Nonsurgical antimicrobial photodynamic therapy in moderate vs. severe peri-implant defects: A clinical pilot study

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Objective: Recent review articles have shown that open debridement is more effective in the treatment of peri-implantitis than closed therapy. However, surgery may result in marginal recession and compromise esthetics. The purpose of this study was to assess the efficacy of nonsurgical antimicrobial photodynamic therapy (aPDT) in moderate vs. severe defects. **Method and Materials:** The study encompassed 16 patients with a total of 18 ailing implants. Ten of these implants showed moderate bone loss (< 5 mm; Group 1) and eight implants severe defects (5 through 8 mm; Group 2). All implants received aPDT without surgical intervention. At baseline and 2 weeks, 3 months and 6 months after therapy, peri-implant health was assessed including sulcus bleeding index (SBI), probing depth (PD), distance from implant shoulder to marginal mucosa (DIM), and clinical attachment level (CAL). Radiographic evaluation of distance from implant to bone (DIB) allowed comparison of peri-implant hard tissues after 6 months. **Results:** Baseline values for SBI were comparable in both groups. Three months after therapy, in both groups, SBI and CAL decreased significantly. In contrast, after 6 months, CAL and DIB increased significantly in Group 2, not in Group 1. However, DIM-values were not statistically different 6 months after therapy in both groups. **Conclusion:** Within the limits of this 6-month study, nonsurgical aPDT could stop bone resorption in moderate peri-implant defects but not in severe defects. However, marginal tissue recession was not significantly different in both groups at the end of the study. Therefore, especially in esthetically important sites, surgical treatment of severe peri-implantitis defects seems to remain mandatory. *(Quintessence Int 2013;44:1–10; doi: ##.####/j.qi.a######)*

Key words: antibacterial photodynamic therapy, laser, peri-implantitis

Peri-implantitis is an inflammatory process around an implant, characterized by soft tissue inflammation and loss of supporting marginal bone.[1] Recent literature has summarized that peri-implantitis can be found in between 28% and 56% of subjects and between 12% and 43% of implant sites.[1,2] Due to the fact that a continually increasing number of patients are treated with dental implants, the frequency of peri-implant complications will rise over the long term.[3]

The primary goal of peri-implantitis treatment is to stop the progression of inflammation, which requires decontamination of the implant surface and, finally, augmentation of the defect. Conservative, resective, and regenerative treatment in conjunction with various methods of additional surface decontamination has been proposed.[4-6] However, based on these reports it appears that this goal is difficult to achieve.[2] At present, there is no reliable evidence suggesting which could be the most effective intervention for treating peri-implantitis.[7]

Factors influencing peri-implantitis treatment may include the surface texture of the

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implant, bone defect morphology, bone graft material, membrane exposure, and alteration of the reactive superficial titanium oxide during the decontamination procedure.[4,6] Additionally, it is known that conventional decontamination with dental curettes and air powder abrasives do not result in either sterile or isotonic sites.[8] To overcome these problems, various dental laser systems have been proposed for this purpose including CO_2 laser-energy,[8] diode lasers,[9,10] Er:YAG lasers,[11] and Er,Cr:YSGG laser.[12,13] Based on a recent review of the literature, it was concluded that treatment of peri-implantitis lesions with lasers as an adjunct to conventional treatment may lead to better clinical results than conventional treatment alone.[14]

Moreover, recent literature demonstrated that nonsurgical therapy was not found to be effective in peri-implantitis lesions. Based on evidence, it seems that the outcome of nonsurgical therapy is unpredictable. Open debridement including surface decontamination was more effective in the treatment of peri-implantitis than closed debridement.[14] In addition, open debridement including surface decontamination resolved peri-implantitis, promoted bone fill, and could result in re-osseointegration.[1]

However, surgical therapy may result in marginal recession of the soft tissue and thereby compromise the esthetic outcome.[15] Therefore, it seems to be of notable clinical importance to establish therapies that can stop the progression of the inflammation without recession of the mucosa. Antimicrobial photodynamic therapy (aPDT) was shown to be effective in the treatment of periodontitis.[16] Therefore, the aim of this study was to evaluate if aPDT is able to stop the progression of moderate and severe peri-implant bony defects.

