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Editorial

NSAID's-periodontal considerations and host modulatory therapy

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Host Modulatory Therapy (HMT) is currently one of the supplementary therapeutic options accessible for periodontal disease intervention. Initially, surgical procedures such as scaling and root planing (SRP) and other mechanical methods were used to treat periodontal disorders. Adjunctive treatments used to be limited to antimicrobials, such as systemic or local antibiotics and antiseptics. The host reaction is modulated in new adjunctive techniques.

The host side of the host-bacteria interaction can be treated using HMT. The majority of the tissue degradation that results in the clinical symptoms of periodontitis is caused by the host reaction. Through the management of some components of the chronic inflammatory response, HMTs provide the chance to modulate or lessen this damage. In order to improve the chances of wound healing and periodontal stability, HMTs reduce excessive or pathologically raised inflammatory processes rather than "switching off" normal defence systems or inflammation. When systemic HMTs are used to treat a patient's periodontal disease, there may be additional advantages for other inflammatory problems such diabetes, rheumatoid arthritis, osteoporosis, arthritis, and cardiovascular disease.

1. Role of Arachidonic Acid Metabolism in the Pathogensis of Periodontal Diseases

Metabolite of arachidonic acid influences most of the fundamental processes that make up inflammation. They are involved in pain and fever, oedema, enhanced vascular permeability, vasodilatation and constriction, and neutrophil and monocyte chemotaxis. Their mechanisms of action can include direct induction of biological effects, augmentation of the effects of other mediators, or induction or inhibition of the release or activation of other mediators that are important for the actions of arachidonic acid products. Cellular phospholipases that are triggered chemically, such as by exposure to the complement split product C5a, as well as physically, mechanically, and physically stimulated, release arachidonic acid from the membrane. Steroids have the ability to block release.

Cyclooxygenase transforms arachidonic acid into prostaglandins, prostacyclin, and thromboxane A2, whereas the lipoxygenase pathway produces the leukotriene family of chemicals. Although the lipoxins are a different family of arachidonic acid metabolites produced by PMNs, their characteristics are still a work in progress. Macrophages and fibroblasts, the primary source of prostaglandins in periodontal tissues, are stimulated by IL-1 to produce prostaglandins. Prostaglandins appear to have a significant role in alveolar bone resorption, according to compelling data. Alveolar bone loss is considerably reduced by systemic treatment of nonsteroidal anti-inflammatory

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medications that block prostaglandin synthesis and cyclooxygenase activity. Prostaglandin metabolism, in particular, plays a role in the tissue damage linked to periodontal disease. Inflamed periodontal tissue contains prostaglandins, particularly PGE2. [1]

2. Non Steroidal Antiinflammatory Drugs

NSAIDs lessen tissue inflammation by blocking prostaglandins. They are applied to many chronic inflammatory diseases, discomfort, and acute inflammation. Salicylates (like aspirin), indomethacin, and derivatives of propionic acid (like ibuprofen, flurbiprofen, and naproxen) are examples of NSAIDs. Researchers have looked into how well NSAIDs work in individuals with periodontitis to prevent PGE2 production, which lowers inflammation and inhibits osteoclast activity in the periodontal tissues.

When used as an HMT for periodontics, NSAIDs have several substantial drawbacks. They are known to cause major adverse effects, including as gastrointestinal issues, bleeding (due to decreased platelet aggregation), and impairment of the liver and kidneys. Reduced inflammation is the outcome of selective COX-2 medications inhibiting COX-2.

