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Non-surgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine: a prospective, randomized, controlled clinical study

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Abstract

Objectives: The aim of this prospective, parallel group designed, randomized controlled clinical study was to evaluate the effectiveness of an air-abrasive device (AAD) for non-surgical treatment of peri-implantitis.

Material and Methods: Thirty patients, each of whom displayed at least one implant with initial to moderate peri-implantitis, were enrolled in an oral hygiene program (OHI) and randomly instrumented using either (1) AAD (amino acid glycine powder) or (2) mechanical debridement using carbon curets and antiseptic therapy with chlorhexidine digluconate (MDA). Clinical parameters were measured at baseline, 3 and 6 months after treatment [e.g. bleeding on probing (BOP), probing depth (PD), clinical attachment level (CAL)].

Results: At 6 months, AAD group revealed significantly higher ($p < 0.05$; unpaired *t*-test) changes in mean BOP scores when compared with MDA-treated sites ($43.5 \pm 27.7\%$ *versus* $11.0 \pm 15.7\%$). Both groups exhibited comparable PD reductions (AAD: 0.6 ± 0.6 mm *versus* MDA: 0.5 ± 0.6 mm) and CAL gains (AAD: 0.4 ± 0.7 mm *versus* MDA: 0.5 ± 0.8 mm) ($p > 0.05$; unpaired *t*-test, respectively).

Conclusions: Within its limitations, the present study has indicated that (i) both treatment procedures resulted in comparable but limited CAL gains at 6 months, and (ii) OHI+AAD was associated with significantly higher BOP reductions than OHI+MDA.

Key words: air powder flow; air-abrasive device; amino acid glycine powder; non-surgical; peri-implantitis; plastic curettes

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Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests related to this study.
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The consensus report of the 6th European Workshop on Periodontology has confirmed that peri-implant diseases are infectious in nature (Lindhe & Meyle 2008). Peri-implant mucositis describes an inflammatory lesion that resides in the mucosa, while peri-implantitis also affects the supporting bone (Heitz-Mayfield 2008). The key parameter for the diagnosis of peri-implant mucositis is bleeding on gentle probing (BOP). In contrast, peri-implantitis is characterized by crestal bone level changes in conjunction with BOP and pus forma-

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tion with or without concomitant deepening of peri-implant pockets (Lang & Berglundh 2011). Nowadays, there is substantial evidence supporting the view that a poor oral hygiene, a history of periodontitis and cigarette smoking must be considered as risk indicators for peri-implant diseases (Heitz-Mayfield 2008). Most recently, the characteristics of biofilms in peri-implant disease have been extensively reviewed and linked to a mixed anaerobic infection dominated by Gram-negative bacteria but also high numbers of peptostreptococci and staphylococci (Mombelli & Décaillet 2011). According to a cause-related concept, the disruption of bacterial plaque biofilms was defined as primary objective for the treatment of peri-implant diseases (Mombelli & Lang 1994b). To accomplish this goal, several non-surgical treatment approaches including mechanical and ultrasonic debridement, the adjunctive use of chemical agents (i.e. irrigation with local disinfectants, local or systemic antibiotic therapy), or laser application have been used for non-surgical therapy of peri-implant mucositis and peri-implantitis (Renvert et al. 2008b). While mucositis lesions were proven to be reversible (Lang & Berglundh 2011, Salvi et al. 2011), non-surgical therapy of peri-implantitis was not found to be effective (Lindhe & Meyle 2008). In particular, previous controlled clinical studies have indicated that the effectiveness of non-surgical instrumentation of peri-implantitis lesions was unpredictable and the beneficial clinical outcome may be limited to a short-term period of 6–12 months (Karring et al. 2005, Schwarz et al. 2005b, 2006a, b, Renvert et al. 2006, 2008a, 2009). These results may primarily be explained by the fact that none of the currently available methods or devices used for implant surface debridement are effective in completely eliminating bacterial plaque biofilms from roughened titanium implant surfaces (Schwarz et al. 2005a, 2006d, e), thus impeding the establishment of a new bone-to-implant contact (Schwarz et al. 2006c). In order to overcome some of these limitations, the application of air-abrasive devices (AADs) has also been suggested for the treatment of peri-implant diseases. While these systems were effective to obtain a thorough debridement and decontamination of titanium implant surfaces, their application was associated with microscopically visible surface alterations (Augthun et al. 1998, Kreisler et al. 2005, Schwarz et al. 2009). However, these surface changes were influenced by the nature, particle size and composition of the powder. In particular, a difference in size by a factor of 2.5 as noted between different amino acid glycine powders (density = 2.16 g/cm^3) was not associated with any alterations at moderately rough titanium implant surfaces when compared with a sodium bicarbonate powder (density = 1. 61 g/cm^3) (Schwarz et al. 2009). Even though the hardness of glycine is not tabulated today, it is already known to be less abrasive compared with bicarbonate (Petersilka et al. 2003). Most recently, the effectiveness of an AAD using amino acid glycine powder applied by a newly designed nozzle to prevent emphysema formation in the adjacent tissue was compared with that of an Er:YAG laser device for non-surgical treatment of severe peri-implantitis. The results indicated that clinical improvements were similar but limited in both groups at 6 months (Renvert et al. 2011b). However, when considering the clinical limitations reported for non-surgical therapy of peri-implantitis lesions (Lindhe & Meyle 2008), one may speculate that severe disease progression has exceeded the potential clinical indication for both treatment approaches.