METHOD AND MATERIALS

Dental laser

aPDT was performed with the medical handheld battery-operated diode laser HELBO TheraLite laser (HELBO minilaser 2075 F dent; HELBO Photodynamic Systems), which is operated in a continuous mode of laser beam delivery. This laser emits a beam of monochromatic light with a wavelength of 660 nm and a power output of 100 mW. When the Helbo 3D Pocket Probe is used, a power density of 60 mW/cm^2 is applied. The latter has an 8.5-cm-long flexible fiber optic tip curved at an angle of 60 degrees. The fiber is shielded with a tube made of stainless steel up to 8.0 mm long, with a conical polished tip at the front end. The exposure of the relevant area is effected radially around the conical tip of the Helbo 3D Pocket Probe, as well as axially at its tip (total area of the 8.0-mm-long tip: 0.17 cm^2). Therefore, within an irradiation time of 10 seconds, an energy fluence of 3.53 J/cm^2 is applied. As the laser device is classified as laser category 2M, the operator and patient did not have to wear any eye protection: a temporary exposure time (up to 0.25 seconds) is not judged dangerous for the eye, as long as the diameter of the laser beam is not narrowed by optical instruments such as lenses or telescopes.[16] The system was employed in combination with a commercial photosensitizer dye (phenothiazine chloride, HELBO Photodynamic Systems) (Fig 1). The photosensitizer dye contained 3,7-bis (dimethylamino)phenothiazin-5-ium chloride (methylene blue) at a concentration of 1% buffered to pH 3.5 with a citrate buffer, isotonized and viscosity-modified with 1% hydroxypropyl methylcellulose.[16] Dosage of the photosensitizer was 10 mg/ml.

Patients

The study included 16 patients who were sent to the Department of Oral and Maxillofacial Surgery in 2010 for therapy with a total of untreated 18 ailing implants (progressive vertical bone loss, probing depth [PD] ≤ 8 mm, bleeding on probing). All patients were informed that surgical debridement was more effective than closed debridement[14] but may result in marginal recession. Moreover, patients were advised that in this Department, presurgical oral hygiene was mandatory including oral hygiene instruction, plaque control with use of chlorhexidine solution (0.3 %) (Chlorhexamed, Blendax), and calculus removal (T1, Fig 2). Moreover, aPDT was performed 2 weeks later to minimize local infection (T2, Fig 2).[17] Surgical CO_2-laser therapy was

Fig 1 Medical diode laser (λ = 660 nm) HELBO TheraLite laser (HELBO minilaser 2075 F dent), Helbo 3 D Pocket Probe, and photosensitizer (phenothiazine chloride, dosage 10 mg/ml).

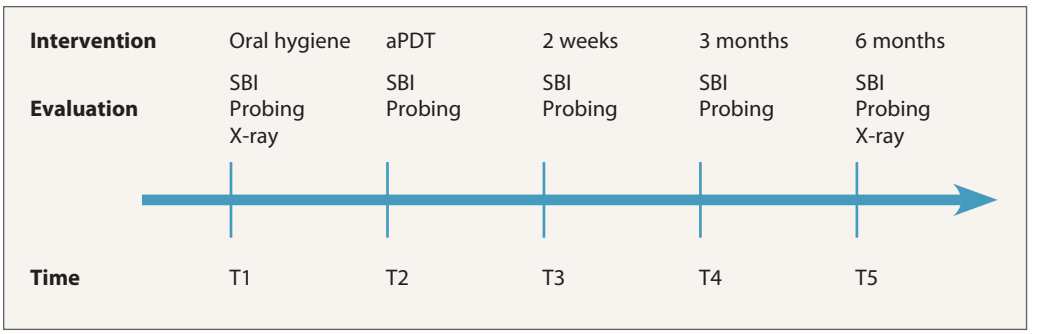

Fig 2 Outline of the clinical study.

planned after a 3-month period following this presurgical regimen if aPDT was not able to stop the progression of bone loss.

