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Original Article

Effects of host modulation by nonsteroidal anti-inflammatory drugs on periodontal disease: a systematic review and meta-analysis

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Purpose: Nonsteroidal anti-inflammatory drugs that prohibit biosynthesis of arachidonic acid metabolites have been considered potent host modulation agents. The aim of this review was to determine the effect of nonsteroidal anti-inflammatory drugs adjunctive with nonsurgical periodontal treatment in patients with periodontal disease. **Materials and Methods:** Three electronic databases were searched to identify relevant studies. The methodological quality and mean differences of the change in clinical attachment level and probing depth were analyzed according to Cochrane review methods. **Results:** Twelve studies were included in the methodological assessment and nine studies were suitable for inclusion in the meta-analysis. The mean difference in the clinical attachment level gain did not differ significantly between the nonsteroidal anti-inflammatory drugs and control groups at any observation time. The highest mean difference in clinical attachment level gain was 0.30 mm at 4 weeks (95% confidence interval = -0.37 to 0.97). There was a significant mean difference in the probing depth reduction, of 0.34 mm (95% confidence interval = 0.29 to 0.40) at 6 weeks. **Conclusion:** Therefore, nonsteroidal anti-inflammatory drugs have additional therapeutic effect when administrated with nonsurgical periodontal treatment. **(J Dent Rehabil Appl Sci 2017;33(1):7-18)**

Key words: anti-inflammatory agents, non-steroidal; drug therapy; meta-analysis; periodontal diseases; prostaglandins

Introduction

Periodontitis is an infectious disease initiated by periodontal pathogens.[1] It is well known that periodontal disease primarily develops due to bacterial infection. However, the initiation and progression of the diseases can vary among individuals based on genetic traits, systemic conditions, and environmental factors.[2-4] There is a large body of literature showing that both surgical and nonsurgical periodontal therapies are effective against periodontitis by removing pathogenic dental plaque and calculus.[5] However, some patients do not respond to conventional periodontal therapy[6] or show highly elevated susceptibility to periodontal infection.[7] Investigations of the mechanism underlying these phenomena have revealed that the immune response of subjects appears to play a critical role in the development and manifestation of periodontal diseases.[8,9]

In order to provide a better treatment modality to

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patients who are highly susceptible to periodontal diseases, host modulation using various therapeutic agents targeting the manipulation of the inflammatory pathway has been proposed as an adjunctive treatment with conventional periodontal therapy.[10-13] Nonsteroidal anti-inflammatory drugs (NSAIDs) are among the most potent agents due to their direct inhibition of cyclooxygenase, which is responsible for the production of arachidonic acid metabolites. These metabolites are involved in tissue breakdown in periodontal diseases[14] via the activation of the secretion of matrix metalloproteinase and osteoclasts. NSAIDs are therefore expected to have a strong efficacy for host modulation in patients with periodontal diseases.

In spite of the effectiveness of NSAIDs in host modulation, few systematic literature reviews have attempted to fully evaluate their impact in periodontal therapies, although it has been well established that the therapeutic effect of a host modulation agent can be synergistic when it is coupled to a cause-related periodontal therapy.[15-17]

Therefore, the aim of this review was to compare the effects of adjunctive use of NSAIDs with nonsurgical periodontal treatment (NSPT) on periodontal disease based on the available literature. Following focused question was addressed: "In patients with periodontal disease, is there an additional benefit of the oral administration of NSAIDs adjunctive with NSPT compared to control in terms of the clinical attachment level (CAL), probing depth (PD), and adverse outcomes in randomized controlled trials?"

Materials and Methods

Data and literature source

Relevant studies in electronic databases were identified by searching MEDLINE (from January 1, 1976 to March 11, 2015), EMBASE (from January 1, 1985 to March 11, 2015), and the Cochrane Central Register of Controlled Trials (from January 1, 1987 to March 11, 2015) without any restriction on language according to the Cochrane review methods.[18] A search strategy was developed by combining text words and MeSH terms for MEDLINE and adapted to other databases (supplementary Appendix 1). In addition, key articles on periodontology published in the Journal of Periodontology, Journal of Clinical Periodontology, or Journal of Periodontal Research as well as the gray literature from January 1, 2005 to March 11, 2015 were searched manually for any missed relevant articles.[19]

Inclusion criteria and study selection

To be eligible for inclusion in our review, the studies had to fulfill the following criteria:

1. Randomized controlled trials that compared the effects of NSAIDs and NSPT versus NSPT only.
2. Subjects diagnosed with one of the classified types of periodontal disease,[20] with the exception of gingival diseases and periodontitis associated with endodontic lesions.
3. All drugs administered via the oral route.
4. NSPT performed in both the NSAIDs and control groups.
5. Outcome included at least one clinical parameter of CAL or PD.