Therefore, the aim of the present study was to assess the effectiveness of AAD using amino acid glycine powder for non-surgical treatment of initial and moderate peri-implantitis lesions, using mechanical debridement and local antiseptic therapy as control.

Material and Methods

Study population and design

The study population consisted of 32 partially or fully edentulous patients (12 men and 20 women; mean age 60.6 ± 38.6 years) exhibiting a total of $n = 43$ implants (Table 1), all suffering from initial to moderate peri-implantitis (Mombelli & Lang 1994a). All patients attended the Department of Oral Surgery, Heinrich Heine University, Düsseldorf, Germany for surgical treatment procedures. Before participation, each patient was given a detailed description of the procedure and was required to sign informed consent forms. The study was in accordance with the Helsinki Declaration of 1975, as revised in 2000 and the study protocol was approved by the ethics committee of the Heinrich Heine University.

Table 1. Distribution of different implant systems in both groups at baseline

Group	BRA	CAM	FRI	ITI	TSV	NI
AAD ($n = 23$)	2	5	2	5	6	3
MDA ($n = 20$)	4	7	0	4	3	2

BRA, Brånemark System® (cylindrical screw, machined surface), Nobel Biocare AB, Göteborg, Sweden; CAM Camlog Screw Line® (cylindrical screw, microrough surface), Camlog Biotechnologies AG, Basel, Switzerland; FRI Frialit® (cylindrical-stepped screw, microrough surface), Dentsply Friadent, Mannheim, Germany; ITI ITI® (cylindrical screw, microrough surface), Institut Straumann AG, Basel, Switzerland; TSV Tapered Screw Vent® (tapered screw, microrough surface), Zimmer Dental, Freiburg, Germany; NI, non-identifiable implant systems; AAD, air-abrasive device; MDA, carbon curets+local antiseptic therapy.

The patients were randomly assigned to the following treatment procedures: (i) AAD, or (ii) mechanical debridement and local antiseptic therapy.

Randomization and power calculation

Randomization was performed according to a computer generated protocol (RandList®, DatInf GmbH, Tübingen, Germany).

For the given sample size of 15 patients (two drop outs) per group, a 80% power detecting a 1 mm difference in CAL changes between groups was calculated (Power and Precision, Biostat, Englewood, CO, USA). For the power analysis, a standard normal distribution was assumed. The probability of a Type I error was set at .001 Sigma (1.25) (i.e. standard deviation of the sampled population) was estimated based on the standard deviations observed in previous studies (Schwarz et al. 2005b, Schwarz et al. 2006a).

Patient selection

For patient selection, the following inclusion criteria were defined (Schwarz et al. 2010): (1) presence of at least one screw-type titanium implant exhibiting clinical [i.e. probing depth (PD) ⩾4 mm, BOP and suppuration] and radiographic (loss of supporting bone ≤ 30% compared with the situation after implant placement) signs of initial or moderate peri-implantitis, (2) no

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implant mobility, (3) single tooth and bridgework restorations without overhangings or margins, (4) no evidence of occlusal overload (i.e. occlusal contacts revealed appropriate adjustment), (5) presence of at least 2 mm of keratinized attached mucosa, (6) treated chronic periodontitis and proper periodontal maintenance care, (7) a good level of oral hygiene [plaque index (PI) <1] (Löe 1967), (8) no systemic diseases which could influence the outcome of the therapy [i.e. diabetes (HbA1c <7), osteoporosis, bisphosphonate medication], (9) non-smoker. Hollow cylinder implants were excluded from the study.