Treatment protocol

After a 2-week period of oral hygiene measures, all implants received aPDT without surgical intervention (T2). Due to this nonsurgical treatment, all restorations were left in situ. Before laser treatment, peri-implant pockets were rinsed with the photosensitizer, employing a blunt cannula and starting from the bottom of the pocket to achieve both a complete filling of the pocket and coating of the implant surface. The amount of utilized photosensitizer depended on the depth of the pocket; clinical experience has demonstrated that 0.1 ml was sufficient for three to four peri-implant defects. After a 3-minute residence time, the pockets were rinsed with sterile saline solution to remove excess photosensitizer. Employing the dedicated laser probe, the remaining photosensitizer was activated for 10 seconds per site, ie a total energy fluence of 3.53 J/cm^2 was applied maximally in deep pockets per site. Laser application was performed circumferentially at six sites per implant; ie, each implant was exposed to laser irradiation for 1 minute. The application time of both the photosensitizer and laser light was monitored by a time-controller belonging to the aPDT system. Antibiotics were not administered at any time before or after surgery.

Data collection

The peri-implant status of each subject was assessed at the beginning of the hygiene phase (T1; ie, 2 weeks before the laser intervention), immediately prior to aPDT (T2), 2 weeks after therapy (T3), and 3 (T4) and 6 months (T5) following therapy (Fig 2).

The peri-implant parameters were assessed according to the criteria proposed by Buser et al[18] and Mombelli et al[19] at four implant sites (mesial, distal, oral, vestibular) in millimeters, referenced to the implant shoulder or prosthetic crown and measured with a periodontal probe PCP 11 (Aesculap). These included sulcus bleeding index (SBI), PD, distance between the implant shoulder to the marginal mucosa (DIM), and clinical attachment level (CAL) (Table 1). Positive values for DIM[18] indicate that the implant shoulder protruded from the mucosa, and negative values indicate a submucosal position of the implant shoulder. PD[18] describes the distance from the mucosal margin to the clinical pocket base.

Table 1 Means and standard deviations at various times of evaluation for Group 1 (moderate bony defects < 5 mm) and Group 2 (severe bone loss between 5 and 8 mm)

	Time	SBI	PD (mm)	DIM (mm)	CAL (mm)	DIB (mm)
Group 1 (n = 10)	T1	1.8 ± 1.3	3.3 ± 0.8	0.5 ± 0.5	3.8 ± 1.3	3.9 ± 0.8
	T2	0.7 ± 0.8	2.6 ± 0.6	0.8 ± 1.1	3.4 ± 1.3	NS
	T3	0.7 ± 0.7	2.8 ± 0.8	0.6 ± 1.3	3.4 ± 1.1	NS
	T4	0.8 ± 0.8	2.5 ± 0.6	0.8 ± 0.3	3.3 ± 1.3	NS
	T5	1.1 ± 0.9	2.9 ± 0.5	0.7 ± 0.4	3.6 ± 0.7	3.6 ± 0.8
Group 2 (n = 8)	T1	1.5 ± 1.2	5.8 ± 0.8	0.9 ± 1.2	6.7 ± 0.9	6.8 ± 0.8
	T2	0.9 ± 0.4	4.6 ± 0.7	1.6 ± 1.6	6.2 ± 1.1	NS
	T3	0.6 ± 0.6	4.7 ± 0.7	2.1 ± 1.3	6.8 ± 1.2	NS
	T4	1.1 ± 0.8	4.5 ± 0.6	2.3 ± 1.4	6.8 ± 0.7	NS
	T5	1.3 ± 1.1	6.5 ± 0.9	1.6 ± 1.2	8.1 ± 0.9	8.7 ± 0.7

NS, not specified

Consequently, the value for CAL was calculated as the sum of PD and DIM (CAL = PD + DIM).[18]

At baseline (T1), eight patients (five in Group 1 and three in Group 2) had undergone various radiographic diagnostics at the general dental practitioner (intraoral and extraoral projections). In the other eight patients, panoramic radiographs (Orthoralix; Digora, Soredex) were obtained in this Department. Six months after therapy (T5), panoramic radiographs were taken in all 16 patients in this Department. The radiographic distance DIB (distance from implant shoulder to the first bone contact) was calculated at mesial and distal sites, according to the method of Buser.[18] The implant features, with design characteristics of known size, facilitated radiographic measurements of crestal bone level at the approximal sites.

