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## Mast Cell: A periodontal inflammatory marker

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### Abstract

**Background:** Among the cells involved in immune and inflammatory responses in periodontal disease, mast cells have been shown to be capable of generating a large number of biologically active substances. Mast cells are mobile, bone-marrow-derived, granule-containing immune cells that are found in all connective tissue and mucosal environments and in the peripheral and central nervous systems. Mast cells are able to phagocytose, process and present antigens as effectively as macrophages. The present study was undertaken to quantify the mast cells in health and disease, whether they correlate degree of inflammation and clinical features of periodontitis

**AIM:** The aim of this study was to evaluate the distribution, and their relationship with the degree of inflammatory infiltrate in gingiva from patients with periodontal disease.

**Source:** Forty patients (OPD, DEPT. OF Periodontics). 10 each: Aggressive periodontitis, Chronic Periodontitis, gingivitis and healthy mucosa.

**Materials And Methods:** Gingival specimens of 10 chronic cases, 10 aggressive periodontal cases, 10 gingivitis cases for experimental group and 10 healthy cases as control group in routine periodontal flap and crown lengthening procedures. Specimens: stained by toluidine blue for mast cell counting and hematoxyline/eosin to assess inflammation. Inflammation and mast cells were assessed in 40 x magnification.

**Results:** Mast cell counts were found to be higher in aggressive and chronic periodontitis than healthy/gingivitis cases.

**Conclusion:** Mast cell number is directly proportional to inflammation of tissue.

**Keywords:** Mast Cell, periodontal inflammatory marker

### Introduction

Bacterial plaque has been implicated as the primary etiological factor in inflammatory periodontal disease, but recently several studies have focused on the role of the immune system in the evolution of periodontal disease, indicating that bacterial antigens trigger an immune pathological reaction and that the susceptibility of the patient determines the ultimate outcome of the disease process <sup>[1]</sup>. Mast cells are large granular cells that arise from a multipotent CD 34+ precursor in the bone marrow and are normally distributed throughout connective tissues <sup>[2]</sup>. Mast cells are also known as "unicellular endocrine glands" due to their ability to release a wide variety of chemical mediators that have a potent biological action on target tissues. In inflammatory lesions of the oral cavity, neo-vascularization and presence of inflammatory cells is an expected finding <sup>[3]</sup>. The aim of this study was to evaluate the distribution, and their relationship with the degree of inflammatory infiltrate in gingiva from patients with periodontal disease.

### Materials and Methods

The study was undertaken after approval from the Institutional Ethical Committee. Patients were selected from the outpatient department of Department of Periodontology and Implantology, BIDS, Patna. Informed consent was taken from the patient after explaining the biopsy procedure. Forty patients, 10 each of Aggressive periodontitis, Chronic Periodontitis, gingivitis and healthy mucosa were selected. The patients had no systemic diseases and had not used any medications with probable effects on periodontal tissues for the previous 2 months; they were non-smokers with no special hormonal conditions, such as pregnancy, menopause, menstruation or puberty. All the subjects signed a consent form and their oral cavities were

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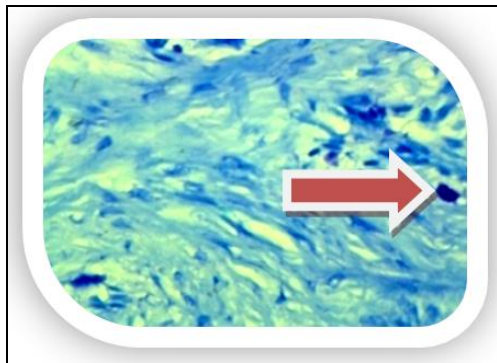
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examined by two observers. The clinical examination, including Ture-sky-Glimore-Glickman plaque index (PI), Modified Loe and Silness gingival index (GI), probing depth (PD), clinical attachment loss (CAL) and bleeding on probing (BOP) were recorded using a Williams probe. Four groups were allotted:-

