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Predictive value of clinical and microbiological parameters for the treatment outcome of scaling and root planing

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Abstract

Objectives: To compare the clinical and microbiological outcome of non-surgical periodontal therapy after 6 months with data obtained after hygienic phase or 6 weeks after completion of non-surgical therapy, in order to evaluate the value of clinical and microbiological parameters to predict treatment success.

Material and Methods: Clinical and microbiological data were available from 271 sites in 10 systemically healthy non-smokers with moderate-to-advanced chronic periodontal disease (24–32 sites per individual). Subgingival plaque samples were tested for the presence of *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Tannerella forsythensis* and *Treponema denticola* using RNA probes.

Results: Stepwise multiple linear regression analysis revealed a significant impact of the number of sites with visible plaque index > 1 after hygienic phase on the bleeding tendency of a subject at month 6 (p < 0.01). Furthermore, an association could be demonstrated between the number of residual pockets (PD > 3 mm) 6 months after therapy and the number of bleeding sites and suppurating sites after hygienic phase (p = 0.016). Six weeks after therapy, the mean total bacterial loads had a significant impact on the bleeding tendency of a subject at month 6 (p < 0.01). Although the average numbers of sites with persisting *P. gingivalis, A. actinomycetemcomitans, T. forsythensis* and *T. denticola* seemed to be very similar 6 weeks and 6 months after therapy, large variations were noted between subjects, and therefore the microbiological status of a subject at week 6 could not predict the status at month 6. **Conclusions:** The present study showed a limited potential of microbiological tests, performed after hygienic phase or shortly after non-surgical periodontal therapy, to predict the clinical outcome 6 months later, but confirmed the importance of an establishment of perfect oral hygiene before non-surgical therapy.

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Today, therapy cannot be evaluated quickly after its completion. The key parameters probing pocket depth and attachment level, for example, may continue to improve over a period of more than 6 months (Kaldahl et al. 1996). A set of parameters allowing an early evaluation of treatment success would allow the clinician to decide rapidly whether additional, alternative or perhaps more aggressive therapy is necessary for his patient. A set of parameters with an established predictive value would also permit to make more realistic promises to the patient regarding the treatment result.

The issue of periodontal risk factors has been discussed widely in recent

years. Risk factors have been discussed to influence periodontal tissues on a site and on a subject level (for a review, see (Rees 2003). While the paramount role of smoking and systemic diseases, notably diabetes, has been clearly established on the subject level, little is known about the predictive value of most clinical and microbiological parameters on treatment outcome. The utility of assessing them at various time points before, during and after treatment thus remains to be validated.

In recent years microbiological tests have been introduced allowing the dentist to determine the presence of selected periodontal microorganisms. Can these tests help the clinician to determine success or failure of non-surgical therapy earlier than by re-evaluating the case after several months using clinical parameters only? To answer this question, the utility of these tests in the clinical decision-making process, and for the prediction of clinical outcomes needs to be determined.

The aim of the present study was to compare the clinical outcome of nonsurgical periodontal therapy after 6 months with baseline (BL) data, and to microbiological data obtained shortly after completion of therapy, in order to evaluate the predictive value of clinical and microbiological parameters to predict treatment success.

Material and Methods Subjects

Six male and four female subjects (mean age 45.0 ± 8.7 years, range 32-62 years) with moderate-to-advanced periodontal disease participated in this study. Subjects were systemically healthy, were non-smokers, were not taking any medication and had not been treated with antimicrobial agents at least during 3 months before the study. In order to be included, subjects had to have at least 20 teeth, including four molars. Prior to the study all subjects had received supragingival scaling and oral hygiene instructions (hygienic phase). The subjects included had at least four teeth with a pocket probing depth (PPD) of 6 mm or more.