Due to the cumulative interceptive supportive therapy (CIST)-protocol[20] and in accordance with the respective periodontal literature,[21] defects were classified as moderate when the periodontal probe was stopped at a depth of no more than 5 mm. Accordingly, a PD of 5 through 8 mm classified defects as severe.

Statistical analysis

Statistical analysis was performed using a commercial computer program (Microsoft Excel, v. 97). Data are presented as means ± standard deviation or as counts or proportions (Tables 1 and 2).

Two-tailed Student's *t* tests permitted comparison of the clinical and radiologic parameters at T1 vs. T4 and T5. A *P* value ≤ .05 in the two-tailed test was considered to indicate statistical significance.

RESULTS

Clinical observations

Following aPDT, none of the patients needed pain relief. Moreover, postoperative edemas were not seen and all of the treated defect sites healed uneventfully. Clinical observations are shown in Fig 3. No implant was lost during the 6-month follow-up period.

Clinical parameters

Table 1 and Table 2 provide information on the clinical parameters in Groups 1 and 2, which include SBI, PD, DIM, and CAL. With respect to SBI, there was no statistically significant difference at T1 between the two groups. Moreover, both groups demonstrated very similar values during the whole study: at T1 (beginning of the hygiene phase), the highest SBI values were observed. Subsequently, SBI each dropped

Table 2 **Analysis of clinical and radiographic parameters (means and standard deviations): DIB and CAL values indicate at T5 that progression of bone resorption was stopped successfully only in Group 1**

Parameter		Mean ± SD	No.	*t* value	Statistically significant difference
SBI	T1: Group 1 / Group 2	1.8 ± 1.3 / 1.5 ± 1.2	10 / 8	0.69	No
	T4: Group 1 / Group 2	0.8 ± 0.8 / 1.1 ± 0.8	10 / 8	1.125	No
	T5: Group 1 / Group 2	1.1 ± 0.9 / 1.3 ± 1.1	10 / 8	-0.66	No
PD	Group 1: T4 / T1	2.5 ± 0.6 / 3.3 ± 0.8	10	-3.99	Yes
	Group 2: T4 / T1	4.5 ± 0.6 / 5.8 ± 0.8	8	-5.72	Yes
	Group 1: T5 / T1	2.9 ± 0.5 / 3.3 ± 0.8	10	-2.40	Yes
	Group 2: T5 / T1	6.5 ± 0.9 / 5.8 ± 0.8	8	2.05	Yes
DIM	Group 1: T4 / T1	0.8 ± 0.3 / 0.5 ± 0.5	10	3.0	Yes
	Group 2: T4 / T1	2.3 ± 1.4 / 0.9 ± 1.2	8	2.64	Yes
	Group 1: T5 / T1	0.7 ± 0.4 / 0.5 ± 0.5	10	1.5	No
	Group 2: T5 / T1	1.6 ± 1.2 / 0.9 ± 1.2	8	1.54	No
CAL	Group 1: T4 / T1	3.3 ± 1.3 / 3.8 ± 1.3	10	-1.15	No
	Group 2: T4 / T1	6.8 ± 0.7 / 6.7 ± 0.9	8	0.37	No
	Group 1: T5 / T1	3.6 ± 0.7 / 3.8 ± 1.3	10	-0.85	No
	Group 2: T5 / T1	8.1 ± 0.9 / 6.7 ± 0.9	8	4.10	Yes
DIB	Group 1: T5 / T1	3.6 ± 0.8 / 3.9 ± 0.8	10	-1.125	No
	Group 2: T5 / T1	6.8 ± 0.8 / 8.7 ± 0.7	8	-6.27	Yes

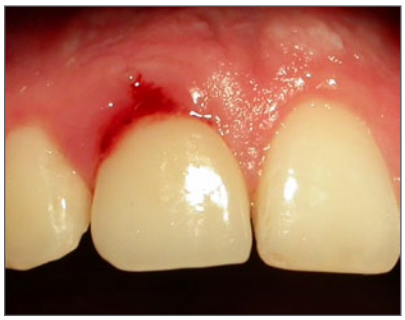

Fig 3a Clinical aspect of a chronic peri-implantitis lesion.