Data extraction and analysis

Studies to be included were selected independently by two reviewers (D.Y.K. and J.C.P.). After removing duplicates, suitable studies were identified by applying screening at the title/abstract level and then by reading the full texts. The level of agreement between reviewers in the screening process was assessed using Cohen's kappa coefficient.[21] Data of identified studies were extracted independently by two reviewers using a prefabricated data extraction form. Disagreement was discussed by two reviewers, and if not resolved it was mediated by taking advice from the other authors. The methodological quality of each included study was assessed independently using the Cochrane risk-of-bias assessment tool.[22] Suggested protocols were searched as part of the evaluation of reporting bias.[23] To describe characteristics of the studies, data about subject populations

and experimental designs were collected. The mean and standard deviation (SD) values of the changes in CAL and PD were collected for each group at baseline and the time of observation. If there were ambiguous or incomplete data, we contacted the authors by e-mail. The differences in the CAL gain and PD reduction between groups were calculated as mean and 95% confidence interval values. The results of the quantitative analysis were checked using both random-effect and fixed-effect models. To quantify heterogeneity, the I^2 statistic was used to estimate the proportion of inconsistency from true differences.[24] Review Manager software (v.5.2, Nordic Cochrane Center, Copenhagen, Denmark) was used to perform the statistical tests.

Results

Identification of studies

The search of the databases initially identified 712 articles (Fig. 1), of which 287 duplicated articles were removed. At title/abstract screening, 400 articles that clearly were not consistent with the inclusion criteria were excluded. Reading the full texts of the remaining 25 articles resulted in the identification of 12 potentially relevant studies. The reasons for excluding 13 articles are tabulated in supplementary Appendix 2. The agreement between the two reviewers throughout the screening process was rated as good, with a Cohen's kappa coefficient of 0.69.

Study characteristics and patient populations

The demographic and experimental characteristics of the included studies are summarized in Table 1. There were differences between the studies in the kind of NSAIDs, daily dose, administration period, and observation period. The examined area, type of tooth, and number of data points per subject also varied among the studies. Treatment outcomes were presented using various outcome variables. For most of the studies, CAL or PD was reported as mean ± SD values at the subject-level observation unit.[25-33]

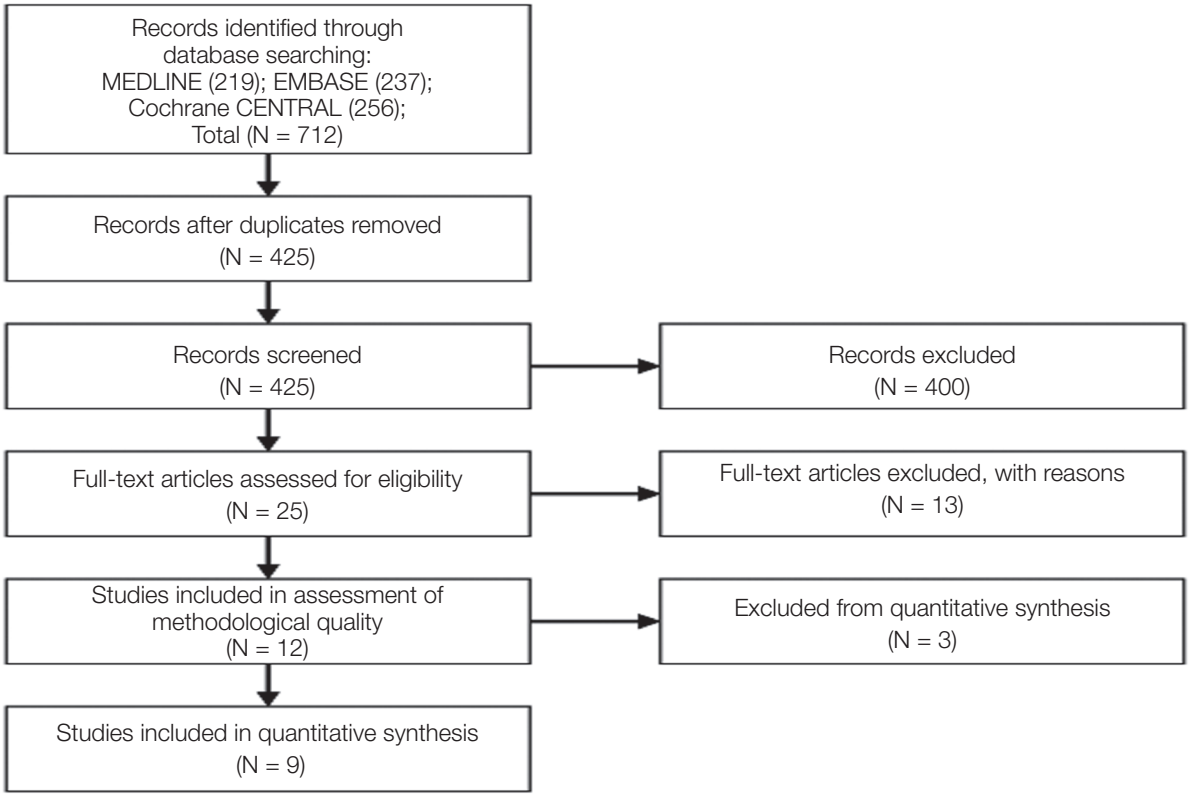

Fig. 1. Flow diagram.