Clinical protocol

One site on each tooth was selected on the basis of pocket depth and bleeding on probing (BOP) as follows: the deepest pocket was selected for each tooth; if several pockets were equally deep, the one BOPt was selected; if several equally deep pockets were bleeding, the sites were selected in such a way that they were as far apart from neighbouring study sites as possible. After the assessment of plaque index (PLI) (Silness & Löe 1964), supragingival plaque Table 1. Classification of clinical outcomes at month 6 on the basis of the subject

Category	Criteria
A	Absence of pockets deeper than 5 mm,
	no sites with further attachment loss >1 mm between baseline and month 6,
	absence of suppuration.
В	Absence of pockets deeper than 6 mm, no more than one site with further attachment
	loss of $>1 \text{ mm}$ between baseline and month 6,
	absence of suppuration.
С	Presence of one or more sites with pockets > 6 mm, and/or further attachment loss of
	>1 mm in several sites between baseline and month 6,
	and/or suppuration in one or several sites.

was removed if necessary and subgingival microbial samples were taken from the study sites using sterile paper points. One paper point was inserted to the bottom of the pocket and withdrawn after 10 s. The samples were not pooled; 24-32 specimens were taken in each subject. PPD were measured to the nearest millimetre with a standard periodontal probe (round tip, 0.4 mm in diameter and marks at 3, 6, 9, 12 mm). To determine attachment level changes longitudinally, the distance between the probe tip and a reference point on a splint was recorded. BOP was determined after 10 s. These parameters were recorded 2-3 weeks after the end of the hygienic phase and constituted BL.

The patients were then submitted to systematic deep scaling and root planing of all diseased teeth, carried out with manual and sonic instruments. Treatment was provided by an advanced graduate periodontal student and was usually completed in four sessions. After treatment, the pockets were irrigated with 0.1% chlorhexidine; the patients used the same solution as an oral rinse during 1 week after each treatment session. Clinical parameters were recorded again 6 months later.

Microbiological procedures

Paper points were stored in 4 M guanidinium thiocyanate 2-mercaptoethanol. The samples were analysed by the Institut für angewandte Immunologie (IAI, Zuchwil, Switzerland) using oligonucleotid probe technology according to standard procedures (Dix et al. 1990). They were hybridized to a specific probe for the ssrRNA of Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Tannerella forsythensis, Treponema denticola and to a universal bacterial probe (IAI and MicroProbe Corporation, Bothell, WA, USA). Bacterial counts were calculated by comparison with homologous reference standards and expressed as count $\times 10^6$. Microbiological parameters were recorded at BL, 6, 12 weeks and 6 months after treatment.

Data analysis

The results were analysed on the level of the subject and on the level of the site separately. On a site level, κ scores were calculated to evaluate the agreement between the presence and absence of target microorganisms at week 6 and month 6. Multiple linear regression models were calculated to explain treatment outcomes on the level of the subject and the site with clinical features recorded at BL, and microbiological data obtained at BL, 6 or 12 weeks after completion of therapy. To account for patient-specific effects in the site-specific analyses, the level of response (intercept) was assumed to depend on the individual.

To further evaluate the predictive value of microbiological parameters for clinical success of therapy, the patients were subdivided into three outcome categories on the basis of three clinical criteria (Table 1) assessed 6 months after treatment. On the site level, an outcome was considered successful if at month 6 a pocket was no longer deeper than 4 mm and not bleeding.

Results Patient-specific analysis

Clinical and microbiological data were available for analysis from 271 sites in 10 patients (24–32 per individual). Tables 2 and 3 list the clinical parameters for the 10 subjects individually. In both tables the patients are listed with increasing number of BOP-positive sites at month 6 (BOP+, M6).

