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# SHORT TERM EFFECT OF NON SELECTIVE CYCLOOXYGENASE INHIBITOR AS AN ADJUNCT FOR THE TREATMENT OF PERIODONTAL DISEASE

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# ABSTRACT

Aim: The purpose of this study was to evaluate a non selective COX-inhibitor derived from propionic acid, ibuprofen, as an adjunct for periodontal disease treatment. Materials and Methods: 30 patients with progressive periodontal disease entered this study in order to examine clinical effects of a non-steroidal anti-inflammatory drug - ibuprofen, used as an adjunct to non-surgical periodontal treatment. After scaling and root planning, patients were randomly assigned to either receive orally 200 mg of ibuprofen per day for one month (group B), or not receive the drug (group A). Results: The obtained results show that the mechanical periodontal treatment brought to resolution the gingival inflammation with both groups as displayed by clinical improvement in PD, PI and BOP. They also showed statistically similar values (p>0.05) of PD reduction on day 14 and on day 30. Conclusion: We may conclude that systemic ibuprofen had relatively minor influence on resolution of gingival inflammation in periodontal disease.

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#### INTRODUCTION

Periodontal disease, a high prevalent infectious injury is caused by specific bacterial species. However, most of the tissue breakdown is indirectly caused by toxic bacterial products that trigger the host response. Host derived enzymes known as matrix metalloproteinases (MMPs), changes in osteoclast activity by cytokines and other inflammatory mediators like prostanoids are responsible for most periodontal tissue destruction. The cyclooxygenase (COX) products of the arachidonic acid; prostanoids (PGs) and thromboxane (TxB2) are strong vasoactive mediators and stimulators of bone resorption. 1-3 Several host modulatory agents, non-steroidal anti-inflammatory drugs (NSAIDs), sub-antimicrobial dose of doxycycline and bisphosphonates or combination of drugs have been successfully used as an adjunct to non-surgical mechanical periodontal therapy. NSAIDs block the proinflammatory cytokines; sub-dose of doxycycline inhibit the metalloproteinases and the bisphosphonates block the osteoclast activity. 4,5 The basis of antiinflammatory drugs in periodontal disease treatment is related to the control of prostaglandin E2 (PGE2) associated with increased gingival inflammation and alveolar bone loss through the inhibition of enzyme cyclooxygenase. 1,3,6,7 Therefore purpose of this study was to evaluate a non selective COX-inhibitor derived from propionic acid, ibuprofen, as an adjunct for periodontal disease

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treatment, using probing pocket depth (PD) to verify the inflammation reduction.

# **MATERIALS AND METHODS**

# Sample Distribution and Study Design

This was a randomized, double blind parallel group study. 30 patients (14 males and 16 females) aged 28-50 years with at least 20 teeth, who had two or more teeth with clinical attachment loss (CAL) ≥6 mm and one or more sites with PD ≥5 mm were selected according to the established periodontitis criteria. Exclusion criteria were: presence of systemic diseases, pregnancy or nursing women, gastric ulcer, hypersensitivity to anti-inflammatory drugs, use of antibiotic, corticoids, immunosuppressive drugs or any NSAIDs within the past three months. The 30 patients were randomly assigned to experimental (group B) and control groups (group A). Group B received periodontal treatment that consisted of conventional scaling and root planning (SRP), using ultrasonic scaler and Gracey curettes at baseline combined with 200mg/day of ibuprofen for 1 month. Group A received SRP only. Periodontal treatment was performed by a blinded experienced periodontist (BP).

# Clinical Procedures, Measurements and Monitoring

At baseline, medical and dental histories were recorded and the following clinical parameters were measured: PD measured at 6 sites/teeth using an UNC 15 probe, bleeding on probing (BOP) and dental plaque (PI) (Table 1). All patients received

mechanical therapy (SRP), oral hygiene instruction and group B also received orientation about medication at baseline. Then, at the second appointment (2 weeks), clinical measurements were recorded and drug use was assessed by means of medication package inspection and pills counting. Also, oral hygiene instruction was reinforced. At the end of 4 weeks, all clinical measurements were retaken by the same blinded operator and medication use was reviewed.

# Statistical Analysis

The data obtained was tabulated and analyzed statistically. The data obtained for plaque and gingival inflammation were analysed using the Mann Whitney U-test and probing pocket depth measurement by the Student t-test. A p-value of less than 0.05 was considered statistically significant.

#### **RESULTS**

All 30 patients completed the study, presenting 100% of medication compliance, and no complain of adverse reactions to the ibuprofen therapy. Compliance was monitored at both recall and final exam visits by the assessment of the amount of medication taken from the original prescription package.

The results for plaque levels at the various time intervals indicated that there were no statistically significant differences between the group A and group B. The data for bleeding on probing and probing pocket depth are given in Table 1 and 2.