Oral hygiene programme

At 4 weeks before the treatment, all patients were enrolled in an oral hygiene program (OHI) and received supramucosal/gingival professional implant/tooth cleaning using rubber cups and polishing paste (Clean Polish® Kerr Hawe, Bioggio, Switzerland) and oral hygiene instructions on two to four appointments according to individual needs. A supramucosal/gingival professional implant/tooth cleaning and reinforcement of oral hygiene was performed at baseline (immediately before treatment) as well as 2, 4, 6, 8, 10, 12, 16, 20 and 24 weeks after treatment.

Clinical measurements

The following clinical measurements were performed immediately before treatment (baseline), as well as at 3 and 6 months using a periodontal probe (PCP 12): (1) PI (Löe 1967), (2) BOP, evaluated as present if bleeding was evident within 30 s after probing, or absent, if no bleeding was noticed within 30 s after probing, (3) PD measured from the mucosal margin to the bottom of the probeable pocket, (4) mucosal recession (MR) measured from the implant neck (IN) to the mucosal margin and (5) clinical attachment level (CAL) measured from IN to the bottom of the probeable pocket. The primary outcome variable was CAL. All measurements were made at six aspects per implant: mesiovestibular (mv), midvestibular (v), distovestibular (dv), mesio-oral (mo), midoral (o) and disto-oral (do) by one blinded and previously calibrated investigator (N. S.).

Intra-examiner reproducibility

Five patients, each showing two implants with PDs $\geqslant 4$ mm on at least one aspect, were used to calibrate the examiner. The examiner evaluated the patients on two separate occasions, 48 h apart. Calibration was accepted if measurements at baseline and at 48 h were within a millimetre at $>90\%$ of the time.

Treatments

In both groups, treatment was performed under local anaesthesia. AAD (Air Flow Master®, Perio-Flow® nozzle, EMS) was used with amino acid glycine powder (Air-Flow® Perio Powder, EMS) (d_{v10}: 5 μm, d_{v50}: 20 μm, d_{v90}: 63 μm; corresponding to the size below which is 10%, 50% (median particle size) and 90% of the total material volume, respectively) (Fig. 1a). The submucosal application was accomplished using a specially designed nozzle, consisting of a thin flexible plastic tube (length: 1.7 cm; diameter: 0.8 mm at the tip) that was fitted with three orthogonally orientated holes at 0°, 120° and 240° to the long axis of the tube (Fig. 1b). This specific design is associated with a horizontal exit of the air powder mixture and a reduced pressure of 1 bar, thus preventing emphysema formation in the adjacent tissue. The handpiece (Air-Flow® EL-308/A, EMS) was guided in a circular motion from coronal to apical parallel to the implant surface in a non-contact mode. The instrumentation time at each aspect (i.e. mesial, distal, vestibular and oral) was limited to 5 s, as recommended by the manufacturer (Figs 1c and d).

Mechanical debridement (MDA) was performed using carbon curets (Straumann, Waldenburg, Switzerland) followed by pocket irrigation with a 0.1% chlorhexidine digluconate solution (Corsodyl®, GlaxoSmithKline Consumer Healthcare, Bühl, Germany) (CHX) and submucosal application of 1% CHX gel (Corsodyl® Gel, GlaxoSmithKline Consumer Healthcare). In this group, instrumentation was carried out until the operator felt that the implant surfaces were adequately debrided (Figs 2a and b). All treatments were performed by the same experienced operator (T. S.).

Statistical analysis

The statistical analysis was performed using a commercially available software program (PASW Statistics 19.0, SPSS Inc., Chicago, IL, USA). Mean values

