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ORIGINAL ARTICLE

Healing following ultrasonic debridement and PVP-iodine in individuals with severe chronic periodontal disease: A randomized, controlled clinical study

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Abstract

Objective. Antiseptics and antibiotics delivered either locally or systemically have been used as an adjunct to scaling and root planing procedures in order to control the subgingival biofilm and thereby enhancing the treatment outcome. The results presented in the literature are, however, inconclusive. Povidone-iodine (PVP-iodine) has a bactericidal effect and is effective against most bacteria, including putative periodontal pathogens. The aim of the present study was to evaluate the clinical effect of PVP-iodine as an adjunct to ultrasonic scaling in the treatment of severe chronic periodontitis. ***Material and Methods.*** Twenty patients were recruited to the study. Each test site and the related quadrant were randomly assigned to one of four different treatment modalities: ultrasonic scaling+subgingival irrigation with 0.5% PVP-iodine for 5 min/tooth, ultrasonic scaling+subgingival irrigation with sterile saline solution for 5 min/tooth, subgingival irrigation with sterile saline solution for 5 min/tooth, and subgingival irrigation with 0.5% PVP-iodine for 5 min/tooth. The individuals were followed longitudinally for 6 months. ***Results.*** The present study showed that non-surgical periodontal therapy by means of an ultrasonic device was effective in attaining a healthy periodontal status in patients with severe periodontal lesions. No additive effect was found when PVP-iodine was included. ***Conclusions.*** Ultrasonic debridement using Odontogain® is effective in controlling infection in patients with severe chronic periodontitis. PVP-iodine does not add any clinical benefit to the ultrasonic debridement alone under these circumstances.

Key Words: *Iodine, non-surgical treatment, periodontal healing, ultrasonic debridement*

Introduction

The primary etiologic factor in periodontitis is bacterial plaque, which results in inflammation in the adjacent tissue leading to progressive destruction of the supporting periodontal tissues [1]. The periodic mechanical removal of subgingival microbial biofilms is essential for controlling inflammatory periodontal disease since bacteria can repopulate pockets within weeks following active therapy [2]. The effectiveness of subgingival debridement procedures has shown that 5–80% of treated roots harbor residual plaque or calculus, and that the deeper the pockets and furcation involvement, the more deposits that are left behind [3]. Furthermore, several studies have shown the presence of residual pockets and disease recurrence after periodontal therapy [4]. Residual sites seem to be associated with bacterial re-colonization and further periodontal disease progression [5].

Since mechanical root debridement is a technically demanding procedure, antiseptics and antibiotics delivered locally or systemically have been used as adjuncts to scaling and root planing procedures in order to control the subgingival biofilm, thereby enhancing the treatment outcome. The results presented are, however, inconclusive [6].

Povidone-iodine (PVP-iodine) is the iodophor of elemental iodine [7]. It has a bactericidal effect and is effective against most bacteria including putative

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periodontal pathogens [8,9]. Furthermore, PVD-iodine does not initiate sensitivity reactions [10]. The emergence of PVP-iodine-resistant microorganisms has not been detected to date [11]. PVP-iodine has been used earlier in the treatment of periodontal disease together with ultrasonics to enhance treatment outcome [12–14]. The effects in these studies were small, however. In a larger material, Rosling et al. [4] showed that topical application of PVP-iodine in conjunction with mechanical root debridement established conditions that improved the clinical outcome.

The aim of the study was to evaluate the clinical effect of PVP-iodine as an adjunct to ultrasonic scaling in the treatment of chronic periodontitis.

The null hypothesis tested was that topical use of PVP-iodine in conjunction with mechanical root debridement does not improve the clinical outcome in patients with severe periodontal disease.

Material and methods

Subjects

Twenty patients (8 M, 12 F), aged 39–68 years at baseline (mean age 54 years, SD 7.7) referred for treatment of advanced periodontal disease and fulfilling the inclusion criteria were recruited to the study.

Individuals with at least one single-rooted tooth/quadrant with at least one site showing probing pocket depth (PPD) $\geq$6 mm and bleeding on probing (BoP) after a 1-month period of plaque control resulting in the presence of $\leq$15% plaque (P) were included. Exclusion criteria included allergy to iodine, thyroid dysfunction, requirement of antibiotic prophylaxis prior to dental treatment or systemic antibiotic treatment within the preceding 3 months, pregnancy, diabetes, or any medical condition comprising a contraindication for routine dental treatment. Smokers were not included in the study. The protocol was approved by the Ethics Board at Sahlgrenska Academy, Göteborg University, and written informed consent was obtained from each subject.