Table 2 shows the age and the number of investigated sites for each patient; furthermore, it lists the individual number of sites showing a PLI > 1, the mean

Table 2.	Clinical data at basel	ine (BL) and 6 n	nonths after non-surgical	therapy (M6) for the	e 10 subjects treated i	n this study
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PAT	Age	n^*	PLI	>1*	Mear	n PPD	PPD<	4 mm*	PPD>	5 mm*	BO	P+*	SU	P+*
			BL	M6	BL	M6	BL	M6	BL	M6	BL	M6	BL	M6
1	34	26	0	2	4.27	2.65	11	25	7	0	26	3	0	0
2	43	24	1	2	4.04	3.04	10	19	5	2	21	8	0	0
3	32	28	1	0	5.14	2.96	4	21	14	0	22	8	2	0
4	39	32	0	0	4.97	3.66	7	19	10	7	15	10	0	0
5	49	28	0	0	7.11	3.89	0	11	27	3	28	11	9	1
6	45	28	1	2	3.86	2.89	15	22	5	0	20	12	0	0
7	47	28	0	2	4.71	3.29	6	18	10	1	16	13	0	0
8	49	24	0	7	5.88	3.17	4	16	11	0	22	14	0	0
9	62	25	3	7	6.08	3.16	0	20	15	2	25	17	1	0
10	50	28	5	3	5.75	3.43	3	17	10	2	28	25	2	0
Mean	45.0	27.1	1.1	2.5	5.2	3.2	6.0	18.8	11.4	1.7	22.3	12.1	1.4	0.4
SD	8.7	2.4	1.7	2.6	1.0	0.4	4.9	3.8	6.4	2.2	4.5	5.9	2.8	1.0

*Numbers of sites. The number of BOP-positive sites at month 6 (BOP+, M6) sorts subjects. PAT, patient; BOP, bleeding on probing; PPD, pocket probing depth; PLI, plaque index; Sup, suppuration.

Table 3. Numbers of sites initially deeper than 3 mm (PPD > 3 pre-treatment), and numbers of these sites gaining or loosing at least 2 mm of clinical attachment (>1 mm gain, >1 mm loss)

PAT	n	PPD>3 pre-treatment	AL cl base to mo	nange line onth 6
			>1 mm gain	>1 mn loss
1	26	15	6	0
2	24	14	3	0
3	28	24	10	0
4	32	25	5	2
5	28	28	12	2
6	28	13	0	0
7	28	22	8	0
8	24	20	10	0
9	25	25	15	0
10	28	25	6	0
Mean	27.1	21.1	7.5	0.4
SD	2.4	5.3	4.4	0.8

PPD, pocket probing depth; PAT, patient.

PPD, the number of shallow sites (PPD <4 mm), the number of deep sites (PPD > 5 mm), the number of sites BOPt and the number of sites showing signs of suppuration at BL and 6 months after non-surgical therapy (M6). Before therapy the overall mean probing depth of the selected sites was 5.2 ± 1.0 mm, 6.0 of these sites per patient had a PPD < 4 mm and $\overline{11.4}$ were deeper than 5 mm. After therapy, the overall mean probing depth of the selected sites was 3.2 ± 0.4 mm, 18.8 sites had a $PPD < 4 \,\mathrm{mm}$ and the number of sites deeper than 5 mm was reduced to 1.7. Table 3 shows the changes in clinical attachment levels of sites initially more

than 3 mm deep. On average, 7.5 of 21.1 examined sites showed at least 2 mm of attachment gain, and only 0.4 of these sites showed 2 mm or more attachment loss.

Table 4 shows the numbers of samples positive for Pg, Aa, Tf and Td, as well as the estimated mean bacterial load (TL). At least one sample was positive at BL for Pg, Tf and Td in all subjects, and eight persons were also positive for Aa. On average, 14.9 of 27.1 sites per patient were positive for Pg. This number was reduced to 4.7 at week 6, and decreased slightly to 3.0 at month 6. With regard to Aa 5.2 sites were positive at BL, 3.3 at week 6 and 1.3 at month 6. The average number of sites positive for Tf was reduced from 17.7 to 5.2, and was again at 5.2 6 months later. The number of Td-positive sites was reduced from 16.1 to 4.5; 4.9 sites were found positive for this organism at month 6. The last column of Table 4 shows the percentage of sites with a negative test for all four-target microorganisms 6 months after treatment. This value turned out to be correlated highly significantly with the subject's age (p = 0.005). Thus, the older the patient the bigger the chance that the target organisms were not suppressed below detection levels. A highly significant linear correlation was found between the age of the subject and the total bacterial load at BL (p = 0.001).