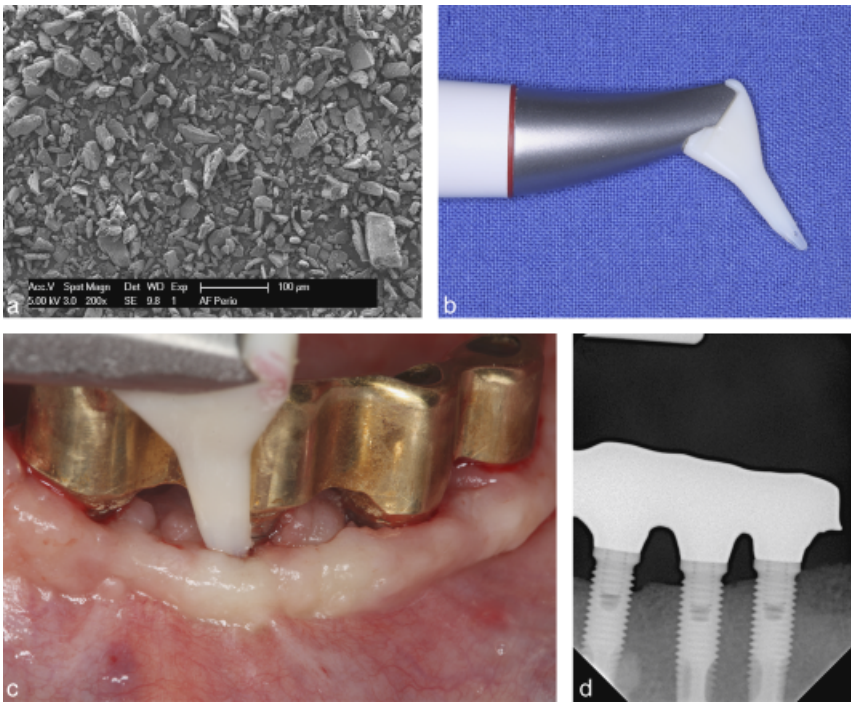

Fig. 1. (a) Scanning electron microscopic view of the amino acid glycine powder. (b) AAD handpiece with the connected flexible nozzle. (c) Non-surgical application of the nozzle within the peri-implant pocket. (d) Intra-oral radiograph indicating an initial bone loss of about 2–3 mm (region 041).

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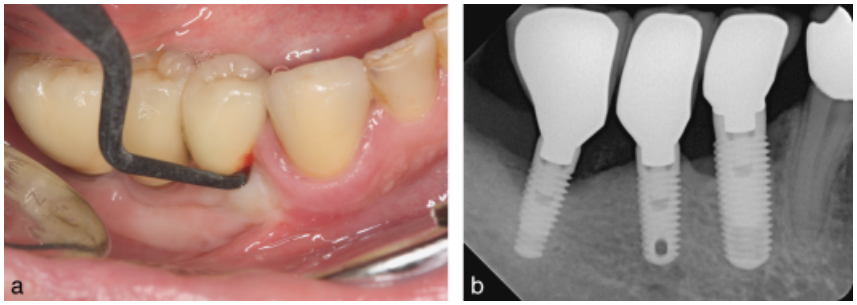

Fig. 2. (a) Non-surgical instrumentation in the MDA group. (b) Intra-oral radiograph indicating a moderate bone loss of about 4 mm (region 044). Implant 046 was not included in the present study but scheduled for a surgical treatment procedure.

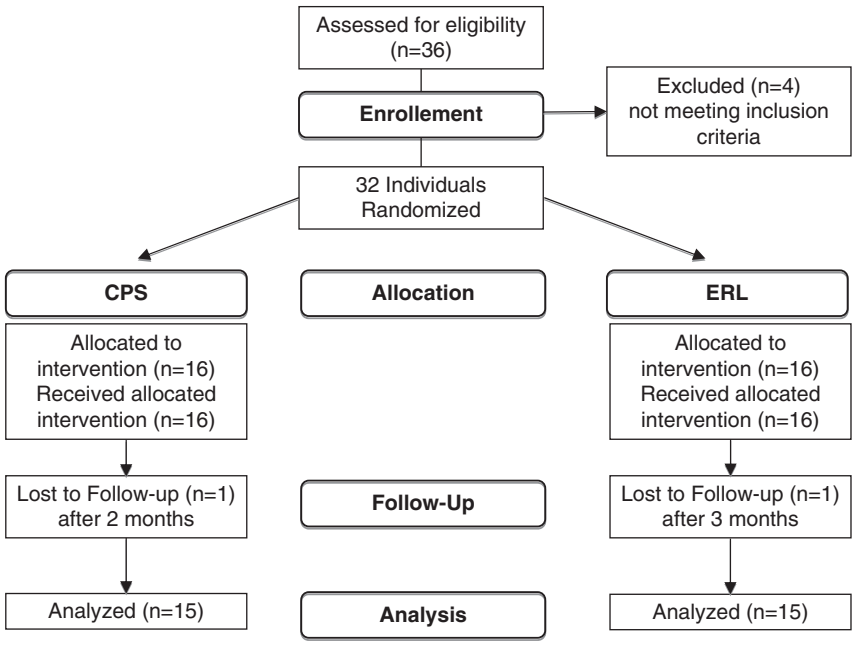

Fig. 3. A consort E-flowchart of the enrolment, allocation, follow-up and analysis.

