

The Effect of Piascledine in the Treatment of Chronic Periodontitis

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Abstract:

Objective: The aim of this study was to evaluate the clinical efficacy of an herbal medicine, Piascledine, on the treatment of chronic periodontitis.

Materials and Methods: Twenty-four patients with chronic periodontitis participated in this interventional case control investigation. After initial screening the subjects were divided into case and control groups. Clinical measurements including plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD), bone level (BL) and clinical attachment level (CAL) were recorded for both groups, before and one month after scaling and root planing (SRP). The case group received 300 mg Piascledine capsules once a day for three months and the control group was only treated with SRP. The measurements were repeated in both groups at the 3 and 6 month recall visits.

Results: Improvements were observed in all clinical parameters in both groups during the study period. PI as a covariate factor showed no significant difference between the two groups ($P>0.05$). On the other hand CAL, BOP, PPD and BL showed reduction in all patients, but no significant difference was found between the two groups.

Conclusion: According to the results obtained in the present study, Piascledine does not seem to have a favorable effect in the treatment of chronic periodontitis.

Key Words: Avocado/Soybean unsaponifiables; Periodontal therapy; Piascledine

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INTRODUCTION

A mixture of one third Avocado and two thirds soybean oil is known as Avocado/Soybean unsaponifiables (ASU, Piascledine) and has been used to treat various connective tissue disorders [1].

Interleukin-1 (IL-1) can stimulate synovio- cytes and chondrocytes to produce colla- genase. Previous studies have suggested that ASU may reduce or even reverse the destruc- tive effects of this protein in osteoarticular dis- eases [2,3]. Piascledine has also been shown to suppresses matrix metalloproteinase-3 (MMP- 3) secretion and stimulate the production of

tissue inhibitors of matrix metalloproteinase-1 (TIMP-1). Therefore it seems that ASU might possess structure-modifying properties in os- teoarthritis by inhibiting degredation and pro- moting repair in cartilaginous tissues [4].

Extracellular matrix integrity of the gingiva and other soft connective tissues is mainly provided by fibroblasts. These cells are major components of wound healing and mediate several pathological events and conditions. Former investigations have demonstrated that various MMPs and their inhibitors (TIMPs) are synthesized by human fibroblasts, *in vitro*. Stromelysin-1 (MMP-3) and gelatinase A

Table 1. The mean and standard deviation (SD) of PPD and BL in case and control groups at different stages.

PPD and BL	N (case , control)	Minimum (mm) (case , control)	Maximum (mm) (case , control)	Mean (mm) (case , control)	SD (mm) (case , control)
PPD1	11 , 12	5.32 , 4.80	6.64 , 7.57	6.09 , 6.52	0.406 , 0.827
PPD2	11 , 12	4.84 , 4.05	6.23 , 7.40	5.54 , 6.04	0.466 , 1.041
PPD3	11 , 12	4.52 , 3.40	5.79 , 7.02	5.11 , 5.50	0.461 , 1.123
PPD4	11 , 12	4.02 , 3.23	5.20 , 6.91	4.69 , 5.21	0.437 , 1.070
BL1	11 , 12	9.35 , 9.38	9.94 , 9.96	9.58 , 9.72	0.166 , 0.199
BL2	11 , 12	9.31 , 9.23	9.69 , 10.20	9.59 , 9.73	0.108 , 0.295

PPD= probing pocket depths, BL= bone level

(MMP-2) have a principal role in connective tissue remodeling and are known to degrade proteoglycans and cleave elastin, gelatin and collagen fibers, mainly type III [1].

Metalloproteinases, particularly MMP-2, MMP-1 and MMP-13 have been observed in periodontal diseases [5]. Interleukin-1 β is produced by mononuclear cells during the course of the disease and stimulates periodontal ligament fibroblasts to secrete stromelysin and collagenase. Disharmony between the expression of MMPs and TIMPs, in periodontitis can lead to gingival tissue destruction [1].

Preelastic and especially mature elastic fibers are hydrolyzed by Human Leukocyte elastase (HLE) in gingival and periodontal inflammatory states. Considering that ASU has a protective effect against HLE, its application may be useful in treating gingivitis and periodontitis [6,7].

In addition, in vitro studies have demonstrated that ASU can partially block the response of cultured gingival fibroblasts to IL-1 β [1]. Therefore it would seem logical to investigate the use of Piascledine in the reduction of inflammation in periodontal tissues.

The aim of the present study was to evaluate the effect of ASU on the treatment of chronic periodontitis.

MATERIALS AND METHODS

Twenty-four patients with chronic periodontitis (9 males and 14 females) were selected for this interventional, randomized and case-control study. They were recruited from those

referred to the Department of Periodontics, Faculty of Dentistry, Tehran University of Medical Sciences. All procedures were explained to the participants and verbal consent was obtained from each subject prior to completing their medical and dental histories. A diagnosis of chronic periodontitis was made according to clinical and radiographic findings.