Periodontal treatment

Following the pre-examination (visit before baseline) each subject was given information about their periodontal condition and recommended treatment. This was followed by thorough supragingival scaling and each subject was instructed in proper plaque control using a toothbrush and interdental brushes [15]. When a plaque index $\leq$15% was obtained, the treatment started, i.e. at baseline. In each subject, four test sites, each of them located in single-rooted teeth and in different quadrants, were selected based on having a PPD of 6 mm or greater. By drawing from a sealed envelope, each test site and the related quadrant were randomly assigned to one of four different treatment modalities: 1) ultrasonic scaling (Odontogain®; XO-CARE A/S, Denmark), 42,000 Hz+subgingival irrigation with 0.5% PVP-iodine for 5 min/tooth; 2) ultrasonic scaling (Odontogain®, XO-CARE A/S), 42,000 Hz+subgingival irrigation with saline for 5 min/tooth; 3) subgingival irrigation with saline for 5 min/tooth; 4) subgingival irrigation with 0.5% PVP-iodine for 5 min/tooth.

The treatment procedures were carried out by an experienced dental hygienist (LK).

The treatment was performed in two quadrants [1,4] at the same time and the other week at in the other two quadrants [2,3].

Quadrants receiving mechanical debridement were treated under local anesthesia. Xylocaine adrenaline (2%) was used in the anesthetic procedure (Astra AB, Mölndal, Sweden).

Clinical examination

Plaque score (P), PPD, and BoP were studied at the pre-examination. P was calculated as the percentage of surfaces positive for plaque (presence/absence). PPD measurements were recorded parallel to the long axis of the tooth using a 1-mm scaled periodontal probe (North Carolina PCP-15; Hu-Friedy, Chicago, Ill., USA) at six location points around the circumference of each tooth as the distance between the gingival margin and the bottom of the probable pocket, to the nearest whole millimeter. In conjunction with performing the probing depth measurement, the area was observed for the presence/absence of BoP.

In each subject one single-rooted tooth/quadrant was selected which had at least one approximal pocket with a probing depth of $\geq$6 mm. Among the selected sites, all had a suprabony location. The selected sites were exposed to further assessments at baseline, 3 and 6 months. The following parameters were recorded: plaque score, PPD, BoP, and probing attachment level (PAL), using a standardized periodontal probe and the cemento-enamel junction as a reference point. All measurements were made by one blinded investigator (ÅL).

Statistics

One tooth per/quadrant from each individual was used as registration site. Descriptive statistics were used for presentation of registered data. The Kruskal-Wallis test and the Wilcoxon rank-sum test were used to evaluate whether there was a difference between the treatment groups. To analyze differences between different follow-up times, Friedman's test and the Wilcoxon signed-rank test were performed.

The primary end-point was PPD in the tested groups. With 20 patients, the statistical power is 0.80

to detect a 1-mm difference between the treatment groups of 0.8 standard deviations or more at a significance level of 0.05 [16].

Results

Overall findings

The clinical findings for each treatment group are described in Table I. The individual mean percentage of surfaces with supragingival plaque was 77%at pre-examination, and the corresponding BoP score was 86%. The effects of the supragingival plaque control (pre-examination – baseline) at the selected sites are presented in Table I. The plaque scores at the re-examinations were $<10\%$. The mean probing depth in the selected sites at pre-examination was 6.9 mm and became 6.7 mm during the plaque control period (pre-examination 1 – baseline) (Table I).

Ultrasonic groups (1 and 2)

At pre-examination, the plaque score in both groups 1 and 2 was 78.6%. During the course of therapy the plaque score was markedly improved ($p < 0.05$). Less than 12% at the 3-month registration and less than 10% at the 6-month examination of the selected sites harbored visible amounts of plaque (Table I).

The bleeding score was more than 80% at pre-examination, and this value remained nearly stable at the baseline registration. At 3 months, the percentage of BoP-positive sites had been reduced to 37% in group 1 and 35% in group 2, and at 6 months the figures were 31% and 32%, respectively, in groups 1 and 2 (Table I).