Table 1. Characteristics of included studies

Study	Methods	Participants	Interventions	Clinical outcomes	Notes
Aras 2007	RCT Sample size at entry: naproxen: 17, placebo: 17 Study duration: 6 weeks Unit of observation: subject	Age (mean ± SD, yrs): 36.24 ± 2.4 Sex % F: 50%, not mentioned in each group Smoker: excluded DM: excluded	Rx) naproxen 275 mg 1T for 6 weeks Ctrl) placebo for 6 weeks Com) supragingival scaling, OHI at baseline	Subject based: mean ± SD of PD, GI, PI, gingival bleeding index at baseline and 6 weeks (full mouth) mean ± SD of PD, GI, PI, gingival bleeding index at baseline and 6 weeks (sample site)	Probing site: full mouth Sampling sites: two deepest periodontal pockets in the maxilla incisors, canines, and premolar No. of probing sites: not mentioned
Azoubel 2008	RCT Sample size at entry: etoricoxib: 11, placebo: 10 Study duration: 30 days Unit of observation: subject, site	Patient: AP, age of 18 - 35 years, had ≥ 20 teeth, had ≥ 4 sites in different teeth showing PD of ≥ 4 mm, and had ≥ 2 sites showing PD of ≥ 7 mm Age (mean ± SD, yrs): etoricoxib: 32.4 ± 6.5, placebo: 34.6 ± 7.6 Sex % F: etoricoxib: 91%, placebo: 90% Smoker: excluded DM: excluded	Rx) etoricoxib 120 mg 1T for 7 days Ctrl) placebo 1T for 7 days Com) OHI and SRP during drug intake, weekly oral hygiene reinforcement and prophylaxis until 30 days	Subject-based: mean ± SD of PD, CAL, gingival recession, visual plaque index, and BOP at baseline and 30 days Site-based: frequency (%) of sites showing CAL gain of ≥ 2 mm Dropout and reason: Rx) 1, lost to follow-up (did not return for reassessment)	Probing site: sites showing baseline PD of >3 mm only and gingival increase was excluded Sampling sites: deepest periodontal pockets in the maxilla anterior and premolar No. of probing sites: 6
Buduneli 2010	RCT Sample size at entry: meloxicam: 26, placebo: 24 Study duration: 4 weeks Experimental unit: subject	Patient: CP, Caucasian, had ≥ 4 sites showing CAL of ≥ 4 mm and PD of ≥ 5 mm and two of which were in the anterior region Age (mean ± SD, yrs): meloxicam: 48.4 ± 2.1, placebo: 47.2 ± 1.9 Sex % F: meloxicam 50%, placebo 50% Smoking: excluded DM: excluded	Rx) meloxicam 7.5 mg 1T for 10 days Ctrl) placebo for 10 days Com) SRP on day 3 of drug intake	Subject-based: mean ± SD of PD, CAL, dichotomous plaque index (present or absent), and papilla bleeding index (PBI) at 10 days and 4 weeks after SRP Dropout and reason: Rx) 3, Ctrl) 2, discontinued study	Probing, sampling sites: 2 sampling sites of the single-root teeth showing PD of ≥ 5 mm, CAL of ≥ 4 mm, and BOP (+) No. of probing sites per tooth: 2 (mesiobuccal, distobuccal)
Jeffcoat 1991	RCT Sample size at entry: naproxen: 7, placebo: 8 Study duration: 3 months Unit of observation: subject	Patient: AP, had ≥ 5 sites showing radiographic evidence of rapidly progressive periodontitis, age < 35, and had ≥ 20 teeth Age (range, yrs): 18 - 41 Sex F %: overall 73%, no detail per group Smoking: not mentioned DM: excluded	Rx) naproxen 1000 mg per day for 3 months Ctrl) placebo for 3 months Com) SRP 2 weeks before drug intake, OHI 2 weeks before drug intake and after 1, 2, 3 months of drug intake	Subject-based: mean ± SD of the change in CAL and GI between baseline and 3 months Site-based: radiographic: frequency (%) of tooth showing increased, no change, or decreased bone	No. probing sites: five test sites which had evidence of rapidly progressive periodontitis
Ng and Bissada 1998	RCT Split mouth design (SRP / no SRP) Sample size at entry: no detail (32 subjects were randomly assigned to one of the 4 treatment group) Study duration: 24 weeks Unit of observation: subject	32 subjects with generalized periodontitis that radiographic bone loss is greater than 50% in at least 2 matching tooth types per subject, PD of ≥ 5 mm in 1 or more sites in at least 2 matching types Age (range, yrs): 32 - 72 yrs Sex F %: overall 44%, no detail per group Smoking: smoker = 15, non-smoker + former smoker = 17 DM: excluded	Rx1) doxycycline 200 mg (first day), 100 mg (other day) per day for 6 weeks Rx2) ibuprofen 800 mg per day for 6 weeks Rx3) doxycycline + ibuprofen for 6 weeks Ctrl) placebo for 6 weeks Com) SRP at baseline	Subejct-based: mean ± SD of PI, GI, PD, CAL at baseline, 3, 6, 9, 12, and 24 weeks	Probing sites: full mouth No. of probing sites per tooth: 6 (using pre-fabricated stents)