Stepwise multiple linear regression analysis was used to test the relationship between several clinical and microbiological parameters and the number of sites with a positive BOP 6 months after therapy. The number of sites with a PLI>1 and total bacterial load 6 weeks after therapy had a significant impact on the bleeding tendency at month 6 (p < 0.01, Figs 1 and 2).

The number of sites with a residual pocket depth greater than 3 mm 6 month after therapy was scrutinized next. Stepwise multiple linear regression analysis revealed an association of this parameter with the number of bleeding sites and suppurating sites at BL (p = 0.016). This parameter was also associated with increased number of sites positive for Pg at 6 weeks and Aa at month 3 (p = 0.004). No significant association could be established between observations at BL or 6 weeks after therapy and the presence of sites deeper than 3 mm BOP.

The classification of the subjects according to treatment outcome (see Table 1) yielded the following results: three of four patients with a good clinical outcome, fulfilling the criteria of category A, had been positive for Aa at BL, and none of them was positive anymore at month 6. All these subjects furthermore were Pg positive at BL in about 30% of the tested sites. Two subjects showed persistence of Pg at month 6, one subject in one site and the other in three sites, while the other two subjects were entirely negative. Tf was frequently detected in all subjects of category A at BL and was again detected 6 months later. Td was also frequently seen at BL. At month 6, two subjects remained positive for this species, one subject in one site and the other in three sites. Three patients matched one or several of the criteria of category C: Two of them were positive for Aa at BL, two at week 6 and all at month 6. All these subjects were

Table 4. Microbiological data for the 10 subjects treated in this study

PAT n	n	n MEAN T		N TL		Pg			Aa			Tf			Td				N%			
		BL	W6	M3	M6	BL	W6	M3	M6	BL	W6	M3	M6	BL	W6	M3	M6	BL	W6	M3	M6	M6
1	26	6.53	1.77	2.85	4.23	9	0	1	0	1	0	0	0	12	0	0	1	26	0	2	0	92.3
2	24	2.21	5.27	2.79	5.84	11	5	4	2	2	7	4	0	9	6	2	3	2	5	6	5	75
3	28	5.15	4.52	3.65	2.36	11	3	2	0	1	1	0	0	18	2	3	2	16	2	1	0	92.9
4	32	9.88	6.69	11.90	9.76	11	13	10	1	8	6	8	4	24	10	15	11	14	5	8	8	62.5
5	28	20.01	14.01	22.72	5.51	26	5	19	8	23	13	26	4	26	9	25	10	26	7	24	7	50
6	28	9.90	6.14	10.24	3.94	9	1	0	1	7	0	2	0	8	3	5	7	7	2	4	1	75
7	28	13.36	6.18	7.57	5.50	12	8	3	0	9	4	0	3	20	8	4	4	11	8	3	7	67.9
8	24	16.89	19.97	8.14	8.58	19	3	4	3	0	1	0	0	19	1	1	4	19	6	3	3	83.3
9	25	28.67	10.19	20.75	9.53	22	2	10	13	0	0	1	2	21	4	12	7	19	5	13	11	32
10	28	18.35	8.56	6.01	5.04	19	7	1	2	1	1	1	0	20	9	3	3	21	5	4	7	71.4
Mean	27.1	13.1	8.3	9.7	6.0	14.9	4.7	5.4	3.0	5.2	3.3	4.2	1.3	17.7	5.2	7.0	5.2	16.1	4.5	6.8	4.9	70.2
SD	2.4	8.0	5.3	7.1	2.5	6.1	3.9	5.9	4.2	7.1	4.3	8.1	1.8	6.1	3.7	7.9	3.4	7.8	2.5	7.0	3.8	18.7

TL, (total bacterial load), universal probe expressed as 10⁶ bacteria per sample); Pg, Aa, Tf, Td, numbers of positive sites; *N*%M6, percentage of sites negative for all target organisms at M6. Pg, *Porphyromonas gingivalis*; Aa, *Actinobacillus actinomycetemcomitans*; Tf, *Tannerella forsythensis*; Td, *Treponema denticola*.