There was a statistically significant mean probing depth reduction ($p < 0.05$) for the sites treated with the ultrasonic device between the pre-examination registration (mean PD 6.8 mm) and 3- and 6-months follow-up (mean PD 4.8 and 4.5 mm, respectively). A significant difference was found between baseline and the 3- and 6-month follow-up ($p < 0.05$). No significant difference was seen between ultrasonic scaling+iodine (group 1) and ultrasonic scaling+saline (group 2) (Figure 1).

At pre-examination, 100% of the selected sites in group 1 had a probing depth of ≥ 6 mm. At 3 months, 31.6% of the pockets were ≥ 6 mm and were reduced further to 15.8% at 6 months. In group 2, 100% of the selected sites were ≥ 6 mm at pre-examination, 21.1% at 3 months, and 15.8% at 6 months. The mean change in PAL at 6 months was 0.7 mm in group 1 and 0.6 in group 2 (Table I, Figure 1).

Irrigation groups (3 and 4)

At the pre-examination registration the plaque score in group 3 was 74.7% and 76.1% in group 4. During treatment the plaque score improved ($p < 0.001$) and became less than 15% at 3 months, and less than 10% of the selected sites harbored visible amounts of plaque at 6 months (Table I).

At pre-examination the bleeding score was more than 85%. This value was slightly reduced at baseline. At 3 months the percentage of BoP-positive sites had been reduced to 69.7% in group 3 and 76.3% in group 4, and at 6 months group 3 had 62.5% and group 4 had 79.2% of positive sites (Table I).

A significant difference can be seen between baseline and 6 months ($p < 0.05$) in PPD reduction for treatment 3. There were no significant alterations in mean probing depth between all the other time-points. The probing depth reduction between pre-examination and 3 and 6 months, respectively, was less conspicuous compared with that for groups 1 and 2. At pre-examination, 100% of the sites had a PPD ≥ 6 mm. After 3 months, group 3 had 73.7% and 89.5% in treatment 4. This difference between the groups was not statistically significant ($p > 0.05$). The value at the 6-month follow-up was 57.9% for group 3 and 78.9% for group 4. A statistical significant difference ($p < 0.05$) regarding PPD reduction was found between baseline and 6-month follow-up. The gain in probing attachment level was of minor entity in treatments 3 and 4 (Table I, Figure 1)

Comparison of the different treatment groups (1, 2, 3, and 4)

For treatment groups 1 and 2, and 3 and 4 no significant differences were seen ($p > 0.05$) regarding percentage of plaque or BoP (Table I). Regarding changes in PPD, between groups 1 and 3, 1 and 4, and 2 and 4 significant differences were seen at the 3- and 6-month follow-up examinations ($p < 0.05$) (Table I). No significant statistical differences were found between the four groups regarding changes in PAL (Figure 1).

Discussion

The aim of this study was to determine the context for which subgingival irrigation with PVP-iodine might be clinically beneficial in the treatment of chronic periodontitis. PVP-iodine did not add any clinical advantage to the ultrasonic debridement alone, therefore the null hypothesis is accepted.

The result of the present study showed that non-surgical periodontal therapy using an ultrasonic device was effective in attaining a healthy periodontal status in patients with severe periodontal lesions. These results confirm that non-surgical treatment, by means of an ultrasonic device, is effective in reducing PPD and in resolving the infection. This is concordant with the findings in

Table I. Clinical data from the 6-months' follow-up study, describing plaque percentage, bleeding on probing (BoP%), probing pocket depth (PPD) and percentage of different PPD categories. In addition, changes in probing attachment level (PAL) between baseline and the follow-up periods are reported

	Pre-examination				Baseline				3 Months				6 Months			
Treatment	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Plaque (%)	78.6±13.8	78.6±15.3	74.7±16.1	76.1±15.8	6.6±14.0	3.9±9.4	2.6±7.9	1.3±5.7	11.8±25.5	2.6±7.9	11.8±28.1	5.3±13.4	9.2±23.9	2.6±7.9	7.9±18.7	9.2±20.8
BoP (%)	88.2±12.8	80.3±27.1	86.8±15.3	86.8 ± 15.3	80.3±15.3	78.9±15.1	78.9±17.2	81.6±20.1	36.8±24.1	34.2±26.5	69.7±24.4	76.3±21.2	30.6±26.5	31.9±28.2	62.5±30.0	79.2±19.6
PPD																
≤4 mm	0	0	0	0	0	0	0	0	42.1	47.4	0	0	47.4	57.9	0	0
5 mm	0	0	0	0	0	0	0	0	26.3	31.6	26.3	10.5	36.8	26.3	42.1	21.1
≥6 mm	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	31.6	21.1	73.7	89.5	15.8	15.8	57.9	78.9
PAL change (mm)									0.5±1.1	0.6±0.3	0.1±0.7	0.3±1.1	0.7±1.2	0.6±0.9	0.1±0.8	0.5±1.1

