CLINICAL EFFECT OF IBUPROFEN AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL DISEASE TREATMENT

ABSTRACT: Twenty-five 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 A), or not receive the drug (group B). The obtained results show that the mechanical periodontal treatment brought to resolution the gingival inflammation with both group of patients. Although the mean values of the used indices were lower in group A than in group B, those differences were neither statistically nor clinically significant. We may conclude that systemic ibuprofen had no significant effect on plaque, gingival or bleeding index scores.

KEY WORDS: non-steroidal anti-inflammatory drugs, ibuprofen, periodontal disease

INTRODUCTION

Bacteria and bacterial products have been considered for a century to be of primary importance in the etiology of periodontal disease. Over the past two decades, however, it has become evident that their presence alone is not sufficient to clarify the pathophysiological mechanisms of periodontal tissue destruction.

More recently, several studies have emphasized the role of the host’s immunoinflammatory responses during the destruction of periodontal tissues. Evidence suggests that arachidonic acid metabolites are implicated as leading biochemical mediators in the periodontal tissue destruction. Arachidonic acid is a polyunsaturated fatty acid that is liberated from membrane phospholipids of the cells involved in inflammatory reaction. By enzyme cyclooxygenase free arachidonic acid is oxidized to prostanoids, which include prostaglandins, prostacyclin and tromboxane, metabolites with potent biological activities. Recent data support the concept that one of the distinguishing host response mechanisms which are associated with periodontal disease progression is the local formation and secretion of prostaglandin E₂ (PGE₂) that has proinflammatory properties and can stimulate bone resorption. Studies have shown positive cor-
relation of PGE₂ levels within the periodontal tissues and within gingival crevicular fluid to the clinical expression of periodontal disease. Furthermore, examination of healthy and diseased human periodontal tissues suggests that local production of arachidonic acid metabolites and PGE₂ in particular is closely associated with periodontal status and it appears to reflect the disease activity.

Inhibition of PGE₂ synthesis can be achieved by using one of the three major pharmacological approaches. The first approach is to stabilize cell membrane, suppress the cellular degranulation and reduce the level of free arachidonic acid by exploiting the biochemical properties of steroids. The second approach is to prevent the oxidation of arachidonic acid and the subsequent hydrolysis to form PGE₂ by using antioxidants. The third approach is the direct inhibition of the enzyme cyclooxygenase through the action of non-steroidal anti-inflammatory drugs.

The purpose of this study was to investigate clinical effects of ibuprofen, one of the non-steroidal anti-inflammatory drugs, applied as an adjunct to conservative periodontal disease therapy.

**MATERIAL AND METHODS**

Twenty-five patients, fourteen males and eleven females, suffering from periodontal disease entered this study. All of them received mechanical periodontal treatment — dental plaque and supra and subgingival calculus removal. After thorough scaling and root planing, patients were randomly assigned to either receive orally 200 mg of ibuprofen per day for one month (group A), or not receive the drug (group B). Oral hygiene instructions were given to both group of patients and they were motivated for oral hygiene. Plaque index (Green-Vermillion), gingivitis index (Ramfjord) and gingival bleeding index (Cowell) were used to assess the periodontal status of the patients. The criteria for scoring were as follows:

**Plaque index:**

- 0 = no plaque and calculus on tooth surfaces
- 1 = plaque and calculus on the gingival third of tooth surface
- 2 = plaque and calculus on the middle third of tooth surface
- 3 = plaque and calculus on the incisal third of tooth surface

**Gingivitis index:**

- 0 = absence of signs of inflammation
- 1 = mild to moderate inflammation, not extending around the tooth
- 2 = mild to moderately severe inflammation, extending all around the tooth
- 3 = severe gingivitis with tendency to bleeding and ulceration

**Gingival bleeding index:**

- 0 = no bleeding on probing
- 1 = bleeding within 30 sec. after probing
2 = bleeding immediately after probing
3 = spontaneous bleeding

Patients were examined, and data were collected at the baseline, and then once each of the four consecutive weeks after the mechanical treatment.

RESULTS

The obtained results showed that mechanical periodontal treatment brought to resolution the gingival inflammation with both group of patients. The mean values of periodontal indices used in this study remained significantly low throughout the examination period but with constant increase. Although the mean values of plaque, gingivitis and gingival bleeding index were lower in group A than in group B, the differences were not statistically significant. The reason for that could be a small number of examinees but, in our opinion, those differences were not clinically significant either. The reason could also be the low dose of the drug, or it could be the low anti-inflammatory effect of the systemically administrated ibuprofen on gingival tissues. Nevertheless, it can be stated that in our study, no clinical effect of systemic ibuprofen was observed.