Fig. 1. Correlation between plaque index (PLI) at baseline (BL) and bleeding tendency at month 6.

also Pg, Tf and Td positive in more than 30% of the tested sites at BL, and all remained positive for these three target organisms throughout the examination period. All three patients of category B were positive for all target organisms at BL. The microbiological response to therapy was variable.

In each of two subjects, two monitored sites lost more than 1 mm of clinical attachment. In these patients all four-target organisms were still detected in several sites at month 6.

Site-specific analysis

Tables 5 and 6 show the correlation between the presence or absence of Aa or Pg at week 6 and at month 6. The specificity and sensitivity of the presence of Aa at week 6 to predict its presence at month 6 was 0.89 and 0.42, respectively. The specificity and sensitivity of the presence of Pg at week 6 to predict its presence at month 6 was 0.83 and 0.23, respectively. The κ scores of agreement between the presence or absence at week 6 and month 6 on a site level were 0.05 for Pg, 0.17 for Aa, 0.26 for Tf, 0.29 for Td, indicating a poor-to-moderate predictive value.

The correlation of the presence or absence of Aa at week 6 to the clinical result at month 6 is shown in Table 7. The correlation of the presence or absence of Pg at week 6 to the clinical result at month 6 is shown in Table 8. This analysis includes only pockets deeper than 5 mm and known to harbour the respective microorganism at BL (Aa: 35 sites, Pg: 89 sites). Sensitivity and specificity of the specified microbiological tests were calculated assuming that success, defined as PPD < 5 mm not bleeding

at month 6, was the status attempted to be detected by the tests. The specificity and sensitivity of the absence of Aa to predict local clinical success were 0.57 and 0.35, respectively; the specificity and sensitivity of the absence of Pg to predict local clinical success were 0.60 and 0.18, respectively.

Multiple regression analyses were performed to determine the influence of BL conditions on gain of clinical attachment between BL and month 6. In addition, the influence of the presence or absence of Aa and Pg on gain of clinical attachment between BL and month 6 was evaluated. These analyses did not yield any consistent significant correlations.

Only one site showed signs of suppuration at month 6 (Table 2). This condition persisted in one patient who had nine suppurating sites at BL.

Discussion

The aim of the present study was to relate the outcome of non-surgical periodontal therapy to information available after the hygienic phase, or shortly after scaling and root planing.

Tables 2 and 3 show that most of the cases included in the present study were advanced and that, in general, treatment response was very favourable. A systematic review on clinical efficacy of subgingival debridement in the treatment of chronic periodontitis determined a weighted mean of attachment gain of 0.64 mm in pockets initially deeper than 4 mm, and a reduction of pocket depth of 1.18 mm (van der Weijden & Timmerman 2002). In the present study,



Fig. 2. Correlation between total bacterial load (TL) 6 weeks after therapy (W6) and bleeding tendency at month 6.

Table 5. Correlation between the presence or absence of Aa at week 6 and at month 6 (n = 271 sites)

Aa week 6	Aa m	onth 6
	positive	negative
Positive	5	28
Negative	7	231

Specificity 0.89, sensitivity 0.42, κ 0.17. Aa, *Actinobacillus actinomycetemcomitans*.

Table 6. Correlation between presence or absence of Pg at week 6 and at month 6 (n = 271 sites)

Pg week 6	Pg month 6				
	positive	negative			
Positive	7	40			
Negative	23	201			

Specificity 0.83, sensitivity 0.23, κ 0.05. Pg, *Porphyromonas gingivalis*.

the mean attachment gain amounted to 0.91 ± 1.21 mm and pocket depth decreased by 1.95 ± 1.40 mm. Table 3 shows that non-surgical therapy resulted in a high number of sites showing more than 1 mm of attachment gain, and that further loss of attachment could be kept to a minimum of four sites out of 271.