and standard deviations were calculated for each variable and group using the patient as statistical unit. The data rows were examined with the Kolmogorow–Smirnow. The unpaired *t*-test was used for between group comparisons of mean baseline values (i.e. PI, PD, MR, CAL) and the changes in mean values from baseline to 3 and 6 months. Between group comparisons of mean BOP values at baseline were performed using the non-parametric Mann–Whitney test. The α error was set at 0.05.

Results

A total of $n = 30$ patients (i.e. two patients exhibiting one implant each refused to continue follow-up due to personal reasons after 2 and 3 months, respectively) (Fig. 3) terminated the observation period of 6 months and served for the statistical analysis (AAD: $n = 15$ patients exhibiting $n = 22$ implants; MAD: $n = 15$ patients exhibiting $n = 19$ implants). Non-surgical application of AAD was not associated with any emphysema formation. The post-operative wound healing was considered as generally uneventful in both groups. In particular, no complications such as allergic reactions, swellings, abscesses or infections were observed throughout the whole study period.

The mean PI, BOP, PD, MR and CAL values at baseline and after 3 and 6 months as assessed in both groups are presented in Tables 2 and 3. At the baseline examination, between group comparisons revealed no statistically significant differences in any of the investigated parameters ($p > 0.05$, unpaired *t*-test, Mann–Whitney test, respectively).

Mean PI scores remained low throughout the entire study period, without showing any significant differences between both groups ($p > 0.05$, unpaired *t*-test, respectively).

Mean BOP values were reduced in both AAD and MDA groups after 3 and 6 months of healing. Between group comparisons revealed a significant difference in mean BOP reductions at 3 and 6 months in favour of the AAD group ($p < 0.05$, $p < 0.05$, respectively; unpaired *t*-test) (Table 3).

The mean PD, MR and CAL values in both groups at baseline and after 3 and 6 months of healing are summarized in Tables 2 and 3. Both AAD- and MDA-treated sites exhibited a reduction of mean PD values at 3 and 6 months. Similarly, mean CAL values were improved in both groups at 3 and 6 months. Between group comparisons revealed no significant differences in mean PD and CAL reductions at 3 and 6 months ($p > 0.05$, respectively; unpaired *t*-test) (Tables 2 and 3).

Changes of mean PD and CAL values after 3 and 6 months of healing in both groups at sites with initial pocket depths of 1–3, 4–6 and $\geqslant 7$ mm are presented in Fig. 4a–d.

Basically, it was observed that greatest PD and CAL changes were observed at moderate sites (4–6 mm) in both groups. In particular, at 3 months, mean PD reductions and CAL gains were 1.1 ± 0.1 and 0.8 ± 0.1 mm in the AAD, and 1.0 ± 0.1 and 1.0 ± 0.2 mm in the MDA group, respectively. At 6 months, these values slightly decreased to 0.9 ± 0.1 and 0.6 ± 0.1 mm in the AAD, and 0.6 ± 0.1 and 0.6 ± 0.1 mm in the MDA group, respectively.

In contrast, initial sites (1–3 mm) exhibited the least amount of PD and CAL changes. In particular, at 3 months, mean PD reductions and CAL gains were 0.2 ± 0.1 and 0.2 ± 0.2 mm in the AAD, and 0.2 ± 0.1 and 0.1 ± 0.1 mm in the MDA group, respectively. At 6 months, these values remained almost unchanged, thus exhibiting 0.1 ± 0.1 and 0.2 ± 0.2 mm in the AAD, and 0.1 ± 0.1 and 0.3 ± 0.2 mm in the MDA group, respectively (Fig. 4a–d).

Advanced sites ($\geqslant 7$ mm) were only observed at two aspects in the AAD group, showing mean PD and CAL changes of 3.5 ± 1.5 and 3.0 ± 2.0 mm as well as 2.0 ± 0.0 and 2.0 ± 1.0 mm at 3 and 6 months, respectively (Fig. 4a–d).