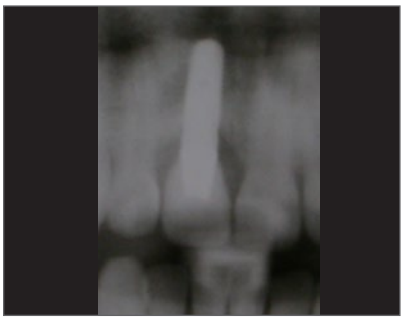

Fig 3b Radiograph showing vertical bony defects indicating progressive peri-implant bone resorption at T1.

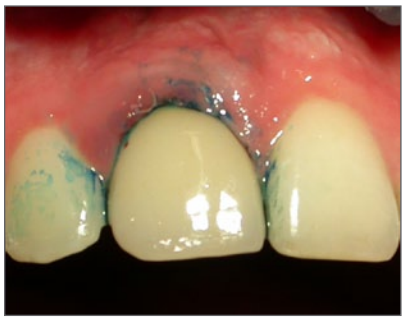

Fig 3c Clinical aspect following application of photosensitizer.

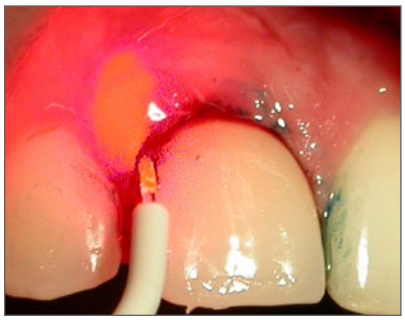

Fig 3d Nonsurgical intervention: application of diode laser light.

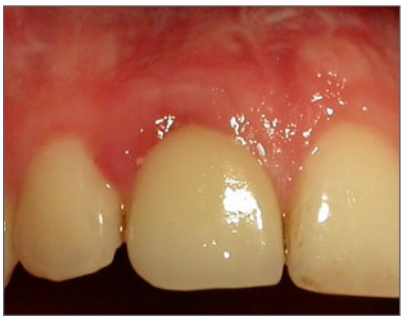

Fig 3e Clinical aspect 6 months after therapy: local infection is visible.

about one index-point at the time of laser application (T2) and increased slightly further into the investigation (T4, T5). Therefore, patients in both groups demonstrated comparable oral hygiene.

Three months after therapy (T4), in both groups, values for PD had decreased significantly and values for DIM had increased significantly. However, 6 months after therapy (T5), statistically significantly better PD values were found in Group 1, whereas in Group 2 these values were significantly worse when compared to T1. At T5, DIM values were not statistically different in both groups when compared to those at T1. Therefore, aPDT resulted in similar peri-implant soft tissue conditions without marginal recession within the 6-month follow-up.

The CAL in the two groups showed clearly different values (moderate vs. severe defects) due to the design of the study. CAL values accordingly decreased, especially in Group 1, and were almost stabile in Group 2 between T1 and T4. At T4, CAL levels in both groups did not differ significantly from those obtained at T1. Moreover, the difference between T1 and T5 was not statistically significant within Group 1 ($P > .05$) (Table 2). In contrast, in Group 2, CAL values were significantly worse at T5 as compared those at T1. Therefore, CAL values indicated that attachment loss was stopped successfully at T5 only in Group 1, not in Group 2.

Radiology

Table 1 provides data on the bony attachment (DIB values) in both Groups at T1 and T5. Again, due to the design of the study, DIB values demonstrated clearly different values in both groups. However, within Group 1, the difference between T1 and T5 was not statistically significant ($P > .05$) (Table 2).

In contrast, in Group 2, DIB values were significantly worse at T5 as compared those at T1. Therefore, DIB values indicated (in accordance with the CAL values) that progression of bone resorption was stopped at T5 successfully only in Group 1, not in Group 2.

