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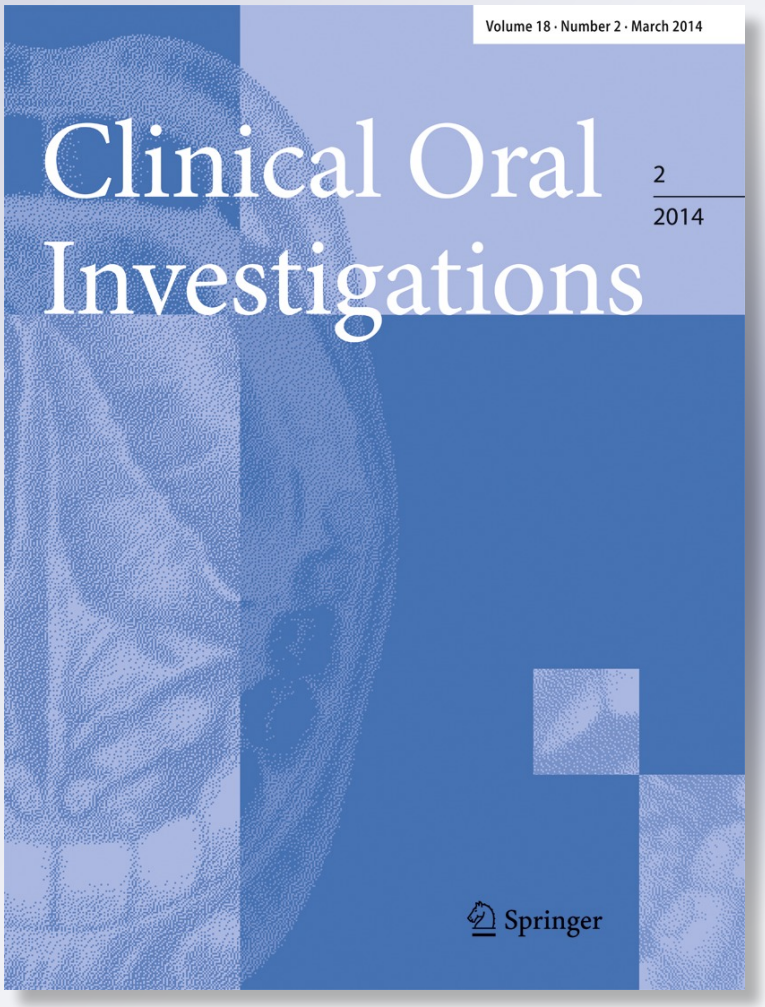

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ORIGINAL ARTICLE

Clinical results after nonsurgical therapy in aggressive and chronic periodontitis

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Abstract
Aim This study aims to analyze factors influencing treatment results in aggressive (AgP) and chronic (ChP) periodontitis.
Methods ChP [probing pocket depth (PPD)≥3.5 mm, attachment loss≥5 mm at >30 % of sites; age>35 years] and AgP (clinically healthy; PPD≥3.5 mm at >30 % of sites, radiographic bone loss≥50 % at 2 teeth; age≤35 years) were examined prior and 3 months after nonsurgical therapy according to the full-mouth disinfection concept. Adjunctive systemic antibiotics were used if *Aggregatibacter actinomycetemcomitans* had been detected at baseline.
Results In 31 ChP (12 female, 10 smokers; 4,808 sites) and 28 AgP (16 female, 9 smokers; 4,769 sites), overall mean PPD reductions were less favorable in AgP (0.9±0.5 mm) than in ChP (1.3±0.4 mm; p=0.033). PPD reductions and relative vertical probing attachment level gain were more favorable at sites with initial PPD≥6 mm, bleeding on probing, and for adjunctive systemic antibiotics. Furthermore, PPD reductions were more favorable for increased baseline tooth mobility and maxillary teeth, whereas AgP, female sex, and multirooted teeth were associated with less favorable PPD reduction.
Conclusion Regarding PPD reduction, AgP responded less favorably to nonsurgical treatment than ChP.