The clinical outcome 6 months after standard periodontal therapy was related to clinical and microbiological data available before and a few weeks after therapy. Even if the relatively small number of subjects precluded extensive

statistical testing, several associations could be established. The correlation between PLI and clinical outcome is noteworthy, and corroborates well-established clinical rules concerning the importance of meticulous oral hygiene for long-term success of therapy (Rosling et al. 1976, Nyman et al. 1977). Patients with several sites with visible plaque (PLI>1) after hygiene instructions showed a higher number of bleeding sites 6 months after treatment (Fig. 1). Multiple regression analysis revealed that this association was independent of the PLI at month 6. In addition, the correlation between total bacterial load at week 6 and bleeding at month 6 (Fig. 2) further reinforces the importance of good oral hygiene, because this parameter reflects the non-specific re-population of treated sites in the absence of efficient mechanical plaque control. The presence of bacterial deposits visible with the naked eye in a large number of sites correlating to the number of bleeding sites at follow up may be a useful point in patient motivation to perform optimal oral hygiene procedures. This finding also emphasizes the importance of establishing a plaque-free environment before proceeding to further therapy.

The persistence of bleeding upon probing has been associated with a moderate risk for future attachment loss and the absence of bleeding has been shown to be a good predictor for no loss of attachment in other studies (Lang et al. 1990, Joss et al. 1994). In the present study, the bleeding assessed shortly after hygienic phase had no discernible influence on outcome. This reinforces the notion that for the evaluation of the initial phase of treatment the assessment of the quality of plaque control seems to be particularly useful, whereas during maintenance the assessment of bleeding upon probing may be more useful.

The limitations in reproducibility of microbiological parameters have been debated previously (Mombelli et al. 1989, van Steenbergen et al. 1996) and limit the potential value for site-specific microbial diagnosis. Although the numbers of persisting positive sites in general seemed to be very similar 6 weeks and 6 months after therapy, one can see in Table 4 that, on the individual level, large variations were noted, and that therefore the microbiological status at week 6 had no predictive value for the status at month 6. Although overall percentages of target organisms are pretty stable, there is a high degree of variability from subject to subject, and even more so from site to site, resulting in a disappointing sensitivity of A. actinomycetecomitans or P. gingivalis at week 6 to predict presence at month 6 (Tables 5 and 6). On the other had, sites negative at week 6 had a very small chance to score positive at month 6, resulting in a good specificity in prediction.

A correlation of the subject's age with microbial parameters at BL (total bacterial load) as well as at month 6 (percentage of sites with a negative test for all four target microorganisms) is in agreement with reports showing generally higher levels of some putative periodontal pathogens in older subjects (Duffau et al. 2004).

In the present study, an attempt was made to define success using a few clinical parameters, both on the level of the patient (Table 1), as well as on the level of the site (Tables 7 and 8). Although the number of subjects precluded formal statistical testing, some patient-specific observations could be made: none of the patients with a good clinical outcome (category A) was A. actinomycetecomitans positive at month 6, two subjects were also entirely P. gingivalis negative, and the other two subjects were positive only in isolated sites. On the other hand, all three subjects falling into response category C were A. actinomycetecomitans positive at month 6. All these subjects were also Pg, Tf and Td positive throughout the examination period. These results con-

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Table 7. Correlation of the presence or absence of Aa at week 6 to the clinical success (PPD < 5 mm) at month 6 in 35 pockets deeper than 5 mm and positive for Aa at baseline

Aa	Clinical success (PPD<5 mm, no bleeding)				
	yes	no			
Positive at week 6, $n = 13$	10	3			
Negative at week 6, $n = 22$	18	4			

PPD, pocket probing depth; Aa, *Actinobacillus actinomycetemcomitans*. Specificity 0.57, sensitivity 0.35, κ -0.04.