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Table 2. Clinical parameters (mean ± SD) at baseline and 3 months for the AAD ($n = 15$ patients) and MDA ($n = 15$ patients) groups

	Baseline	3 months	Difference
Plaque index			
AAD	1.2 ± 0.8	1.0 ± 0.8	0.2 ± 0.8
MDA	1.0 ± 0.9	0.8 ± 0.8	0.2 ± 1.2
	NS*		NS*
Bleeding on probing			
AAD	94.6 ± 15.8%	43.0 ± 29.0%	51.6 ± 28.6%
MDA	95.3 ± 9.6%	70.4 ± 29.8%	24.8 ± 29.8%
	NS†		<0.05*
Probing depth			
AAD	3.8 ± 0.8 mm	3.0 ± 0.7 mm	0.8 ± 0.5 mm
MDA	4.0 ± 0.8 mm	3.2 ± 1.0 mm	0.8 ± 0.9 mm
	NS*		NS*
Mucosal recession			
AAD	1.0 ± 1.1 mm	1.1 ± 1.2 mm	− 0.1 ± 0.4 mm
MDA	0.7 ± 0.8 mm	0.7 ± 0.8 mm	0.0 ± 0.5 mm
	NS*		NS*
Clinical attachment level			
AAD	4.8 ± 1.3 mm	4.1 ± 1.1 mm	0.7 ± 0.5 mm
MDA	4.8 ± 1.3 mm	4.0 ± 1.2 mm	0.8 ± 1.1 mm
	NS*		NS*

Comparisons between groups (unpaired *t*-test* and Mann–Whitney test†).
AAD, air-abrasive device; MDA, carbon curets+local antiseptic therapy.

Table 3. Clinical parameters (mean ± SD) at baseline and 6 months for the AAD ($n = 15$ patients) and MDA ($n = 15$ patients) groups

	Baseline	6 months	Difference
Plaque index			
AAD	1.2 ± 0.8	1.1 ± 0.8	0.1 ± 0.7
MDA	1.0 ± 0.9	0.8 ± 0.7	0.2 ± 0.8
	NS*		NS*
Bleeding on probing			
AAD	94.6 ± 15.8%	51.1 ± 24.7%	43.5 ± 27.7%
MDA	95.3 ± 9.6%	84.3 ± 15.5%	11.0 ± 15.7%
	NS†		<0.05*
Probing depth			
AAD	3.8 ± 0.8 mm	3.2 ± 0.9 mm	0.6 ± 0.6 mm
MDA	4.0 ± 0.8 mm	3.5 ± 0.8 mm	0.5 ± 0.6 mm
	NS*		NS*
Mucosal recession			
AAD	1.0 ± 1.1 mm	1.2 ± 1.3 mm	− 0.2 ± 0.5 mm
MDA	0.7 ± 0.8 mm	0.7 ± 0.7 mm	0.0 ± 0.7 mm
	NS*		NS*
Clinical attachment level			
AAD	4.8 ± 1.3 mm	4.4 ± 1.3 mm	0.4 ± 0.7 mm
MDA	4.8 ± 1.3 mm	4.3 ± 0.9 mm	0.5 ± 0.8 mm
	NS*		NS*

Comparisons between groups (unpaired *t*-test* and Mann–Whitney test†).
AAD, air-abrasive device; MDA, carbon curets+local antiseptic therapy.