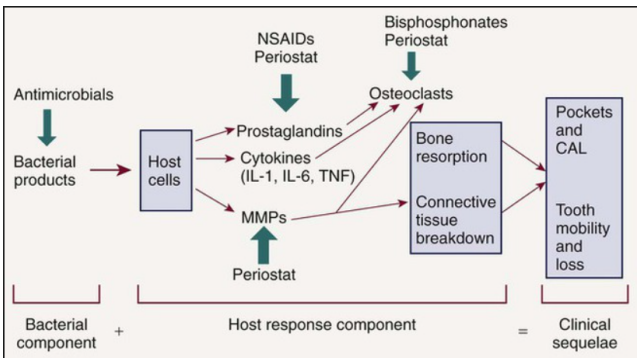

Figure 1:

3. Administration of Nasids in Periodontal Treatment [Animal studies]

According to a 1993 study by Bezzere et al., cytokines, growth hormones, and LPS stimulate the production of cyclooxygenase 2 (COX2), which raises prostaglandin levels. The possibility of lowering periodontal inflammation without the negative consequences of long-term non-selective NSAID use was made possible by selective COX2 inhibitors that inhibited COX2. Alveolar bone loss was delayed by COX2. [2]

Impact of an Anti-Inflammatory Therapy and Its Withdrawal on the Progression of Experimental Periodontitis in Rats was the subject of a 2004 study by Enilson A. Sallum et al. The following five treatment groups (15 animals per) were randomly assigned to the animals, and each group received daily subcutaneous injections: 1) 15 days of saline solution; 2) 45 days of saline solution; 3) 15 days of 3 mg/kg meloxicam; 4) 45 days of 3 mg/kg meloxicam; 5) 15 days of 3 mg/kg meloxicam followed by 30 days of saline solution. The observation of bone loss in the groups treated with selective COX-2 inhibitors is likely due to a partial suppression of arachidonic acid metabolites or prostaglandin production. According to the study's findings, selective cyclooxygenase-2 inhibitors may help slow the bone loss linked to experimental periodontitis, and once they are stopped, there shouldn't be any more effects. [3]

4. Administration of Nasids in Periodontal Treatment [Human Studies]

A clinical trial by Pinho MD et al. (2008) involved 60 patients with a diagnosis of periodontitis who were followed up for 28 days following periodontal therapy together with a selective cyclooxygenase-2 (COX-2) inhibitor. Scaling and root planning (SRP) plus the anti-inflammatory medicine loxoprofen (SRP+Loxoprofen) was administered to the experimental group, while SRP plus a placebo (SRP+placebo) was administered to the control group. SRP+Loxoprofen, the treatment strategy employed in this trial, improved clinical outcomes with a statistically significant decrease in PD and BOP from the baseline to the 14-day follow-up period. The study came to the conclusion that loxoprofen, an anti-inflammatory medication, has the potential to be used as an adjuvant to treat periodontal disease, and that it may reduce probing pocket depth more quickly and effectively than traditional periodontal therapy. [4]

15 patients with active moderate-to-severe periodontitis were enrolled by Jeffcoat et al., and they were given a 50 mg b.i.d. flurbiprofen or a placebo for two months. When comparing the patients treated with flurbiprofen to those treated with placebo over the course of the 2-month research period, radiography revealed noticeably reduced bone loss in the former group. Additionally, compared to the flurbiprofen-treated group, more tooth locations with bone loss were found in the placebo group. [5]

5. Conclusion

1. Even at modest dosages, aspirin has anti-platelet effects because it prevents the formation of thromboxane, which raises the risk of bleeding. In patients who do not have periodontal problems, this may exacerbate bleeding from the gums. Thus, it is not advised to utilise NSAIDs systemically or locally as an addition to traditional periodontal therapy given the costs and advantages.
2. Compared to traditional periodontal care, adjuvant anti-inflammatory therapy may result in a quicker and more significant reduction in probing pocket

depth. NSAIDs are useful in the treatment of chronic periodontitis because they are lipophilic and readily absorbed into gingival tissues.

3. Non-steroidal anti-inflammatory medications (NSAIDs) decrease the loss of alveolar bone by preventing the synthesis of metabolites from arachidonic acid; this therapeutic method may be used in conjunction with traditional mechanical therapy.

6. Conflict of Interest

None.

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