Keywords Aggressive periodontitis · Chronic periodontitis · Full-mouth disinfection

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Introduction

Nonsurgical anti-infective periodontal therapy, i.e., the reduction of supra- and subgingival microbial plaque by subgingival scaling and root planing (SRP), may result in resolution of inflammation, reduced probing pocket depths (PPDs) and clinical attachment gain [1, 2]. SRP of all pockets within 48 h in combination with use of antiseptic for different oral niches [full-mouth disinfection (FMD)] renders some additional benefits in chronic [3, 4] and aggressive [5] periodontitis. Use of systemic antibiotics also results in additional clinical benefits depending on baseline severity [6]. However, nonsurgical therapy is not found to be successful at all treated sites. Some factors influencing failure of nonsurgical therapy are known: deep periodontal pockets [7] and furcation involvement [8]. Nonsurgical therapy is effective in moderate periodontal pockets. In deep pockets, SRP is less effective [1, 2]. However, what difference does diagnosis make? There are some scarce data indicating that nonsurgical as well as surgical periodontal therapy is less effective in aggressive (AgP) than in chronic periodontitis (ChP) [9].

This study is a part of a bigger comparison of treatment results in patients with ChP or AgP [10, 11]. The hypothesis behind this analysis of the study is that SRP in AgP is less effective than in ChP of similar severity. Thus, the aim of the present analysis was to compare SRP between AgP and ChP with regard to patient and site factors.

Material and methods

Sixty-six patients with untreated severe periodontitis (31 generalized severe ChP and 35 AgP) were consecutively recruited at the Department of Periodontology of the Centre for Dental, Oral, and Maxillofacial Medicine (Carolinum), Johann Wolfgang Goethe-University Frankfurt/Main. They had to fulfill the following inclusion criteria: ≥16 years of age, ≥20 remaining teeth, written informed consent to be included into this prospective cohort study on full-mouth disinfection in ChP and AgP [10, 11].

AgP was defined as follows:

- Patient was clinically healthy, i.e., he or she did not suffer from systemic diseases predisposing to periodontitis (e.g., cyclic neutropenia) [12, 13]
- PPD≥3.6 mm at >30 % of sites
- Radiographic bone loss≥50 % at a minimum of two separate teeth
- Age at time of diagnosis≤35 years
- Inclusion into study up to 37 years of age.

Generalized severe ChP was defined as follows:

- PPD≥3.6 mm and vertical clinical attachment loss (PAL-V)≥5 mm at >30 % of sites
- PPD≥7 mm at a minimum of four sites
- More than 35 years of age

Individuals were not included in this study if they required systemic antibiotics for measures that may cause transitory bacteremia (e.g., pocket probing), self-reported chronic disease influencing the serum C-reactive protein level (e.g., rheumatoid arthritis, Crohn's disease, or ulcerative colitis), self-reported infectious disease within the last 8 weeks before examination (history of fever), showed any clinically assessed chronic dermal or mucosal inflammatory condition (e.g., lichen planus), had experienced nonsurgical or surgical periodontal treatment within the last 24 months before examination [9] or systemic or topical subgingival antibiotics within the last 8 weeks before examination [10, 11].

All patients reported their actual body weight and height as well as current and past cigarette smoking. Patients who currently smoked or had quit smoking for <5 years were classified as smokers [14]. Additionally ethnic origin was recorded [10]. The study complied with the rules of the Declaration of Helsinki and was approved by the Institutional Review Board for Human Studies of the Medical Faculty of the Goethe-University Frankfurt/Main (application number 188/06). All participating individuals were informed on risks and benefits as well as the procedures of the study and gave written informed consent.

Clinical examination

Clinical examinations are reported in detail elsewhere [10].