Table 1. (Continued) Characteristics of included studies

Study	Methods	Participants	Interventions	Clinical outcomes	Notes
Özgören 2014	RCT Sample size at entry: tenoxicam: 16, placebo: 16 Study duration: 30 days Unit of observation: subject	Patient: CP, had ≥ 4 periodontal sites showing PD of 4 - 6 mm and radiographic evidence of bone and attachment loss involving the maxillary anterior teeth Age (mean ± SD yrs): tenoxicam: 40.9 ± 8.2, placebo: 42.3 ± 7.3 Sex F %: overall 44%, no details per group Smoking: excluded DM: excluded	Rx) tenoxicam 20 mg per day for 10 days Ctrl) placebo for 10 days Com) SRP at baseline, OHI before baseline and at baseline	Subject-based: mean ± SD of PI, GI, PD, gingival bleeding time index and CAL at baseline and 30 days	Probing sites: 4 teeth (PD of 4 - 6 mm and radiographic evidence of bone and attachment loss involving the maxillary anterior teeth) No. of probing sites per tooth: 6
Pinho Mde 2008	RCT Sample size at entry: loxoprofen 30, placebo: 30 Study duration: 28 days Unit of observation: subject	Patient: periodontitis, had ≥ 20 teeth, 35 - 0 years, had ≥ 2 teeth showing CAL of ≥ 6 mm, and had ≥ 1 site showing PD of ≥ 5 mm Age (mean ± SD): loxoprofen 41 ± 4, placebo: 42 ± 5 Sex F%: loxoprofen 53%, placebo: 53% Smoking: excluded DM: excluded	Rx) loxoprofen 60 mg per day for 28 days Ctrl) placebo for 28 days Com) SRP of half mouth at baseline and contra-lateral side on day 14 and OHI at baseline and 14 days	Subject-based: PD, % BOP, and PI at baseline (demographic data) Site-based: % sites showing PD of < 4, 4 - 7, and ≥ 7 mm at baseline, 14, and 28 days	Probing sites: full mouth No. of probing sites per tooth: 6
Reddy 1993	RCT Sample size at entry: overall 27, no details per group Study duration: 28 days Unit of observation: subject, site	Patient: had a history of periodontitis prior to the age of 35, had ≥ 3 posterior teeth in each of 3 quadrants showing alveolar bone loss of 30 - 50%, and had ≥ 3 sites showing active bone loss as determined by bone scan Age (mean ± SD, yrs): overall: 36.5 ± 7.88, no details per group Sex F %: overall 68% Smoking: not mentioned DM: excluded	Rx1) meclofenamate 100 mg per day for 6 months Rx2) meclofenamate 200 mg per day for 6 months Ctrl) placebo for 6 months Com) OHI, SRP, and occlusal adjustment (when needed) before 2 - 4 weeks from baseline SRP at 3 months and 6 months	Subject-based: GI, PI, PD and CAL at baseline, 3 months, and 6 months Site-based: CAL at baseline to 6 months Dropout and reason: Rx1) 3 (2: gastrointestinal irritation, 1: disliked medication) Rx2) 1 (disliked medication) Ctrl) 1 (military service) Adverse effect gastrointestinal irritation	Probing sites: 6 - 8 teeth (which show high bone-seeking radiopharmaceutical uptake ratio) No. of probing sites per tooth: not mentioned Statistics were expressed as mean ± standard error
Shiloah 2014	RCT Sample size at entry: aspirin: 18, placebo 14 Study duration: 12 months Unit of observation: subject	Patient: chronic periodontitis, self-reported smoker, had ≥ 4 teeth showing PD of 5 mm and attachment loss of > 2 mm Age (mean ± SD yrs): aspirin: 53.25 ± 7.31, placebo: 48.83 ± 8.34 Sex F %: 58% except dropout, not mentioned at entry Smoking: 100% (only smoker included) DM: excluded	Rx) aspirin 325 mg per day for a year Ctrl) placebo for a year Com) extraction of "hopeless", non-restorable tooth, OHI, SRP before baseline	Subject-based: % sites plaque index = 2 and 3, % sites GI = 0, 1, and 2, % sites PD 1 - 3, 4 - 6, and ≥ 7 mm, % sites PAL 0 - 2, 3 - 4, and > 4 mm, % sites BOP at baseline, 3, 6, 9, and 12 months Dropout and reason Rx) 6 (lost to follow up, e.g., job schedule change, moved) Ctrl) 2 (lost to follow up e.g., job schedule change, moved)	Probing sites: full mouth No. of probing sites per tooth: not mentioned