Treatment modalities: 1 = ultrasonic scaling + iodine; 2 = ultrasonic scaling + saline; 3 = saline irrigation; 4 = iodine irrigation.
Data are expressed as the mean ± SD.
$n = 20$.

Figure 1. Mean probing attachment level in mm (SD) at baseline, 3 months, and 6 months for the four different treatment modalities, $n = 20$. SD = standard deviation; USD = ultrasonic debridement.

the published literature [17,18]. In the present study, non-surgical periodontal therapy by means of an ultrasonic instrument plus the use of PVP-iodine or saline solution determined a reduction of PPD values; however, no difference was found between the use of PVP-iodine and saline solution as a cooling agent. These results are concordant with results reported by Grossi et al. [14] who treated a group of individuals with severe chronic periodontitis by means of ultrasonic scaling and antibacterial agents. In a position paper it was furthermore concluded that "...the addition of povidone iodine did not attain a better result than the use of water along with ultrasonic scaling" [19]. This is contrary to the findings of Rosling et al. [12] who studied the effect of 0.5% PVP-iodine used in an ultrasonic scaler as an adjunct to either surgical or non-surgical therapy and found more gain from clinical attachment in deep pockets receiving PVP-iodine plus non-surgical therapy than in other treatment groups. Furthermore, Rosling et al. [4] demonstrated the utility of 0.1% PVP-iodine used in an ultrasonic debridement. At 3, 6, and 12 months their patients showed a significantly lower average of probing depths and significantly more gain in clinical attachment than control patients treated with ultrasonic debridement and a water coolant. After 12 years of supportive periodontal therapy, PVP-iodine-treated patients exhibited significantly less additional loss of clinical attachment and less tooth loss than did control patients. A possible explanation for these conflicting results could be the different baseline characteristics in the included study populations. In the present paper 100% of the selected sites had deep pockets ($\geq$6 mm, mean initial PPD 6.75 mm). The corresponding figure in the work by Rosling et al. [4] was 3.9 mm as a mean value and only 18–21% of all sites had deep ($\geq$6 mm) pockets.

One important factor in the treatment of periodontal disease is removal of the biofilm from the tooth

surface [20]. It is well known that iodine has poor ability to penetrate biofilms and that it is a major advantage for colonizing bacteria to grow in biofilm formations [21]. One of the abilities is the protection from chemical products, for example PVP-iodine. The substantivity of an antimicrobial system implies the ability of that system to maintain adequate antimicrobial drug levels over a sufficient period of time. It is accepted that the amount of antimicrobial drugs necessary to affect the residents of a biofilm is several orders of magnitude greater than the amount required to inhibit planktonic bacteria [20]. Another explanation for not finding any additive effect of iodine could be that we used too low a concentration. Quirynen et al. [6] proposed that repeated applications of PVP-iodine at a 10% concentration were necessary to obtain an effect. In a study by Cargill et al. [22], it was shown that legionellae in biofilms were 135 times more resistant to iodination compared with microorganisms growing in a non-organized or planktonic fashion. Furthermore, to be effective, antimicrobials must reach their target site and be maintained there at sufficient concentrations long enough for their antimicrobial effect to occur. Earlier studies have shown that the antibacterial activity of iodine is of short duration [23,24]. In the present study each tooth were treated for 5 min. Iodine may also be negatively influenced by several biological factors such as dentin matrix, type-I collagen [25]. Earlier, Möller [24] described that iodine was readily inactivated by organic substances in the periodontal pocket.

The conclusions drawn from the present study are that ultrasonic debridement is effective in infection control of patients with severe chronic periodontitis And that povidine-iodine does not add any clinical benefit to the ultrasonic debridement alone under these circumstances.

References

[1] American Academy of Periodontology. Consensus report: dental plaque-induced gingival disease. J Periodontol 1999; 4:18–20.

[2] Sbordone L, Ramaglia L, Gulletta E, Iacono V. Recolonization of the subgingival microflora after scaling and root planing in human periodontitis. J Periodontol 1990;61:579–84.