Clinical parameters were scored at six sites per tooth (mesiobuccal, buccal, distobuccal, mesiooral, oral, and distooral) at baseline and 12 weeks after SRP: gingival bleeding index (GBI) [15] and plaque control record (PCR) [16], PPD (standard probe), and relative vertical probing attachment level (RAL-V) (disk probe) to the nearest 0.2 mm using an electronic probe (Florida Probe, Version 3.2, Gainesville, USA). Bleeding on probing (BOP) was assessed 30 s after probing. Recession was measured to the nearest 0.5 mm using a manual periodontal probe (PCPUNC 15, HuFriedy, Chicago, USA) from the cemento-enamel junction (CEJ) to the gingival margin. If the CEJ was located apically to the gingival margin, the probe tip felt for it and the distance between CEJ and gingival margin was subtracted from PPD to calculate PAL-V [10, 11]. If the CEJ was not available due to restorations, the restoration margin was used as reference. At multirooted teeth furcation involvement was scored using a curved, scaled Nabers probe (Q-2N (SS+SSC) Hu-Friedy) [17].

Microbiological examination

At baseline and 12 weeks after therapy, subgingival plaque was sampled from the deepest pocket in each of the four quadrants. The test site was dried by air and kept dry using cotton rolls. Sterile paper points were inserted to the respective pocket. After 20 s, the paper points were removed. Each patient's four paper points were pooled into one transportation vial. For analysis, a commercially available 16S rRNA gene probe test kit was used (IAI Pado-Test 4.5®, Institut für angewandte Immunologie, Zuchwil, Switzerland) aiming at *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* [11, 18, 19]. This is an oligonucleotide probe test complementary to conservative regions of the 16S rRNA gene, which encodes the rRNA that forms the small subunit of the bacterial ribosome. The detection limit of this test is $10^{3.3}$ for *A. actinomycetemcomitans* and 10^{4} for *T. forsythia*, *P. gingivalis*, and *T. denticola*, respectively.

Antiinfective therapy

Prior to subgingival debridement (SRP), all patients had to provide a PCR≤50 %. SRP was performed in two visits on two consecutive days. Immediately after local anesthesia (UDS, Sanofi-Aventis Deutschland GmbH, Frankfurt/Main, Germany), each patient brushed the back of the tongue for 60 s with 1 % chlorhexidine (CHX) gel (chlorhexamed 1 % gel, GlaxoSmithKline, München, Germany) and rinsed as well as gargled 2 min with 10 ml of 0.12 % CHX solution (ParoEx, John O. Butler, Kriftel, Germany). All teeth exhibiting PPD≥3.5 mm were subgingivally debrided using sonic scalers (Sonicsys, KaVo, Biberach, Germany) and hand instruments. Immediately after instrumentation and 6 days after accomplishment of

debridement, 1 % CHX gel was applied into all debrided pockets three times within 10 min [20]. If *A. actinomycetemcomitans* had been detected from subgingival plaque, 500 mg amoxicillin and 400 mg metronidazole were prescribed three times daily for 7 days (in case of sensitivity to penicillin: 250 mg ciprofloxacin and 500 mg metronidazole two times daily for 7 days) [21, 22]. Oral home care for 14 days after SRP included rinsing two times daily for 60 s with 10 ml 0.12 % CHX solution (ParoEx), then brushing of teeth and the back of the tongue with 1 % CHX gel [10, 11].

Statistical analysis

PPD and RAL-V change 12 weeks after therapy were defined as the main outcome variables. Analysis was performed per protocol.

For all individuals, the body mass index (BMI) and cigarette pack years were calculated. Group frequencies (ChP and AgP) were expressed for sex, current smoking, and *A. actinomycetemcomitans* positive. Group means and standard deviations were calculated for age, number of remaining teeth, pack years, BMI at baseline as well as for GBI, PCR, and BOP at baseline and 12 weeks as well as for the changes between baseline and 12 weeks. For all site-based periodontal parameters (PPD and PAL-V), means per individual were calculated at baseline and 12 weeks as well as for changes from baseline to 12 weeks from which group means and standard deviations were calculated. Furthermore, the sum of all PPD was calculated per individual to describe the size of the interface between periodontal pocket and vascular system [23]. All bacterial counts were log-transformed, and group means and standard deviations were calculated for baseline and 12 weeks [11].