1. 10 samples with healthy tissues (PD less than 3 mm and CAL less than 1 mm)
2. 10 samples with gingivitis or gingival overgrowth (PD less than 4 mm and CAL less than 1 mm)
3. 10 samples with moderate-to-advanced chronic periodontitis (PD and CAL more than 4 mm with BOP)
4. 10 samples with moderated-to-advanced aggressive periodontitis (PD and CAL more than 4 mm with BOP)

### Immunohistochemistry

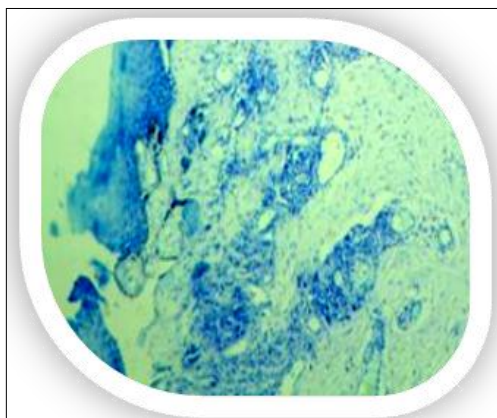
The biopsied specimens were immediately fixed in 10% neutral buffered formalin for further processing. Two tissue sections of 5 µm thickness were cut from each tissue. One section was stained with hematoxylin and eosin and categorized to the particular groups and the second section was stained with special stain, i.e., 1% toluidine blue used specifically to demonstrate mast cells.



**Fig 1A:** Histological Section of Normal Gingiva



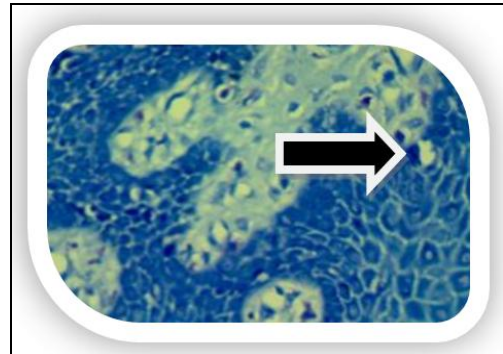
**Fig 1B:** Clinical Section of Normal Gingiva



**Fig 2A:** Histological Section of Normal Gingivitis



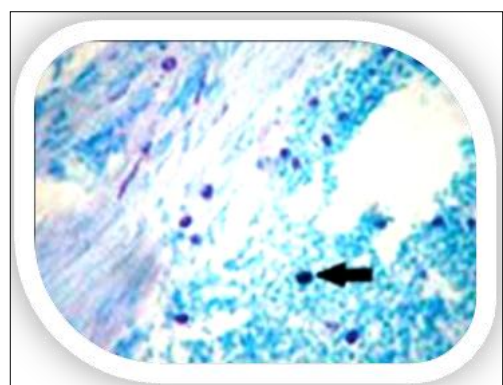
**Fig 2B:** Clinical Section of Gingivitis



**Fig 3A:** Histological Section of Chronic Periodontitis



**Fig 3B:** Clinical Section of Chronic Periodontitis



**Fig 4A:** Histological Section of Aggressive Periodontitis



**Fig 4B:** Clinical Section of Aggressive Periodontitis

## Results

On the whole 10 chronic periodontitis, 10 aggressive periodontitis and 10 healthy and 10 gingivitis biopsies were obtained from total of 29 subjects.

The mean of the clinical parameters (PD and CAL) of the four groups are presented in Table 2.

BOP was positive in all the sites of chronic and aggressive periodontitis (case groups) and 8 sites of healthy/gingivitis (control group). PI was under 2 in all the sites and GI was between the grades 1 and 3.

The results of the analyses of mast cell counts between the four groups are shown in Table 1

There were significantly more mast cells in the aggressive

periodontitis cases as compared to chronic periodontitis cases and the healthy/gingivitis ones ( $P = 0.000$ ); however, the chronic periodontitis cases did not show higher counts as compared to the healthy/gingivitis cases ( $P > 0.05$ ).

In addition, there was no relationship between mast cell counts and degree of inflammation in the four groups.