Table 8. Correlation of the presence or absence of Pg at week 6 to the clinical success (PPD < 5 mm) at month 6 in 89 pockets deeper than 5 mm and positive for Pg at baseline

Pg	Clinical success (PPD<5 mm, no bleeding)				
	Yes	No			
Positive at week 6, $n = 19$	13	6			
Negative at week 6, $n = 70$	61	9			

PPD, pocket probing depth; Pg, *Porphyromonas gingivalis*. Specificity 0.60, sensitivity 0.18, κ -0.09.

firm previous observations between an association of certain microorganisms with the outcome of therapy. For instance, the persistence of pockets deeper than 4 mm was associated with a greater number of sites positive for P. gingivalis (Mombelli et al. 2000). Since studies suggest that the outcome of periodontal treatment is better if particular suspected pathogens, notably P. gingivalis and A. ctinomycetemcomitans, can no longer be detected after therapy (Slots & Rosling 1983, Christersson et al. 1985, Kornman & Robertson 1985, Haffajee et al. 1988, Rodenburg et al. 1990), and that positive sites are at greater risk for further break down (Slots et al. 1986, Bragd et al. 1987, Slots & Listgarten 1988, Fine 1994, Rams et al. 1996), an obvious microbiological goal of therapy would be the absence of periodontal target organisms. Since persisting deep pockets carry an increased risk for further break down (Renvert & Persson 2002). and since absence of bleeding upon probing could be associated with a high probability of periodontal stability (Lang et al. 1990), the clinical goal of therapy would be the elimination of deep pockets and bleeding sites.

Despite these associations, this case series clearly demonstrates the difficulty to predict the clinical outcome on the basis of site-specific data, assessed shortly after treatment (Tables 7 and 8). The specificity and sensitivity of absence of Aa (0.57, and 0.35, respectively), and of Pg (0.60 and 0.18, respectively) to predict local clinical success, indicated a limited discriminatory ability of the absence of these two organisms to identify a site going to respond favourably to therapy at month 6. This was not surprising given the poor agreement between the presence or absence at week 6 and month 6 on a site level for all tested taxa.

Conclusion

The present study showed a limited potential of microbiological tests, performed after hygienic phase or shortly after non-surgical periodontal therapy, to predict the clinical outcome 6 months later, but confirmed the importance of an establishment of good oral hygiene before non-surgical therapy: in patients still showing multiple sites with visible plaque (PLI>1) after the hygienic phase one can expect an increased tendency for BOPt at month 6.

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References

- Bragd, L., Dahlén, G., Wikström, M. & Slots, J. (1987) The capability of Actinobacillus actinomycetemcomitans, Bacteroides gingivalis and Bacteroides intermedius to indicate progressive periodontitis; a retrospective study. Journal of Clinical Periodontology 14, 95–99.
- Christersson, L. A., Slots, J., Rosling, B. G. & Genco, R. J. (1985) Microbiological and clinical effects of surgical treatment of localized juvenile periodontitis. *Journal of Clinical Periodontology* **12**, 465–476.
- Dix, K., Watanabe, S. M., McArdle, S., Lee, D. I., Randolph, C., Moncla, B. & Schwartz, D. E. (1990) Species-specific oligodeoxynucleotide probes for the identification of periodontal bacteria. *Journal of Clinical Microbiology* 28, 319–323.
- Duffau, F., Bolivar, I. & Baehni, P. C. (2004) Presence of periodontal pathogens in a lifetime. *Perio* 1, 67–73.
- Fine, D. H. (1994) Microbial identification and antibiotic sensitivity testing, an aid for patients refractory to periodontal therapy. *Journal of Clinical Periodontology* **21**, 98–106.
- Haffajee, A. D., Dzink, J. L. & Socransky, S. S. (1988) Effect of modified Widman flap surgery and systemic tetracycline on the subgingival microbiota of periodontal lesions. *Journal of Clinical Periodontology* 15, 255–262.
- Joss, A., Adler, R. & Lang, N. P. (1994) Bleeding upon probing. A parameter for monitoring periodontal conditions in clinical practice. *Journal of Clinical Periodontology* 21, 409–414.
- Kaldahl, W. B., Kalkwarf, K. L., Patil, K. D., Molvar, M. P. & Dyer, J. K. (1996) Longterm evaluation of periodontal therapy: I. Response to 4 therapeutic modalities. *Journal* of *Periodontology* 67, 93–102.
- Kornman, K. S. & Robertson, P. B. (1985) Clinical and microbiological evaluation of therapy for juvenile periodontitis. *Journal of Periodontology* 56, 443–446.
- Lang, N. P., Adler, R., Joss, A. & Nyman, S. (1990) Absence of bleeding on probing. An indicator of periodontal stability. *Journal of Clinical Periodontology* 17, 714–721.
- Mombelli, A., Minder, C. E., Gusberti, F. A. & Lang, N. P. (1989) Reproducibility of microscopic and cultural data in repeated subgingival plaque samples. *Journal of Clinical Periodontology* 16, 434–442.
- Mombelli, A., Schmid, B., Rutar, A. & Lang, N. P. (2000) Persistence patterns of *Porphyro*monas gingivalis, *Prevotella intermedia/* nigrescens, and Actinobacillus actino-mycetem-comitans after mechanical therapy of periodontal disease. Journal of Periodontology **71**, 14–21.
- Nyman, S., Lindhe, J. & Rosling, B. (1977) Periodontal surgery in plaque-infected dentitions. *Journal of Clinical Periodontology* 4, 240–249.
- Rams, T. E., Listgarten, M. A. & Slots, J. (1996) The utility of 5 major putative periodontal pathogens and selected clinical parameters to