**Table 1** Bleeding on probing in group A and group B at various time intervals

Groups				
	Group A (SRP only)	Group B (SRP + ibuprofen)	P value	
Baseline	$.90 \pm .25$	$.84 \pm .33$	> .05	
2 weeks	$.61 \pm .178$	$.50 \pm .17$	>.05	
4 weeks	$.40 \pm .16$	$.30 \pm .12$	> .05	

At the pre-treatment assessment, the difference in the baseline mean data for bleeding on probing and probing pocket depths for the two groups was not statistically significant. Both groups exhibited a reduction after treatment in bleeding on probing and pocket depth measurements at various time intervals. Statistical analysis indicated that the differences between the mean value of the groups were not significant at the 2-weeks and 4 week intervals (p>0.05)

**Table 2** Pocket depth in group A and group B at various time intervals

Groups				
	Group A (SRP only)	Group B (SRP + ibuprofen)	P value	
Baseline	$3.7 \pm 1.21$	$3.5 \pm 1.06$	> 0.05	
2 weeks	$3.13 \pm .87$	$2.71 \pm .72$	>0.05	
4 weeks	$2.81 \pm .73$	$2.26 \pm .84$	>0.05	

# **DISCUSSION**

The present clinical study was designed to investigate the effect of oral administration of ibuprofen on the inflammatory response in patients with chronic adult periodontitis. Ibuprofen interferes with the metabolism of arachidonic acid by competitively inhibiting the enzymes of the cyclo-oxygenase pathway, thereby reducing the production of inflammation mediating factors, the main ones being the prostaglandins. Through its effect on prostaglandins, ibuprofen also affects the kinin and histamine systems of inflammation mediation (Vane 1971). In addition, ibuprofen interferes with mitochondrial

oxidative phosphorylation, an effect related to antiinflammation by reduction of the energy required to mount the inflammatory state (Tokumitsu *et al.* 1977).<sup>9</sup>

The results of the current investigation indicated that comparative assessment between the group A and group B after the 2-weeks and 4 weeks, ibuprofen showed lower values for gingival inflammation as measured by bleeding on probing and probing pocket depth but measurements were statistically insignificant. Likewise, other studies have found no differences in PD reduction between control and NSAID group after 28 days. 2,10,11. This may indicate that ibuprofen has apparent beneficial effects in terms of resolution of gingival inflammation in periodontal disease, but the magnitude of the enhancement was small and not clinically relevant. The limited effectiveness may be attributed to the duration, mode of delivery of the agent and its level of efficacy in the gingival tissue. However clinical application of the regime used in this study would not be justified by these results, further research into anti-inflammatory agents as an adjunct in the treatment of periodontal diseases could be considered, in the light of the beneficial effect on gingivitis in the early phase of periodontal treatment reported.

# **CONCLUSION**

This clinical investigation demonstrated that the systemic administration of 200 mg/ day of ibuprofen for 1 month had a relatively minor influence on the degree of inflammatory response for the treatment of patients with chronic adult periodontitis.

Conflicts of Interest: None Source of Support: Nil

# References

- 1. Noguchi K, Ishikawa I. The roles of cyclooxygenase-2 and prostaglandin E2 in periodontal disease. Periodontol 2000. 2007;43:85-101.
- Vardar S, Baylas H, Huseyinov A. Effects of selective cyclooxygenase-2 inhibition on gingival tissue levels of prostaglandin E2 and prostaglandin F2alpha and clinical parameters of chronic periodontitis. J Periodontol 2003;74:57-63.
- 3. Reynolds MA, Prudêncio A, Aichelmann-Reidy ME, Woodward K, Uhrich KE. Non-steroidal antiinflammatory drug (NSAID)-derived poly(anhydride-esters) in bone and periodontal regeneration. Curr Drug Deliv 2007;4:233-239.
- 4. de Queiroz AC, Taba M Jr, O'Connell PA, da Nóbrega PB, Costa PP, Kawata VK *et al.*. Inflammation markers in healthy and periodontitis patients: a preliminary data screening. Braz Dent J 2008;19:3-8.
- Kirkwood KL, Cirelli JA, Rogers JE, Giannobile WV. Novel host response therapeutic approaches to treat periodontal diseases. Periodontol 2000 2007;43:294-315.
- Tipton DA, Flynn JC, Stein SH, Dabbous M. Cyclooxygenase-2 inhibitors decrease interleukin-1betastimulated prostaglandin E2 and IL-6 production by human gingival fibroblasts. J Periodontol 2003;74:1754-1763.
- 7. Offenbacher S, Williams RC, Jeffcoat MK, Howell TH, Odle BM, Smith MA. *et al.* Effects of NSAIDs on beagle crevicular cyclooxygenase metabolites and

- periodontal bone loss. J Periodontal Res 1992;27:207-213.
- 8. Vane, J. R. Inhibition of prostaglanding synthesis as a mechanism of action for aspirin-like drugs. *Nature* 1971;231, 232.
- 9. Tokumitsu, Y., Lee. S. & Ui, M. (1977) Invitro effects of non-steroidal anti-inflammatory drugs on oxidative phosphorylation in rat liver mitochondria. *Biochemical Pharmacology* 1977; 26, 2101-2106.
- 10. Taiyeb Ali TB, Waite IM. The effect of systemic ibuprofen on gingival inflammation in humans. J Clin Periodontol 1993;20:723-728.
- 11. Jeffcoat MK, Reddy MS, Haigh S, Buchanan W, Doyle MJ, Meredith MP. A comparison of topical ketorolac, systemic flurbiprofen, and placebo for the inhibition of bone loss in adult periodontitis. J Periodontol 1995;66:329-338.

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