Discussion

The present prospective, randomized controlled clinical trial was designed to assess and compare the effectiveness of AAD and MDA for non-surgical therapy of initial and moderate peri-implantitis lesions. Over a short-term period of 6 months, both treatment procedures resulted in clinically important BOP and PD reductions as well as CAL (exception AAD at 6 months) gains. Between group comparisons revealed a significant difference in mean BOP reductions at both 3 and 6 months in favour of the AAD group. In this context, it is important to point to an increased efficacy of AAD over MDA to remove bacterial plaque biofilms from roughened titanium implant surfaces. In particular, AAD applied at two distances (1 and 2 mm) and angles (30° and 90°) to biologically contaminated titanium surfaces (R_a: 3.22 ± 0.88 μm) resulted in mean residual plaque biofilm areas (RPB) of 0.0 ± 0.0% to 5.7 ± 5.7% after a single (×1) application. Subsequent to a repeated (×2) surface treatment, mean RPB areas were reduced to 0.0 ± 0.0% (Schwarz et al. 2009). In contrast, previous studies employing the same intra-oral splint system reported higher mean RPB values on roughened titanium surfaces following application (×1) of plastic curets in combination with CHX (58.5 ± 4.9 to 61.1 ± 11.4%) (Schwarz et al. 2005a, 2006e). Based on these findings, one might speculate that AAD was associated with a more effective disruption of the peri-implant biofilm than MDA, thus reducing the amount of bacterial load and subsequently mean BOP scores at 3 and 6 months. However, previous microbiological data have indicated that mechanical debridement with or without local application of CHX is associated with a short-term reduction in total bacterial counts (Renvert et al. 2006, 2008a, 2009). In this context, it must be emphasized that one potential drawback of the present study was the lack of microbiological testing in both groups (i.e. AAD), which may be required in order to clarify this issue. This might particularly be of importance with respect to the multitude of different implant designs and surface characteristics investigated, which in turn may have an impact on either plaque biofilm formation (Renvert et al. 2011a), or the outcome of therapy (Albouy et al. 2011). When interpreting the present results, it was also noted that highest PD and CAL changes were observed at moderate sites (4–6 mm), while initial sites (1–3 mm) revealed the least amount of changes. The latter also indicates that non-surgical instrumentation of shallow pockets was not associated with a loss of CAL, thus pointing to the non-invasiveness of both MDA and AAD.

Basically, the clinical outcomes observed in the MDA group are in agreement with previous studies reporting on non-surgical therapy of moderate to advanced peri-implantitis over a period of 6 months (Karring et al. 2005, Schwarz et al. 2005b, 2006a, Renvert et al. 2006, 2008a, 2009). In particular, Schwarz et al. (2005b) reported on a significant decrease of mean BOP scores from 80% at baseline to 58% after 6 months and a significant mean CAL

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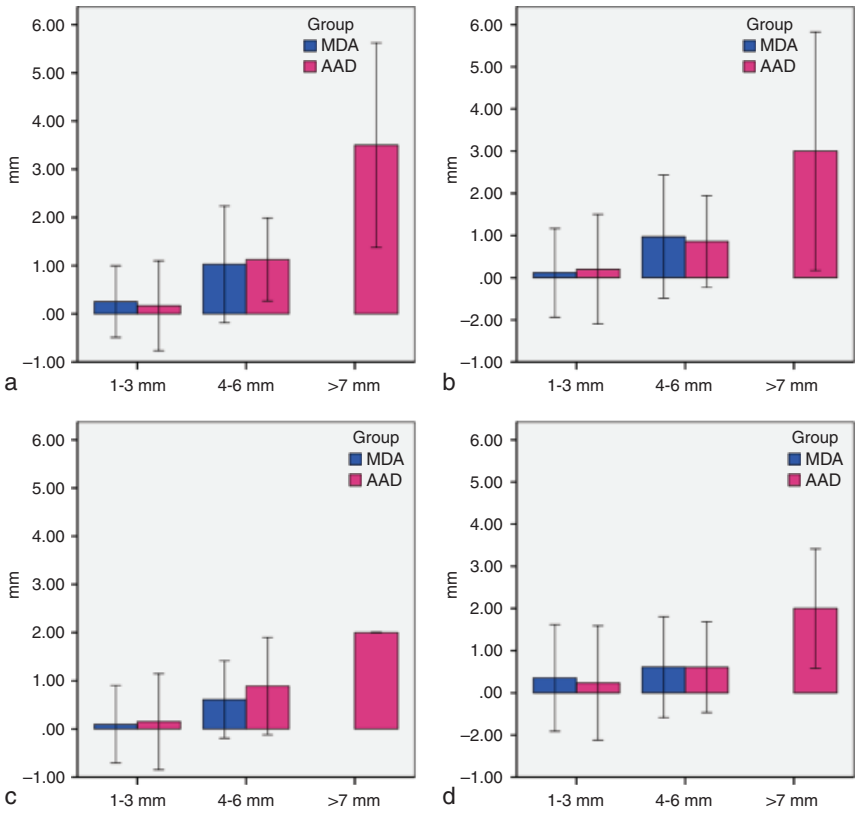

Fig. 4. Changes of mean probing depth (PD) and clinical attachment level (CAL) after 3 and 6 months of healing in both groups at sites with initial pocket depths of 1–3, 4–6 and ⩾7 mm ($n = 41$ implants). (a) PD – 3 months; (b) CAL – 3 months; (c) PD – 6 months; (d) CAL – 6 months.