DISCUSSION

Conventional measures for the therapy of peri-implantitis focus on open debridement, including mechanical and chemical modes of surface decontamination, which has been shown to be more effective than closed debridement.[14] Moreover, according to a consensus statement, different treatment approaches are indicated in moderate vs. severe peri-implant bony defects.[20] According to the CIST-protocol, antiseptic measures are recommended if the PD of the defects is less than 5 mm. In contrast, if a pocket depth of more than 5 mm is found in combination with a bone loss of more than 2 mm, resective or regenerative modes of therapy seem to be necessary.[20]

Recent literature has shown in vitro and in vivo that various laser wavelengths may have the potential to impede the progression of bone resorption caused by peri-implant infections.[8,9,10,22,23] For this purpose, several authors have recommended the use of diode lasers (λ 810 nm and 906 nm)[9,22] and Er:YAG lasers (λ 2.94 µm).[24] Moreover, in the beagle dog, histological studies have shown that CO_2-laser-assisted implant decontamination can even establish re-osseointegration.[8] However, surgical intervention can result in marginal tissue recession, which may compromise esthetics.[15,23] Moreover, it has been noted that nonsurgical treatment of peri-implantitis with use of adjunctive local or systemic antibiotics can reduce bleeding on probing and PDs, but the outcome over the long term was classified as unpredictable.[1] Accordingly, antibiotics were not used in the preset study. These findings legitimate the quest for new laser-assisted treatment procedures to improve the therapy of ailing implants. The limited access of topical agents to peri-implant plaque formation and the development of antibiotic resistance create the necessity for alternative noninvasive strategies to control biofilms and to treat peri-implant diseases.[16]

Accordingly, Haas et al[10] reported on augmentation of peri-implant defects with intraorally harvested autogenous bone following decontamination by photodynamic therapy with toluidine blue plus diode laser light (λ 906 nm) in a total of 24 ailing

implants. This method resulted in a mean bony reapposition of 2 mm (± 1.90 mm) after a 9.5-month observation period. In the present study, nonsurgical aPDT failed to result in bone gain but could stop the progression of the infection in moderate defects. However, in severe peri-implant bony defects, bone resorption was still ongoing 6 months after therapy. Nevertheless, clinical parameters PD and DIM indicated remarkable improvement of the inflammatory status 3 months after aPDT in both types of defects so that CO_2-laser-assisted open debridement seemed not to be indicated at this time.

The results of the present study demonstrated, therefore, limited effects of aPDT in severe peri-implant defects. Recent literature has focused on antibacterial photodynamic effects on biofilms. In a respective study, the impact of aPDT was evaluated in an artificial biofilm model.[25] Therefore, the authors also used phenothiazine chloride as a photosensitizer and a diode laser (λ 660 nm, output power 100 mW). It was concluded that laser irradiation is an essential part of aPDT to reduce bacteria within a layer of 10 µm. However, it was outlined that further studies are needed to evaluate the maximum biofilm thickness that still allows a toxic effect on microorganisms.[25] According to the limited effects of aPDT in severe peri-implant defects it must be assumed that there are different conditions in moderate and severe peri-implant pockets such as thickness of biofilms, concentration of protons (pH), or bleeding that have an impact on the effectiveness of aPDT. In further studies, the penetration depth of phenothiazine chloride in peri-implant soft tissue and bone should be evaluated more in detail.

The present study demonstrates that antibacterial photodynamic nonsurgical treatment of peri-implantitis lesions can stop the progression of inflammation in moderate bony defects. This is in agreement with the conclusion of a recent review to identify the most effective interventions for treating peri-implantitis around osseointegrated dental implants.[7] As yet, the complex phenomenon of peri-implant bone loss is not widely understood.[26,27] Recent literature has even shown that clinical and microbiologic analyses provide consistent findings that suggest differences in the quantity of plaque and bacterial species between teeth and dental implants.[28] Therefore, it was concluded from a review of the literature that, at present, there is no reliable evidence suggesting which could be the most effective interventions for treating peri-implantitis.[7] Moreover, it was summarized that peri-implantitis can be a chronic disease and retreatment may be necessary. Therefore, in accordance with the present findings, aPDT may be especially recommended for the treatment of moderate defects because it enables simple retreatment without surgical intervention.