Between-group comparisons (AgP/ChP) for the main outcome variables (PPD and PAL-V change) were made by analysis of variance adjusted for baseline variables significantly different between the groups (baseline PPD, number of remaining teeth, pack years, and antibiotic treatment). For a clinically relevant difference of 0.4 mm, a type 1 error $\alpha > 0.05$, a standard deviation of change of 0.5 mm, and a per group sample size of 29 a post hoc power analysis was calculated. Further between groups comparisons (AgP/ChP) for PPD and RAL-V were made by independent *t* test and within-group comparisons by paired *t* test. Between- and within-group comparisons for dichotomous parameters were made by χ^2 or Fisher's exact test and for all other parameters by Mann–Whitney *U* test (between groups) or by Wilcoxon test or Friedman and in case of significant differences Wilcoxon test (within group).

Using interactive stepwise multilevel regression analyses, patient- and site-related factors should be identified that influenced the PPD and PAL-V change from baseline to 12 weeks [24, 25]. Multilevel analyses were modeled for the total of all sites and for different categories of baseline PPD (1.0–3.4 mm, 3.6–5.8 mm, and ≥6.0 mm). The following independent variables were entered into the models: for patient related, diagnosis, sex, age, BMI, smoking status, pack years, adjunctive systemic antibiotics, baseline log-transformed numbers of *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythia*, and *T. denticola*; for tooth/site related, BOP, baseline supragingival plaque, multirooted tooth, interproximal site, baseline PPD≥6 mm, jaw, and baseline mobility. GBI was not included in the analysis because of collinearity to BOP. All factors with $p<0.05$ were kept in the models. For statistical analysis, a PC program was used (Systat™ for Windows Version 10, Systat Inc., Evanston, USA) [11].

Results

Sixty patients (ChP, 31; AgP, 29) were enrolled between October 2006 and December 2009. Baseline parameters of these patients had been compared to healthy controls recently [10]. Patient characteristics are given in Table 1. One patient did not attend the 12 week re-examination without giving any reason. Change of serum inflammatory parameters has been reported recently [11]. Nine patients in the ChP group and 13 in the AgP group received systemic antibiotics (Table 2).

A total of 802 teeth (4,812 sites) were included in the ChP and 795 teeth (4,776 sites) in the AgP group. At baseline, 4,808 sites were measured in the ChP and 4,769 in the AgP group. For comparison of baseline and 12 weeks PPD assessments, 4,802 measurement pairs were available for ChP and 4,769 for AgP, respectively. Thus, 10 ChP sites and 7 AgP sites were missing for PPD comparisons. The respective numbers for PAL-V are 4,805 (ChP) and 4,761 (AgP).

Clinical variables were significantly improved in both groups (Table 2). Whereas PAL-V gain was similar for ChP and AgP, PPD reduction was more favorable in ChP than in AgP ($p=0.033$). Post hoc analysis revealed a test power of 85 %. Baseline mean PPD and PPD reduction as well as PAL-V gain are given for different baseline PPD groups separately in Table 3. Interactive stepwise multiple regression analysis identified aggressive periodontitis, female sex, and multirooted tooth to be negatively associated with PPD reduction 12 weeks after SRP (Table 4a). Adjunctive antibiotic therapy, baseline PPD≥6 mm, baseline BOP, baseline mobility, and location in the maxilla were positively associated with PPD reduction 12 weeks after SRP (Table 4a). Adjunctive antibiotic therapy, baseline PPD≥6 mm, and baseline BOP were positively associated with PAL-V gain 12 weeks after SRP (Table 4b). Interactive stepwise multiple regression analyses for PPD reduction and PAL-V gain are given for different baseline PPD groups separately in Tables 5, 6, and 7.

After this study was finished (12 week re-examination), eight dental implants were placed in eight patients (seven male; two ChP, six AgP). Six implants were inserted to replace teeth that had been extracted prior to FMD due to periodontal reasons. Two implants replaced teeth lost due to endodontic perforation and caries 46 and 4 months after re-examination of FMD, respectively.

