIG. Independently of this study, each patient underwent periodontal surgery, as a part of their routine periodontal treatment by one surgeon in an identical manner and technique for all cases. Using No 15 BP blade, Incisional biopsy was done from suitable sites immediately after diagnosis. The specimens were immediately fixed in 10% buffered formalin and sent to Department of Oral and Maxillofacial Pathology for further histopathological processing.

Histochemistry and staining

After routine tissue processing, tissue sections of 5 µm thickness were obtained and deparaffinized and hydrated to distilled water and then stained with 1% Toludine Blue (TB) stain using manufactures guidelines. Sections were then blotted carefully and dehydrated through increasing grades of ethanol to absolute alcohol to xylene and mounted in DPX and observed under light microscope.

Interpretation of Toluidine Blue Staining

For each section, fields devoid of any preservation or fixation artifact and necrosis were selected. Mast cells were then counted manually in 30 high power fields (HPF), under a magnification of 40X in a stepladder fashion without overlapping of the fields. Mast cells Index (MCI) was expressed as an average number of mast cells per 30 high power fields [8] as indicated in the formula below.

$$\text{Mast Cell Index} = \frac{\text{Total number of Mast cells}}{\text{30 (No. of high power fields)}}$$

The result thus obtained was subjected to Statistical Analysis using ANOVA followed by Student's *t*- test. A *P* value of less than 0.05 was considered statistically significant. Post – Hoc Bonferroni Test was performed for multiple comparisons between the groups.

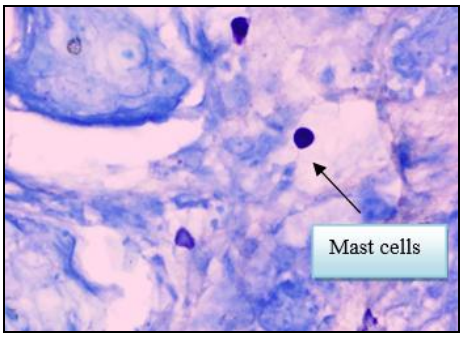
Result

The Mean Mast Cell Index (MCI) (Table 1) was found to be 4.14± 0.25(Mean ± SD) in Group I (Photomicrograph-1), 7.49 ± 0.69 (Mean ± SD) in Group II (Photomicrograph-2), 8.90 ± 1.07 (Mean ± SD) in Group III (Photomicrograph-3). The Mean Mast Cell Index in all the groups when compared using parametric version of ANOVA (Analysis of Variance)

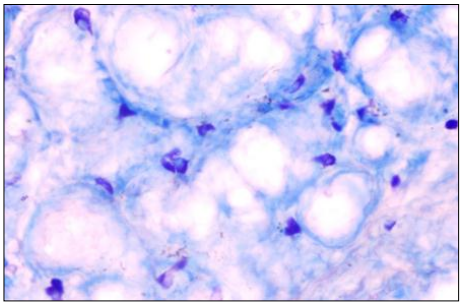
indicated a statistical significant difference with a p value of < 0.001. Thus, quantitative analysis of mast cells with Toludine blue staining revealed statistically significant difference among the three groups examined. Post – Hoc Bonferroni Test was performed for multiple comparisons between the groups after the application of ANOVA test. A statistically significant difference was found between Group I and II with a p value of 0.046. A very highly statistical significant difference was found between group I and III with a p value of < 0.001. Thus, the present study findings indicated a Statistically Significant increase in Mean MCI in Chronic Periodontitis (Group III) followed by Dental Plaque Induced Gingivitis (Group II) when compared no normal gingiva (Group I) as control.

Table 1: Indicating the comparisons of Mean Mast Cell Index amongst all the Groups using ANOVA.

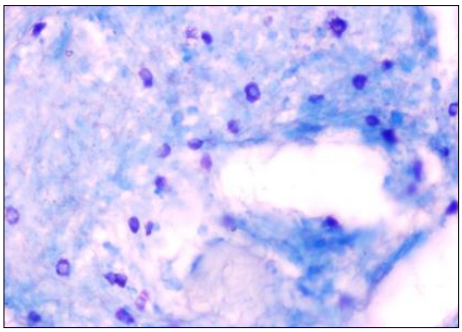
Group (n = 10)	MCI		p – Value
	Mean	SD	
GROUP I	4.140	0.2591	< 0.001*
GROUP II	7.490	0.6903	
GROUP III	8.900	1.0750	



Photomicrograph 1: Mast cells



Photomicrograph 2



Photomicrograph 3

Discussion

Mast cells (MCs) are portions of the innate and adaptive immune system derived from bone marrow (BM) progenitors that are rich in cytoplasmic granules. MC maturation, phenotype, and function are determined by their microenvironment. MCs accumulate at inflammatory sites associated with atopy, wound healing, and malignancies. They interact with the external environment and are predominantly located in close proximity of blood vessels and sensory nerves [9]. When viewed under light microscopy, human MCs usually appear as round or oval in shape with a diameter ranging between 8 and 20 μm depending on the organ in which they are studied [10]. Their nucleus is round or ovoid and their cytoplasm contains numerous secretory granules that are metachromatic with Toluidine blue [11].

MCs are key initiators and modulators of allergic, anaphylactic, and other inflammatory reactions, by induction of vasodilation, promoting of vascular permeability, recruitment of inflammatory cells, facilitation of adaptive immune responses, modulation of angiogenesis, and fibrosis. They express a wide range of receptors, e.g., for IgE (FcεRI), IgG (FcγR), stem cell factor (SCF), complement (including C5aR), and cytokines, that upon activation trigger various signaling pathways [5].

The numbers of mast cells increased in inflamed and healing gingival as discussed by Gunhan, *et al.* [12]. Mast cell numbers were dramatically increased in inflamed sites of periapical granulomas and lichen planus when compared with non lesional sites indicating higher activity of the cells in that area as demonstrated by Walsh *et al.* [13] Histamine, one of the biological and biochemical derivatives of mast cells breaks down the tissue barrier, causes edema and helps cellular infiltration [14]. Also, the expression of matrix metalloproteinases (MMPs) 1, 2, and 8 are strongest in mast cells that are crucial in the degradation of the main components in extracellular matrices [15]. As the Mast cell count in the present study was found to be higher in Chronic Periodontitis than normal healthy gingival suggesting that mast cell counts may be associated with periodontitis leading to the periodontal tissue breakdown.

Furthermore, tryptase another biological and biochemical derivatives of mast cells is believed to have its activity confined to mast cell granules and can cleave the third component of collagen and activate latent collagenase that can participate in tissue destruction in periodontitis. A marked change from gingivitis to periodontitis involves a shift from predominantly T^- cell lesion to a B^- cell lesion. Mast cells seem to be able to present antigens to T cells. The resultant T^- cell activation would activate mast cells, leading to both degranulation and cytokine release [16].

In the present study, an increase in number of mast cells in inflamed site as compared to periodontally healthy sites suggested important dynamic alterations in the migration and localization of mast cells in the evolution of periodontal disease. The significance of the distribution of Mast cells in tissue compartments relates to their influence on nearby cells with resulting stimulatory, inhibitory or toxic effects [3]. The participation of mast cells in the defense mechanism and destructive events both as effector and responsive cells in chronic inflammation, as well as the possible functional populations in periodontal lesions is still debatable. Thus, from the study it was concluded that periodontitis is not unidirectional, but rather it is an interactive one as the same Mast cells that produce the destructive pro inflammatory cytokines can also produce mediators that activate the healing process.

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