Table 1. (Continued) Characteristics of included studies

Study	Methods	Participants	Interventions	Clinical outcomes	Notes
Taiyep Ali and Waite 1993	RCT Sample size at entry: overall 17, no details per group Study duration: 8 weeks Unit of observation: subject	Patient: chronic periodontitis, had 6 Ramfjord teeth with mild to severe bone loss Age (range yrs): overall 28 - 40 Sex F %: overall: 71%, no details per group Smoking: not mentioned DM: excluded	Rx) ibuprofen 800 mg per day for 14 days Ctrl) no medication Com) OHI, SRP (split mouth) at baseline; OHI, oral prophylaxis, and re-evaluation at 2, 4, 6 and 8 weeks	Subject-based: PD, gingival color index, and gingival bleeding at 2, 4, 6, and 8 weeks Dropout and reason: Rx) 2, unknown	Probing sites: 6 teeth (Ramfjord teeth) No. of probing sites per tooth: 6 No placebo medication
Vardar 2003	RCT Sample size at entry: nimesulide 10, naproxen 10, and placebo 10 Study duration: 3 months Unit of observation: subject	Patient: CP, had ≥ 18 teeth, and had ≥ 2 teeth in each quadrant showing PD of ≥ 5 mm and CAL of ≥ 4 mm Age (mean ± SD, yrs): nimesulide: 46.6 ± 14.1, naproxen: 44.0 ± 3.6, placebo: 43.9 ± 6.9 Sex F %: nimesulide: 40%, naproxen: 30%, placebo: 50% Smoker: not excluded DM: excluded	Rx1) nimesulide 200 mg per day for 10 days Rx2) naproxen 550 mg per day for 10 days Ctrl) placebo for 10 days Com) OHI at baseline, SRP at 3 days	Subejct-based: CAL and PD at baseline and 3 months PI, PBI at baseline, 10 days, and 3 months	Probing sites: 4 sites (2 sampling sites x 2 approximal tooth sites adjacent to the sampling site)
Yen 2008	RCT Sample size at entry: overall 131 Study duration: 12 months Unit of observation: site	Patient: CP, had > 16 teeth (at least two of which were molars), had ≥ 4 teeth showing PD of > 4 mm and CAL of > 2 mm, and had ≥ 2 interproximal areas showing radiographic evidence of bone loss Age (mean ± SD, yrs): overall 48.6 ± 9.9 Sex F%: 47% at 3 months Smoker: 34.7% (former smoker: 23.8%, nonsmoker: 39.6%) DM: not excluded	Rx) celecoxib 200 mg per day for 3 months Ctrl) placebo for 3 months Com) SRP at baseline (1/2) and within 1 - 2 weeks (1/2) OHI at baseline, 3, 6, 9, and 12 months oral prophylaxis at 3, 6, 9, and 12 months	Site-based CAL by BPD = 1 - 3, 4 - 6, and ≥ 7 mm PD by BPD = 1 - 3, 4 - 6, and ≥ 7 mm % sites showing CAL gain ≥ 2 mm % sites showing CAL loss ≥ 2 mm Dropout and reason Total 66 (highly publicized finding that subjects taking large dosages of COX-2 inhibitors showed an increased risk for cardiovascular episodes)	Probing sites: full mouth No. of probing sites per tooth: 6 66 subjects were dropout

AP: aggressive periodontitis, BOP: bleeding on probing, BPD: baseline probing depth, CAL: Clinical attachment loss, Com): common intervention, CP: chronic periodontitis, Ctrl): control group, GI: gingival index, GCF: gingival crevicular fluid, DM: diabetes mellitus, OHI: oral hygiene instruction, PD: probing depth, PI: plaque index, Rx): nonsteroidal anti-inflammatory drugs group, SD: standard deviation, yrs: years, Sex % F: percentage of female, SRP: scaling and root planing.

Methodological quality of included studies

The included studies were assessed using the Cochrane risk-of-bias assessment tool (Fig. 2, supplementary Appendix 3). Publication bias could not be assessed due to the small number of appropriate studies (7 for CAL and 8 for PD), since asymmetry of the funnel plot is generally only tested when at least 10 studies are included in a meta-analysis.

