

Bullon P, Fioroni M, Goteri G, et al. Immunohistochemical analysis of soft tissues in implants with healthy and peri-implantitis condition, and aggressive periodontitis. Clin Oral Impl Res 2004;15:553-9. (36 Refs)

Purpose: To describe the inflammatory changes in peri-implantitis and provide data about the pathogenesis

Materials and Methods: During a 2-year period of observation, five cases of peri-implantitis, showing radiographic bone loss, presence of a peri-implant pocket deeper than 5mm, bleeding on probing and swelling of tissues, formed the peri-implantitis group (PG). This group was compared with five cases of healthy peri-implant tissues (HG-no clinically visible plaque accumulation, no probing depth greater than 2mm, and no bleeding on probing) and five cases of aggressive periodontitis (AG). Biopsies were taken from interproximal area of supracrestal peri-implant tissue in PG and the sites with at least 6 mm of probing depth in AG. In HG, the biopsy specimens were obtained during esthetic gingival remodeling interventions around implant abutments. All biopsy specimens were sectioned into two parts to provide the oral (O) and sulcular-junctional (S-J) sides, and then prepared for histological and immunohistochemical analysis. Information regarding the occurrence of systemic illness, smoking status, and the presence of periodontal disease was gathered in all cases. Plaque general index, plaque peri-implant index and implant data were recorded.

Findings and Conclusions: The mean value of plaque index was 9.7% in HG and 51.3% in PG. In HG, only some clusters of inflammatory cells were found in the connective tissue, but in PG a prominent inflammatory infiltrate was present in the lamina propria. In HG, B and T lymphoid cells were rarely observed in the basal-parabasal layer in the S-J and in the O side. On the other hand, B and particularly T cells were abundant in PG and AG cells. The most important differences were established when comparing HG vs. PG and HG vs. AG, but not compared PG vs. AG, for T cells. CD1a-positive cells were observed more frequently in the O, located in the basal-parabasal layers, than in the S-J side. Significant differences were obtained comparing AG vs. HG and AG vs. PG in the O side, but no significant differences were found among the three groups in the S-J side. Vascular proliferation analyzed by immunoreactivity for CD34, factor VIII, and vascular endothelial growth factor was more prominent in PG comparing with HG and AG in the S-J side. Oncoprotein bcl-2 and p53 staining for apoptosis revealed no significant difference in the three groups. In conclusion, above data support the hypothesis that the osseointegration loss process in implant failure is due to an inflammatory mechanism that has some similarities in the types of inflammatory cells, but not in the vascular proliferation pattern, to the features present in aggressive periodontitis.

Steinsvoll S, Helgeland K, Schenck K. Mast cells- a role in periodontal disease? J Clin Periodontol 2004;31:413-9.

Purpose: A literature review is conducted to examine a possible role of mast cell in periodontal disease.

Materials and Methods: Author's research and experience and published literature. Background: Mast cells originate in bone marrow, and stem cell factor finalizes their phenotypic differentiation in peripheral mucosal or connective tissue. Like macrophages, mast cells are able to phagocytose, process, and present antigen effectively. Mast cells perform a pivotal role in mucosal inflammation, host defense and tissue repair. The process of periodontal infection began as a deficiency in immune elimination process (periodontal tissue failed to clear away invading agents). Chronic periodontal disease is initiated as imbalanced of pro- and inflammatory activities linger on.

Findings and Conclusion: Studies have found an increasing number of mast cells in inflamed and healing of periodontal tissue. Mast cells express key enzymes, and release a large quantity of mediators when activated by cytokines or bacterial products. Cytokines (TNF- α) and matrix metalloproteinases (MMP) are implicated in periodontal soft tissue degradation. Mast cell released cytokine (TNF- α) functions as a part of inflammation and host defense was indicated in a study with mast cell deficient mice. TGF- B_1 is a chemoattractant for neutrophils, monocytes and mast cells. This factor is a suspected to take part in formation of inflammatory process of chronic periodontitis. Studies found gingival mast cells were major producers of MMP. MMP have roles in cell migration, wound healing, tissue remodeling and pathogenic role in periodontal and mucosal diseases. The MMP release may lead to rapid extracellular matrix degradation, and periodontal destruction. Mast cell is an important cell in periodontal disease and it needs to be examined further.