Discussion

From a clinical point of view, does chronic periodontitis respond better or worse to SRP than aggressive periodontitis of similar severity? There are some scarce data indicating that nonsurgical as well as surgical periodontal therapy are less effective in AgP than in ChP [9]. This observation is confirmed by this study at least with regard to PPD reduction. Overall, FMD resulted in significant mean PPD reduction (ChP, 1.3 mm; AgP, 0.9 mm; $p<0.001$) and attachment gain (0.4 mm in both groups). These results are in accordance with results reported by other groups [5, 6, 9]. However, mean PPD reduction in the AgP group was less favorable than in the ChP group. Multilevel regression analyses identify AgP to negatively influence PPD reduction but not RAL-V gain. This study originally was designed to compare the effect of nonsurgical periodontal therapy on systemic inflammatory parameters between AgP and ChP [11]. Thus, it is appropriate to also compare the clinical results and respective influencing factors.

A. actinomycetemcomitans cannot be suppressed reliably under detection limits by SRP or flap surgery alone [26, 27]. However, using systemic amoxicillin and metronidazole adjunctively to SRP suppression of *A. actinomycetemcomitans* below detection limits is reliably achieved [28]. This has not been demonstrated for other periodontal pathogens [28]. Thus, we only used adjunctive amoxicillin (or in case of penicillin allergy ciprofloxacin) and metronidazole in *A. actinomycetemcomitans*-positive patients.

In this study, baseline PPD 1.0–3.4 exhibited better PPD reduction (ChP, 0.4 mm; AgP, 0.3 mm) 12 weeks after FMD than a structured review reports for baseline PPD 1–3 mm 6 months after SRP (0.04 mm) [2]. Whereas, the respective review reports small attachment loss (−0.15 mm) after SRP in shallow baseline pockets (PPD, 1–3 mm) [2], the actual study observed small mean RAL-V gains (ChP, 0.2 mm; AgP, 0.1 mm). This observation was also made for moderate (3.6–5.8 mm) baseline pockets. The differences between the results reported for shallow pockets in this study to the structured review may have several reasons. (1) A higher upper threshold (3.4 mm instead of 3 mm) leading to inclusion of deeper pockets, which respond better to subgingival instrumentation. (2) FMD results in better mean PPD reduction and

Table 1 Individuals' characteristics

Parameters	Chronic periodontitis (ChP)	Aggressive periodontitis (AgP)	*p* value
Patients	31	28	
Teeth	802	795	
Sites (measured at baseline)	4,808	4,769	
With baseline PPD 1.0–3.4 mm	2,373	3,017	
With baseline PPD 3.6–5.8 mm	1,583	1,131	
With baseline PPD≥6.0 mm	852	621	
With baseline furcation degree 0	248	377	
With baseline furcation degree I	241	221	
With baseline furcation degree II	108	82	
With baseline furcation degree III	60	48	
Female sex: *n*/frequency (%)	12 (39 %)	16 (57 %)	0.157
Age (years): mean±SD	52.8±7.6	31.4±5.5	<0.001
Ethnicity: *n*/frequency (%)			
African	0	4 (14 %)	0.045
Asian	2 (6 %)	4 (14 %)	
European	29 (94 %)	20 (72 %)	
Remaining teeth (*n*): mean±SD	25.9±2.8	28.4±2.0	<0.001
Current smokers: *n*/frequency (%)	10 (32 %)	9 (32 %)	0.992
Pack years: mean±SD	11.4±17.9	3.7±5.6	0.028
Body mass index (kg/m^2): mean±SD	25.5±3.8	26.5±5.3	0.421

PPD probing pocket depth, *SD* standard deviation

Table 2 Individuals' periodontal variables and change of periodontal variables after therapy