Effects of interventions

Data reported as mean ± SD values with the subject-level observation unit pooled by observation time. The differences in the CAL gain and PD reduction between NSAIDs and control groups using a random-effect model are illustrated in Fig. 3 and Fig. 4 as mean and 95% confidence interval values. There was no significant difference in the mean CAL gain

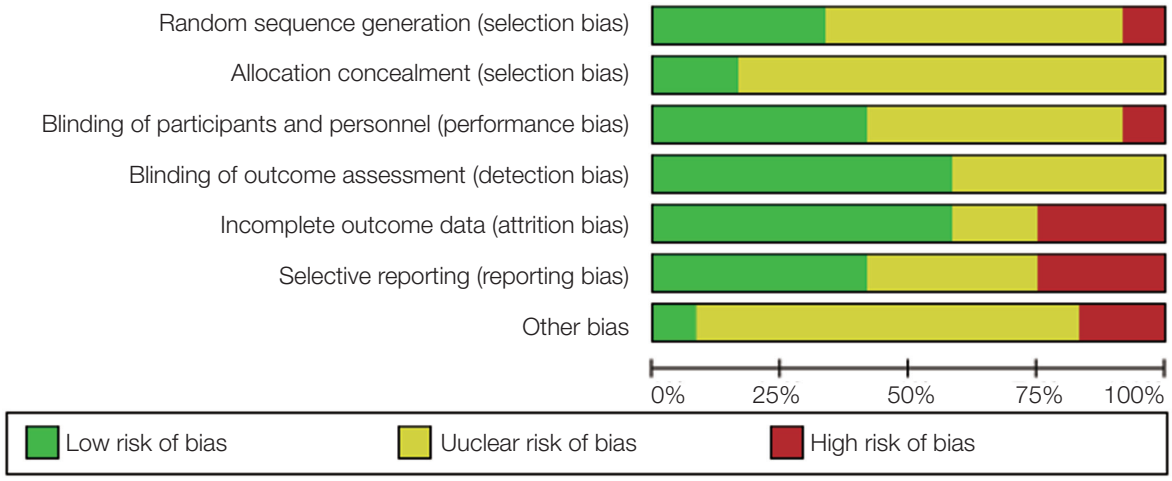

Fig. 2. Risk of bias graph: review authors' judgements about each risk of bias item presented as percentages across all included studies.

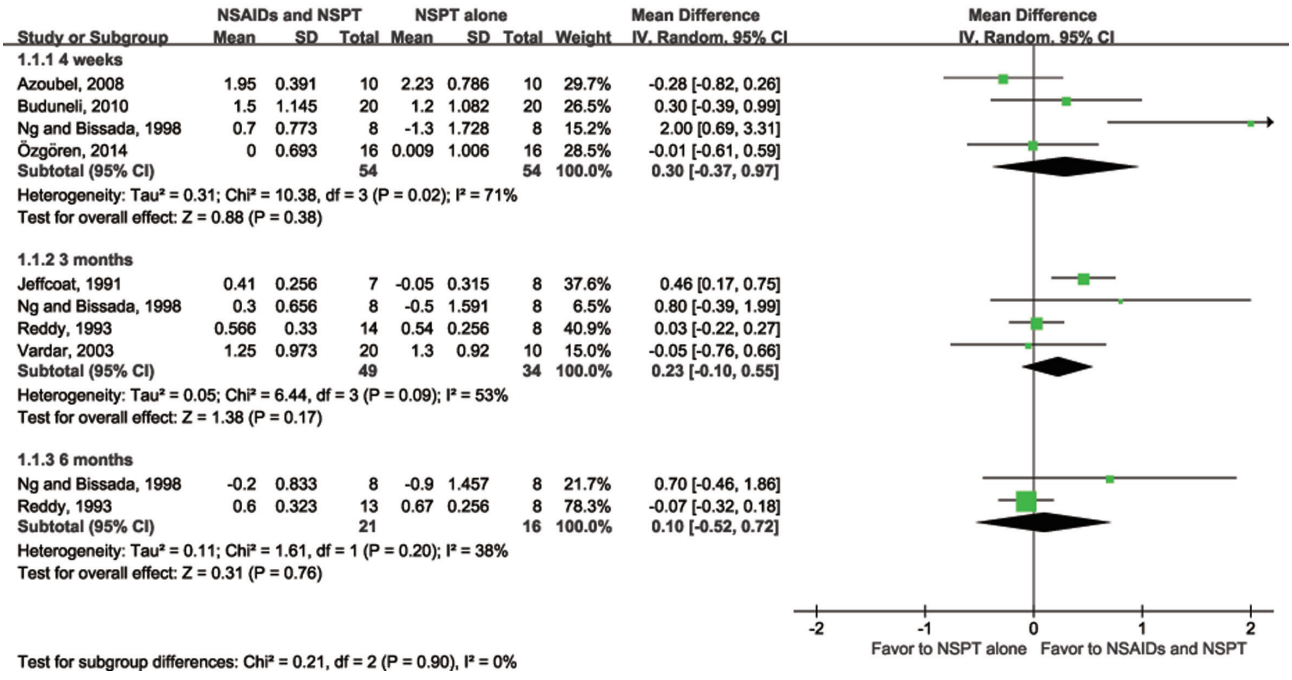

Fig. 3. Forest plot of changes in CAL for in randomized clinical trials that administrated NSAIDs adjunctive to nonsurgical periodontal treatment.

at any observation time (Fig. 3). A significant reduction in PD-with a mean difference of 0.34 mm (95% confidence interval = 0.29 to 0.40)-was seen only at 6 weeks relative to the control group (Fig. 4).