[3] Petersilika GI, Ehmke B, Flemmig TF. Antimicrobial effects of mechanical debridement. Periodontology 2000 2000;28: 56–71.

[4] Rosling B, Hellström MK, Ramberg P, Socransky SS, Lindhe J. The use of PVP-iodine as an adjunct to non-surgical treatment of chronic periodontitis. J Clin Periodontol 2001;28:1023–31.

[5] Claffey N, Nylund K, Kiger R, Garrett S, Egelberg J. Diagnostic predictability of scores of plaque, bleeding, suppuration and probing depth for probing attachment loss: 3.5 years of observation following initial periodontal therapy. J Clin Periodontol 1990;17:108–14.

[6] Quirynen M, Teughels W, De Soete M, Van Steenberghe D. Topical antiseptics and antibiotics in the initial therapy of chronic adult periodontitis: microbiological aspects 2000. Periodontology 2002;28:72–90.

[7] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997;195 Suppl 2:3–9.

[8] Caufield PW, Allen DN, Childen NK. In vitro susceptibilities of suspected periodontopathic anaerobes as determined by membrane transfer assay. Antimicrob Agents Chemother 1987;31:1989–93.

[9] Maruniak J, Clark WB, Walker CB, Magnusson I, Marks RG, Taylor M, et al. The effect of 3 mouthrinses on plaque and gingivitis development. J Clin Periodontol 1992;19:19–23.

[10] Neidner R. Cytotoxicity and sensitization of povidone-iodine and other frequently used anti-infective agents. Dermatology 1997 1997;195 Suppl 2:89–92.

[11] Lanker-Kloossner B, Widmer HR, Frey F. Non-development of resistance by bacteria during hospital use of povidone-iodine. Dermatology 1997;195 Suppl 2:10–3.

[12] Rosling B, Slots J, Christersson LA, Gröndahl HG, Genco RJ. Topical antimicrobial therapy and diagnosis of subgingival bacteria in the management of inflammatory periodontal disease. J Clin Periodontol 1986;13:975–81.

[13] Christersson LA, Rosling BG, Dunford RG, Wikesjö UM, Zambon JJ, Genco RJ. Monitoring of subgingival bacteroides gingivalis and actinobacillus actinomycetemcomitans in the management of advanced periodontitis. Adv Dent Res 1988; 2:382–8.

[14] Grossi SG, Skrepcinsky FB, Decaro T, Robertson DC, Ho AW, Dunford RG, et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. J Periodontol 1997; 68:713–9.

[15] Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. J Clin Periodontol 1981;8:281–94.

[16] Lehmann EL. Statistical methods based on ranks. In: Non-parametrics: New York: Holden-Day; 1975. p. 5–43, 120–44.

[17] Badersten A, Nilveus R, Egelberg J. Effect of nonsurgical periodontal therapy. II. Severely advanced periodontitis. J Clin Periodontol 1984;11:63–76.

[18] Lindhe J, Westfelt E, Nyman S, Socransky SS, Heijl L, Bratthall G. Healing following surgical/non-surgical treatment of periodontal disease. A clinical study. J Clin Periodontol 1982;9:115–28.

[19] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000;71:1792–801.

[20] Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. Periodontology 2000 2002;28:12–55.

[21] Marsh PD, Bradshaw DJ. Physiological approaches to the control of oral biofilms. Adv Dent Res 1997;11:176–85.

[22] Cargill KL, Pyle BH, Sauer RL, Mcfeters GA. Effects of culture conditions and biofilm formation on the iodine susceptibility of *Legionella pneumophila*. Can J Microbiol 1992;38:423–9.

[23] Engström B, Hård Segerstad L, Ramström G, Frostell G. Correlation of positive cultures with the prognosis for root canal treatment. Odont Revy 1964;15:257–70.

[24] Möller ÅJR. Microbiological examination of root canals and periapical tissues of human teeth. Methodological studies. Odontol Tidskr 1966;74 Suppl 1:1–380.

[25] Portenier I, Haapasalo H, Örstavik D, Yamauchi M, Haapasalo M. Inactivation of the antibacterial activity of iodine potassium iodide and chlorhexidine digluconate against *Enterococcus faecalis* by dentin, dentin matrix, type-I collagen, and heat killed microbial whole cells. J Endod 2002;28:634–7.