change from 6.2 ± 1.5 mm at baseline to 5.6 ± 1.6 mm after 6 months. A site-specific analysis revealed that mean BOP was significantly reduced from 82% to 43% at moderate sites (4–6 mm), and from 88% to 53% at advanced sites (>7 mm) (Schwarz et al. 2006a). Mean PD was slightly reduced from 4.5 ± 0.8 to 4.2 ± 0.7 mm at moderate sites (4–6 mm), and from 6.0 ± 1.3 to 5.5 ± 1.0 mm at advanced sites (>7 mm). Mean CAL slightly changed from 5.1 ± 1.0 to 4.9 ± 0.9 mm at moderate sites (4–6 mm), and from 6.6 ± 1.4 to 6.2 ± 1.1 mm at advanced sites (>7 mm) (Schwarz et al. 2006a). Limited clinical improvements following non-surgical application of MDA (without CHX) were also reported by Karring et al. (2005), pointing out that only one site had stopped to bleed at 6 months after therapy. Similarly, Renvert et al. (2006) reported on a not significant mean BOP reduction from 86 ± 14% at baseline to 79 ± 14% at 6 months after MDA application using titanium curets. The corresponding mean PD values remained almost unchanged at 6 months (3.9 ± 0.3 *versus* 3.9 ± 0.4 mm) (Renvert et al. 2006). However, these results were obviously improved subsequent to a repeated (3×) application of CHX, resulting in a mean BOP reduction from 89.2 ± 17.2% at baseline to 62.8 ± 20.1% after 6 months. Similarly, mean PD values were reduced from 3.87 ± 1.16 mm at baseline to 3.68 ± 1.02 mm at 6 months (Renvert et al. 2008a). Interestingly, mean BOP scores were also significantly improved when titanium curets were applied without CHX (Renvert et al. 2009), which is basically in accordance with the results of the present findings. All these data seem to indicate that non-surgical therapy of peri-implantitis using MDA may reveal a limited clinical efficacy to control disease progression. In a most recent clinical study, however, Renvert et al. (2011a, b) also pointed to a limited outcome of clinical treatment using AAD, which was on a level equivalent to that noted for an Er:YAG laser device. In particular, mean PD reduction in the AAD group at 6 months was 0.9 ± 0.8 mm, corresponding to an absence of BOP at 25.0% of treated implants (Renvert et al. 2011b). The discrepancy noted between these results and the present data may be mainly attributed to differences in initial PD values. While Renvert et al. (2011a, b) focused on treatment of severe peri-implantitis, the present study mainly included patients suffering from initial to moderate lesions. Even though clinical improvements (i.e. PD reductions and CAL gains) were more pronounced at moderate sites (4–6 mm), the impact of initial PD on the outcome of non-surgical treatment of peri-implantitis still remains unknown. Similarly, BOP may be used as a predictor for loss of tissue support (Lindhe & Meyle 2008), however, the impact of residual BOP scores on disease progression in both MDA and AAD groups can only be evaluated on a long-term basis.

Finally, it is also important to emphasize that the application of AAD was not associated with any adverse events, such as emphysema formation or wound infections potentially caused by residues of the amino acid glycine powder. This observation is in agreement with recent clinical studies confirming the safety of this device for non-surgical therapy of periodontitis and peri-implantitis (Moene et al. 2010, Renvert et al. 2011b).

In conclusion, the present study has indicated that (i) both treatment procedures resulted in comparable but limited CAL gains at 6 months, and (ii) OHI+AAD was associated with significantly higher BOP reductions than OHI+MDA.

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Clinical Relevance

Scientific rationale for the study: Basic studies on the application of AAD on biologically contaminated titanium implant surfaces are promising. However, clinical data on non-surgical treatment of peri-implantitis lesions comparing AAD with conventional approaches such as MDA are lacking.

Principal findings: The present results have indicated that enrollment in an oral hygiene program (OHI) and non-surgical therapy of peri-implantitis using both AAD and MDA resulted in comparable PD reductions and CAL gains after 6 months of healing. However, mean BOP reductions at 6 months were significantly higher in the AAD when compared with the MDA group.

Practical implications: OHI+AAD may be more effective for the initial therapy of peri-implantitis than OHI+MDA.

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