The number of adverse reactions, the reasons for dropouts, and reports of adverse reactions are listed in Table 1. Reddy et al.[31] reported that their dropouts were due to gastrointestinal discomfort in the group receiving meclofenamate at 200 mg/day, but the reason for other dropouts was unknown in most cases (e.g., lost to follow-up or a discontinued study).

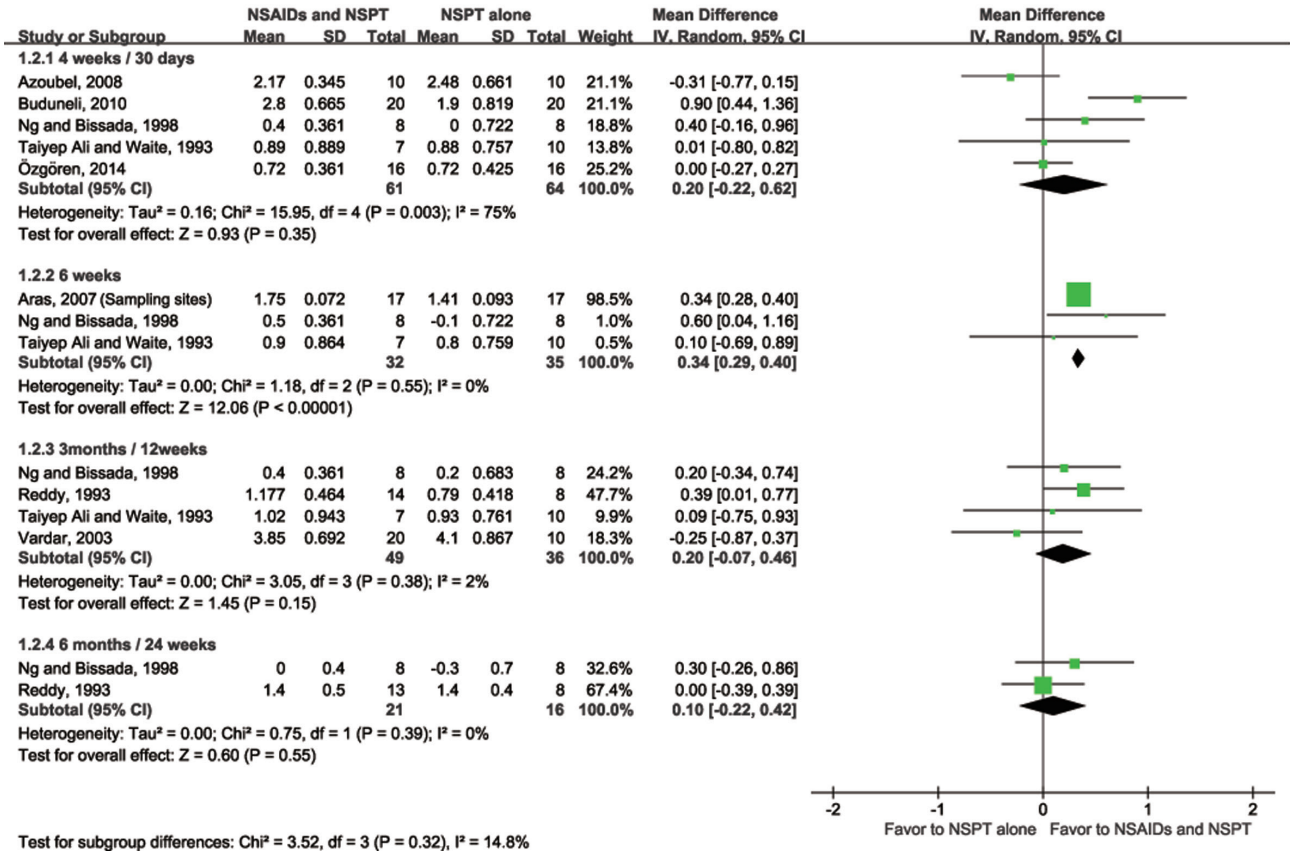

Fig. 4. Forest plot of changes in PD for in randomized clinical trials that administrated NSAIDs adjunctive to nonsurgical periodontal treatment.