Table 1. — Gingival bleeding index

<table>
<thead>
<tr>
<th>Week of examination</th>
<th>Group A</th>
<th>Group B</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.41</td>
<td>1.41</td>
<td>p &gt; 0.05</td>
</tr>
<tr>
<td>I</td>
<td>0.75</td>
<td>0.82</td>
<td>p &gt; 0.05</td>
</tr>
<tr>
<td>II</td>
<td>0.71</td>
<td>0.78</td>
<td>p &gt; 0.05</td>
</tr>
<tr>
<td>III</td>
<td>0.78</td>
<td>0.81</td>
<td>p &gt; 0.05</td>
</tr>
<tr>
<td>IV</td>
<td>0.82</td>
<td>0.89</td>
<td>p &gt; 0.05</td>
</tr>
</tbody>
</table>

Figure 1. — Plaque index — the bars represent mean values for each week of the examination period
DISCUSSION

A number of studies, involving both humans and animals, have been conducted to investigate the effects of non-steroidal anti-inflammatory drugs on periodontal disease progression. The results of most of these studies have shown beneficial effects of these drugs on gingival crevicular fluid levels of PGE$_2$, and on bone loss as well.

Investigating the effects of different non-steroidal anti-inflammatory drugs on experimental periodontitis in beagle dogs, Offenbacher et al. (1992) reported a significant decrease in the gingival crevicular fluid levels of PGE$_2$ in all non-steroidal anti-inflammatory drug-treated animal groups [5]. Abramson et al. (1992) analyzed the clinical and biochemical effects of systemic flurbiprofen on gingivitis in humans. They concluded that gingival crevicular fluid levels of PGE$_2$ were significantly decreased in flurbiprofen-treated patients when compared with the placebo group. One week after drug administration was discontinued, PGE$_2$ levels returned to baseline levels [1]. Haesman et al. (1993), who also investigated effects of systemic flurbiprofen on experimental gingivitis in 21 patients, reported similar results [2]. Jeffcoat et al. (1988) reported of significantly lower bone resorption in 15 patients who received 50 mg flurbiprofen for two months. Several other investigators reported significant bone gain in non-steroidal anti-inflammatory drug treated group of patients [3].

At the same time, studies to correlate these positive effects of non-steroidal anti-inflammatory drugs on gingival crevicular fluid levels of PGE$_2$ and on bone loss, with clinical effects such as plaque scores, gingival and bleeding index scores, often had contradictory results. While Haesman and coworkers, for example, stated that the reduction of gingival crevicular fluid levels of PGE$_2$ coincidences with clinically reduced gingival bleeding scores, several other investigators reported opposite results. Vogel et al. (1984) investigated the
effects of systemic sulindac on experimental gingivitis in 18 male dental students and concluded that there was no significant effect on gingival crevicular flow and bleeding index [6]. Johanson et al. (1990) investigated the effects of naproxen, another non-steroidal anti-inflammatory drug, on gingival inflammation. The results of their study showed that the drug had no significant effect on plaque, gingival and bleeding index scores. A significant effect of naproxen was only seen in resolution of gingivitis after plaque was removed [4].

The results of our study are close to these findings. It seems that, clinically, non-steroidal anti-inflammatory drugs do not lead to a potent anti-inflammatory effect. Rather, they appear to stabilize the existing periodontal condition and diminish or inhibit the rate of disease progression.

CONCLUSION

The results of this study showed no clinical benefit of systemically administered ibuprofen used as an adjunct to non-surgical periodontal therapy.

REFERENCES


Бактерије и бактеријски продукти се већ дуги низ година означавају као главни узроци пародонталне болести. Међутим, данас је јасно да и други механизми, пре свега имунски одговор домаћина, играју велику улогу у деструкцији пародонталних ткива. Истраживања су показала да арахидонска киселина и њени метаболити, а пре свих простагландин E2, имају знатан инфламаторни потенцијал и да доводе до ресорпције алеволарне кости. Резултати многих студија показују да примена нестероидних антиинфламаторних лекова доводи до смањења нивоа простагландина E2 у гингивалној течности и до смањења коштане ресорпције.

Ово истраживање предузето је са циљем да се испита утицај ибупрофена, једног од нестероидних антиинфламаторних лекова, као допуне конзервативној терапији пародонталне болести, на клинички налаз на парodonцијуму. Двадесет петоро пацијената је након конзервативне терапије подељено у две групе. Пацијенти групе A узимали су свакодневно током месец дана 200 мг ибупрофена, док пациенти групе B нису примали никакву медицину. Резултати су показали да је код пацијената обе групе, након уклањања супра- и суб-гингivalних наслага и обраде пародонталних цепова, дошло до значајног смањења инфламације гингиве. Између, пак, средњих вредности плак индекса, гингивалног индекса и индекса крвавања гингиве пацијената две испитиване групе није било статистички а, по нашем мишљењу, ни клинички значајних разлика. Стога закључујемо да ибупрофен није имао значајнији позитиван утицај на клинички налаз на парodonцијуму код ових пацијената.