Inclusion criteria consisted of clinical attachment levels (CALs) and probing pocket depths (PPDs) of more than 3mm, and observation of bleeding on probing (BOP) in at least 2 sites in each quadrant. Exclusion criteria included a history of systemic disease, treatment with antibiotics or corticosteroids in the past 6 weeks, need for antibiotic prophylaxis before routine treatment in dentistry, pregnancy or lactation and heavy alcohol or tobacco use (>10 cigarettes per day).

The present study was carried out in three stages: screening, treatment and evaluation.

The screening stage involved clinical measurement of plaque index (PI, O' Leary index, 1978), PPD, bone level (BL, using periapical radiography) and BOP (bleeding point index, Lenox 1973). This was followed by alginate impressions for making stents in order to measure CALs in future visits. After being selected for the study, the subjects were randomly divided into case and control groups.

At the treatment stage, non-surgical periodontal treatment (scaling and root planing, SRP) was carried out in all subjects by the same examiner. Tooth and root surfaces were instru-

Table 2. The mean and standard deviation (SD) of BOP in case and control groups at 4 stages of measurements.

BOP	N	Minimum	Maximum	Mean	SD
	(case , control)	(case , control)	(case , control)	(case , control)	(case , control)
BOP 1	11 , 12	9.0 , 8.0	10.0 , 10.0	9.7 , 9.7	0.34 , 0.58
BOP 2	11 , 12	5.9 , 6.0	10.0 , 10.0	8.1 , 8.3	1.32 , 1.30
BOP 3	11 , 12	4.5 , 4.5	9.0 , 10.0	6.8 , 6.5	1.70 , 1.96
BOP 4	11 , 12	3.0 , 3.5	8.0 , 10.0	5.1 , 5.7	1.43 , 2.07

BOP= bleeding on probing

mented until they were free of deposits as determined by visual or tactile examination. Oral hygiene instructions were given to the patients at each session, which consisted of tooth brushing and the use of interdental flossing or interdental brushing, as appropriate. All clinical measurements were repeated one month after completion of SRP.

The case group received SRP and 300-mg capsules of Piascledine (Laboratories Expan Science, France), once a day for 3 months, while the control group received only SRP. Patients in the Piascledine group were instructed to take their medication with food. Compliance was assessed by tablet count.

The evaluation stage included two recall visits, one at the last day of Piascledine therapy (month 3) and the other was 3 months after completion of medication. At the first recall, clinical parameters including PI, BOP, PPD and CAL were recorded and oral hygiene instructions were reinforced. These measurements along with BL were repeated at the second recall visit. Regular maintenance therapy (removal of any supragingival calculus) was performed at every visit. The mean values of the clinical parameters obtained at baseline and 1, 3 and 6 months after SRP were calculated for all subjects; and the case and control groups were compared.

The multivariate generalized linear model test and the generalized estimated equation test were used for statistical analysis.

RESULTS

One patient from case group was lost to follow up due to inability to attend regular maintenance

appointments. All examinations were completed for the remaining 23 patients, including 14 women and 9 men. The control and case groups consisted of 12 (mean age, 42.83±5.47 years) and 11 (mean age, 43.18±6.36 years) subjects, respectively.

Treatment with Piascledine was well tolerated and no adverse effects were observed in any of the patients during the study period.

The mean values of the clinical parameters measured in case and control subjects are shown in Table 1 and Table 2. At baseline, there were no statistically significant differences in the clinical periodontal parameters between the study groups ($P>0.05$).

The periodontal conditions of both Piascledine and control groups markedly improved between baseline and reexaminations. In both study groups the improvement in CAL and BL scores was not significant for any of the time points compared to base line. Despite the fact that CALs, BOPs and PPDs of the Piascledine group showed greater improvement than the control group at all time points, these differences were not significant ($P>0.05$). The improvements in CAL, BOP and PPD scores were similar for both study groups at all time points. BL worsened during the investigation period and showed a 0.01 mm decrease in both groups. There was a significant reduction in the PI scores in both case and control subjects; however no significant difference was found between the two groups over the entire study period ($P>0.05$).

DISCUSSION

This study was designed to evaluate the effi-

cacy of Piascledine therapy as an adjunct to scaling and root planing in patients with chronic periodontitis. In the current investigation, disease severity was similar in both Piascledine and control groups at baseline. Following administration of the treatment regimens (SRP with and without Piascledine), significant improvements in clinical parameters were observed. SRP therapy leads to resolution of the inflammatory response and cessation of disease progression. This in turn results in a relative gain of clinical attachment and reduction of probing depth. Scaling and root planing also alter the environment of periodontal pathogens leading to reduced colonization. The clinical parameters evaluated in the present study, demonstrated improvement in both case and control groups.

Our findings showed that the use of Piascledine in combination with SRP did not provide clinical improvement beyond that obtained by SRP therapy alone.

CONCLUSION

According to the results obtained in the present study Avocado and Soybean unsaponifiables do not seem to have a favorable effect in the treatment of chronic periodontitis. However previous investigations reported significant clinical improvement in patients with osteoarthritis and periodontitis following the use of ASU. Therefore further evaluation of the effects of this herbal drug using immunologic markers in a larger number of patients is suggested before definite conclusions are drawn.

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