Discussion

The studies included in this meta-analysis exhibited a substantial heterogeneity, which was due to a few reports of significantly higher mean differences in CAL gain or PD reduction in the NSAIDs group than the control group. For instance, Ng and Bissada[29] reported a higher mean difference in the NSAIDs group at 6 weeks posttreatment, but this was due to a smaller CAL loss in the NSAIDs group compared to the control group, rather than to a superior CAL gain. Likewise, Buduneli et al.[27] enrolled patients with a deeper PD at baseline in the NSAIDs group than the control group, and this may have resulted in a superior PD reduction considering that scaling and root planing reportedly produces a greater PD reduction in sites with a deeper PD.[5,34]

The following differences accounted for the heterogeneity. Firstly, subjects were recruited in each study using different inclusion criteria and exclusion criteria. Secondly, additional oral prophylaxis or oral hygiene instruction was either not planned or, if it was planned, the schedule varied among the studies. These additional procedures may produce an appropriately maintained plaque state in the enrolled patients, and they may have resulted in the treatment responses differing among the studies. In addition, the state of plaque deposition was not presented as a unified outcome variable. Moreover, kinds of NSAIDs the daily dose, administration period, and observation period of the regime varied among the studies. These differences may have contributed to

the inconsistent results.

It is well known that NSAIDs may induce gastrointestinal irritation, and two of the patients who dropped out for this reason had received meclofenamate at 200 mg/day for 6 months.[31] To minimize gastrointestinal irritation and ulcers from NSAIDs, limiting the dose or co-administration with proton-pump inhibitors or H_2-receptor antagonist can be considered.[35] It has been reported that NSAIDs can impair oral mucosal repair as well as gastrointestinal mucosa,[36] so whether NSAIDs compromise gingival repair following NSPT also needs to be investigated.

A recent nationwide retrospective study failed to show any benefit of low-dose aspirin when comparing mean CAL and PD data from 2 335 subjects in a national health and nutrition examination survey.[37] In addition, systematic reviews have found no clear effect of NSAIDs on clinical outcomes.[17] Our present results are in accordance with these previous findings. However, two reviews have found positive effects of NSAIDs on gingival inflammation and periodontal-disease progression by reducing the rate of alveolar bone resorption.[38,39] These conflicting findings are probably due to the bone preservation effect of NSAIDs and it might not be reflected in clinical parameters including PD or CAL. Also, it would take a long time for bony changes to be measurable by periodontal probes.

The methodological quality of the included studies was not high due to the lack of information. In addition, there were problems associated with partial tooth recording (i.e., including only specific types of teeth), which has the risk of overestimating or underestimating the periodontal condition of patients.[40] Not only an adequate number of subjects but also an adequate number of data points from different types of teeth are needed to accurately reflect the status of included subjects. Taking these factors into account, further randomized controlled trials are needed to achieve a high methodological quality, involving subject-level observation units and sufficient data points via full-mouth observations as well as the inclusion of sufficient subjects to achieve an adequate statistical power for detecting a clinically meaningful effect size of NSAIDs.

Conclusion

The present review found clinical benefit for NSAIDs and NSPT over NSPT alone in some administration periods. It is difficult to conclude definitively whether a specific regime is superior to others, although some studies have found significant benefits and the others do not, due to the smallness of the included samples and the substantial heterogeneity among studies.

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비스테로이드성 항염증제를 이용한 숙주조절이 치주질환에 미치는 영향: 체계적 고찰 및 메타 분석

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목적: 아라키돈산 대사물의 생합성을 억제하는 비스테로이드성 항염증제는 잠재적인 숙주조절제로 고려되고 있다. 이 종설의 목적은 비스테로이드성 항염증제를 비외과적 치주치료와 병용하였을 때 치주질환자에 미치는 영향을 평가하는데 있다.

연구 재료 및 방법: 관련된 연구를 확인하기 위하여 세 전자 데이터베이스를 검색하였다. Cochrane의 고찰 방법론에 따라 방법론적인 질, 임상 부착 수준과 탐침 깊이 변화량에 대한 평균 차이를 분석하였다.

결과: 총 12개의 연구의 방법론을 평가하였고 이중 9개의 연구에 대해 메타-분석을 시행하였다. 임상 부착 수준의 변화에 대한 평균차의 경우 모든 관찰기간에서 비스테로이드성 항염증제 군과 대조군간 유의차를 보이지 않았다. 가장 큰 평균차는 4주 때 0.30 mm로 나타났다(95% 신뢰구간 = -0.37 to 0.97). 탐침 깊이 변화에 대한 평균차는 6주 때 0.34 mm (95% 신뢰구간 = 0.29 to 0.40)로 유의차를 보였다.

결론: 이를 토대로 하였을 때 비스테로이드성 항염증제를 비외과적 치주치료와 병용하여 투여하였을 때 부가적으로 치료효과를 증대시킬 수 있을 것으로 생각된다.

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주요어: 비스테로이드성 항염증제; 프로스타글란딘; 약물 요법; 치주 질환; 메타-분석

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