Moreover, there are no reports in the available literature on adverse effects of aPDT in dentistry.[29] Its effects are mediated by singlet oxygen, which directly influences extracellular molecules of microbiota.[29] However, recent medical literature has suggested that topical photodynamic therapy may have adverse effects.[30] Among these, pain, phototoxic, and photoallergic reactions may occur. It has also been reported that singlet oxygen may induce oxidation of cellular DNA.[31] However, healthy human cells detoxify various oxygen species with the use of appropriate enzymes (catalase and superoxide dismutase) and antioxidative substances such as beta carotene, glutathione, alpha tocopherol, ascorbate, and bilirubin.[32] These mechanisms are essential for the survival of aerobic cells because reactive oxygen species are constantly produced during the natural cellular breathing process. This might explain why there is no convincing evidence for a carcinogenic effect of topical photodynamic therapy.[30] Regarding the molecular level of aPDT, it has to be stated that it is unknown if or how this treatment may influence inflammatory proteolytic molecules such as serine proteases, which are engaged in the destruction of soft tissues.[33]

However, using aPDT as the sole treatment regimen, mineralized deposits on the implant surface cannot be completely removed when nonsurgical therapy is performed. Such calculus formations may serve as an attachment base for microbiota and contribute to the progression of the inflammation.[16] Within the 6-month period of this study, evaluation of such adverse

effects cannot be expected, but may be assumed, and could explain the different results between moderate and severe defects. Therefore, the present results indicate that early therapy may be more reliable than treatment of severe defects.

It should be kept in mind that aPDT was regarded in this study only as an additional preoperative hygiene measure before CO_2-laser surgery was scheduled. Therefore, it was surprising that there were no statistically significant different CAL levels between T1 and T4 in both groups, which indicated that progression of CAL loss was stopped in both groups. These findings allowed the authors to reevaluate clinical findings after another 3-month period (at T5).

From a methodical point of view it must be addressed that eight radiographic values were obtained from different, nonstandardized projections provided by the patients' general dental practitioners (T1). Due to radiation protection, provision of new standardized radiographs was not indicated. Accordingly, comparison with the DIB values obtained from standardized panoramic radiographs at T5 is only acceptable from a scientific point of view in the eight patients who had undergone panoramic radiography in this Department at T1, not in the eight patients who had obtained radiography at their general dental practitioner. However, it was shown experimentally in a beagle dog study that there are no statistically significant differences between PD values, radiographically obtained DIB values, and histometrically determined measurements at the time of surgery (ie, T1 in this study).[34] Accordingly, DIB values at both T1 and T5 demonstrated good accordance with the CAL values. Therefore, it seems justified to accept the CAL values as basis of the clinical conclusion.

In addition, one has to be aware that this encompassed different types of ailing implants in the anterior and the lateral aspects of the maxilla and mandible. However, present treatment recommendations do not differ according to the type of implant or prosthetic superstructure.[20] Therefore, the present results seem not to be compromised in this respect.

In the present study, within an irradiation time of 10 seconds, an energy fluence of 3.53 J/cm^2 was applied to the surrounding structures. At first sight, there seems to be a huge difference compared with the fluence reported in a previous study.[35] The authors had reported that an energy fluence of 212.23 J/cm^2 was applied when using a laser probe with a spot size of 0.06 cm in diameter, while all other laser parameters were held constant to the ones used in the present study. Therefore, it must be assumed that Novaes et al[35] had used a Helbo 2D Spot Probe, which does not provide radial irradiation.

At present, especially in esthetically important sites, surgical treatment of peri-implantitis seems to remain mandatory. When surgery is performed, the use of innovative materials for augmentation, such as enamel matrix derivatives, may be of value.[36] Therefore, further studies are necessary to evaluate if photodynamic therapy applied before augmentation or in periodontal risk patients[37] is of value in the therapy of peri-implantitis lesions.

CONCLUSION

Within the limits of this 6-month study, nonsurgical aPDT could stop the bone resorption in moderate peri-implant defects, but ongoing resorption was found in severe defects according to clinically assessed attachment levels. However, marginal tissue recession was not significantly different in both groups at the end of the study. From these results it may be concluded that especially in esthetically important sites, surgical treatment of severe peri-implantitis defects seems to remain mandatory.

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