predict periodontal breakdown in adults on maintenance care. *Journal of Clinical Periodontology* **23**, 346–354.

- Rees, T. D. (2003) Periodontal risk factors and indicators. *Periodontology 2000* **32**, 9–135.
- Renvert, S. & Persson, G. R. (2002) A systematic review on the use of residual probing depth, bleeding on probing and furcation status following initial periodontal therapy to predict further attachment and tooth loss. *Journal of Clinical Periodontology* 29 (Suppl 3), 82–89; discussion 90–81.
- Rodenburg, J. P., van Winkelhoff, A. J., Winkel, E. G., Goene, R. J., Abbas, F. & de Graaff, J. (1990) Occurrence of *Bacteroides* gingivalis, *Bacteroides intermedius* and *Actinobacillus actinomycetemcomitans* in severe periodontitis in relation to age and treatment history. *Journal of Clinical Periodontology* 17, 392–399.
- Rosling, B., Nyman, S., Lindhe, J. & Jern, B. (1976) The healing potential of the periodontal tissues following different techniques of periodontal surgery in plaque-free denti-

tions. A 2-year clinical study. *Journal of Clinical Periodontology* **3**, 233–250.

- Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavica* 22, 121–135.
- Slots, J., Bragd, L., Wikström, M. & Dahlén, G. (1986) The occurrence of Actinobacillus actinomycetemcomitans, Bacteroides gingivalis and Bacteroides intermedius in destructive periodontal disease in adults. Journal of Clinical Periodontology 13, 570–577.
- Slots, J. & Listgarten, M. A. (1988) Bacteroides gingivalis, Bacteroides intermedius and Actinobacillus actinomycetemcomitans in human periodontal diseases. Journal of Clinical Periodontology 15, 85–93.
- Slots, J. & Rosling, B. G. (1983) Suppression of the periodontopathic microflora in localized juvenile periodontitis by systemic tetracycline. *Journal of Clinical Periodontology* 10, 465–486.

- van der Weijden, G. A. & Timmerman, F. A. (2002) A systematic review on the clinical efficacy of subgingival debridement in the treatment of chronic periodontitis. *Journal of Clinical Periodontology* 29, 55–71.
- van Steenbergen, T. J. M., Timmerman, M. F., Mikx, F. H. M., de Quincey, G., van der Weijden, G. A., van der Velden, U. & de Graaff, J. (1996) Discrepancy between culture and DNA probe analysis for the detection of periodontal bacteria. *Journal of Clinical Periodontology* 23, 955–959.

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