**Table 1:** Means of mast cell in four study group

Healthy	Gingivitis	Chronic periodontitis	Aggressive periodontitis	P value
10.8±7.6	18.9± 1.3	13±9.8	34.5±23.1	> 0.05 for AP
10.8±7.6	18.9± 1.3	13±9.8	34.5±23.1	

**Table 2:** The mean probing depth and clinical attachment loss of four groups

Characteristics	Control (n=10)	Gingivitis (n=10)	Localized chronic periodontitis (n=10)	Localized Aggressive Periodontitis (n=10)	P value
PD (MM)	1.41±0.42	2.10±0.00	5.25±0.38	6.15± 0.23	>0.05 for AP
CAL (MM)	0.00±0.00	0.00±0.00	4.63±0.47	5.24± 0.38	

## Discussion

Mast cells originate from pluripotential hematopoietic cells in the bone marrow, undergo part of differentiation in this site, then enter the circulation and complete their differentiation in peripheral mucosal or connective tissue microenvironments<sup>[4]</sup>. Mast cells are scattered throughout gingival connective tissue, often in close association with endothelial cells, but are also found sub and intraepithelially. In inflamed and in healing gingiva, number of mast cells are found to be increased. Mast cells are characterized by oval to round nuclei with the cytoplasm densely packed with bright red granules can be stained with Giemsa stain or toluidine blue stain.

When activated, mast cells may either undergo explosive degranulation and then resynthesize their granules or they may release solitary granules into their environment on an ongoing basis, a process termed "piecemeal degranulation" that has been observed in both the oral mucosa and skin.

### Mast cell mediators

Following degranulation, mast cell mediators are deposited in large quantities in the extracellular environment, where they exert effects on endothelial cells and other cell types. Mast cells may subsequently synthesize and secrete additional mediators that are not preformed in their granules. As described by Walsh, *et al.*, 1991b key mediators that are preformed in mast cells are the serine proteases tryptase, chymase and cathepsin G, histamine, heparin, serotonin, acid hydrolases, and the cytokines tumor necrosis factor-  $\alpha$  (TNF) and interleukin- 16<sup>[5]</sup>. Following activation, mast cells can synthesize a range of mediators, including the interleukins IL- 1, IL- 3, IL- 4, IL- 5, IL- 6, IL- 8, IL- 10, IL- 13, and IL- 16, together with granulocyte- macrophage colony- stimulating factor GM- CSF), platelet- activating factor (PAF), RANTES, macrophage inflammatory protein (MIP- 1a), and the arachidonic acid metabolites prostaglandin 2 and leukotriene C4<sup>[5]</sup>. As discussed by Gunhan, *et al.*<sup>[6]</sup> in inflamed and in healing gingiva, numbers of mast cells increased. Walsh, *et al.*, have showed that 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 that cells in that area.

The results of this study suggest that mast cell counts may be associated with periodontitis. This finding indicates the role of mast cells in chronic periodontal tissue breakdown. One of

the biological and biochemical factors is histamine, which breaks down the tissue barrier, causes edema and helps cellular infiltration. In addition, mast cells are believed to contain most of the body's histamine<sup>[7]</sup>. Another reason is that the expression of matrix metalloproteinases (MMPs) 1, 2, and 8 are strongest in mast cells. MMPs are crucial in the degradation of the main components in extracellular matrices<sup>[8]</sup>. Furthermore, tryptase can cleave the third component of collagen and activate latent collagenase that can participate in tissue destruction in periodontitis. Furthermore, it has been indicated that tryptase activity is confined to mast cell granules. A change from gingivitis to periodontitis involves a shift from predominantly T- cell lesion to a B- cell/plasma 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.

In our study mast cell count is more predominant in aggressive periodontitis which is in contrary to vahabi *et al.*,<sup>[9]</sup> Based on our results, the increase of mast cells has called attention with respect to the possible participation of mast cells in the defense mechanism and destructive events both as effector and responsive cells in chronic inflammation.

## Conclusion

The possible relationships between mast cells and pathogenesis of periodontal diseases has been the goal of this study. In the present study AP cases had higher mast cell counts compared to gingivitis sites or healthy tissues. The therapeutic implications of the findings and suggestions here in presented include strategies directed toward the possible use of drugs to influence mast cell secretion and there by prevent inflammation and maintenance of chronicity or even with the aim of improving periodontal regeneration.

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