Parameters		Chronic periodontitis (ChP) (n=31)	Aggressive periodontitis (AgP) (n=28)	p value
Gingival bleeding index (%)	Baseline	15.5±12.2	12.8±8.9	0.513
	12 weeks	6.8±5.9**	5.8±4.3**	0.755
Plaque control record (%)	Baseline	34.6±17.0	40.3±15.0	0.052
	12 weeks	31.0±17.9	28.6±16.0*	0.649
Bleeding on probing (%)	Baseline	55.9±14.4	47.9±13.0	0.024
	12 weeks	25.8±10.9**	25.9±9.1**	0.970
Probing pocket depth (PPD) (mm)	Baseline	3.9±0.6	3.5±0.7	0.001
	12 weeks	2.6±0.5**	2.5±0.5**	0.288
PPD reduction (mm)		1.3±0.4	0.9±0.5	0.033[a]
Attachment level (mm) (PAL-V)	Baseline	4.2±1.7	2.9±1.4	<0.001
	12 weeks	3.8±1.7**	2.4±1.4**	<0.001
Attachment gain (mm) (ΔRAL-V)		0.4±0.4	0.4±0.4	0.222[a]
Sum of all PPD per person (mm)	Baseline	608.7±113.2	591.9±120.5	0.524
	12 weeks	409.2±82.0**	433.4±80.4**	0.471
A. actinomycetemcomitans (n)/frequency (%)	Baseline[b]	9/29	13/46	0.168
	12 weeks	2/6	2/7	1.000
P. gingivalis [log (n)]	Baseline	6.0±2.0	5.7±2.1	0.258
	12 weeks	3.1±3.2	2.7±2.8	0.261
Baseline PD≥6 mm: n/frequency (%)		852/18	620/13	<0.001

*p<0.05; **p<0.001, statistically significantly different to baseline

[a] Analysis of variance adjusted for baseline PPD, number of remaining teeth, pack years, and antibiotic therapy

[b] Antibiotic therapy

attachment gain compared to quadrant-wise scaling in chronic [3, 4] and aggressive [5] periodontitis. Most studies included in the structured review reported quadrant-wise scaling [2]. (3) In 22 patients, FMD was combined with adjunctive systemic antibiotics due to detection of *A. actinomycetemcomitans* from subgingival plaque. Use of systemic antibiotics has been shown to result in additional clinical benefits [6]. However, higher thresholds cannot explain better clinical results in moderate and deep pockets. In deep baseline pockets (≥6 mm), PPD reductions (ChP, 3.0 mm/AgP, 2.9 mm) are better, and attachment gains are similar to mean results reported by the review (deep pockets≥7 mm) [2]. Regardless of the

Table 3 Baseline probing pocket depth (PPD) and change of PPD as well as of relative vertical attachment level (ΔRAL-V) according to baseline PPD category over all sites (not per patient)

Parameters		Chronic periodontitis (ChP) (n=31)	Aggressive periodontitis; (AgP) (n=28)
Baseline PPD 1.0–3.4 mm		2,373	3,017
PPD (mm)	Baseline	2.3±0.7	2.3±0.7
PPD change (mm)	12 weeks	0.4±0.6	0.3±0.6
RAL-V change (mm)	12 weeks	0.2±2.4	0.1±2.8
Baseline PPD 3.6–5.8 mm		1,583	1,131
PPD (mm)	Baseline	4.6±0.7	4.6±0.7
PPD change (mm)	12 weeks	1.7±1.0	1.4±1.1
RAL-V change (mm)	12 weeks	0.9±1.4	0.8±1.4
Baseline PPD≥6.0 mm		852	620
PPD (mm)	Baseline	7.3±1.2	7.3±1.2
PPD change (mm)	12 weeks	3.0±1.7	2.9±1.8
RAL-V change (mm)	12 weeks	1.0±1.9	1.5±1.7

Table 4 Multilevel regression analysis: dependent variable: a) change of probing pocket depths (PPD) and b) relative vertical attachment level (RAL-V) 12 weeks after baseline

	Estimate	Standard error	*Z* value	*p* value
a) Dependent variable: PPD change; 59 patients/9,568 sites				
Intercept	0.515	0.057	8.988	<0.001
Aggressive periodontitis	−0.104	0.034	−3.057	0.002
Adjunctive antibiotics[a]	0.224	0.069	3.258	0.001
Female sex	−0.196	0.067	−2.919	0.004
Multirooted tooth	−0.173	0.024	−7.312	<0.001
Baseline PPD≥6 mm	1.922	0.033	58.179	<0.001
Baseline bleeding on probing	0.523	0.024	21.929	<0.001
Baseline mobility	0.086	0.018	4.902	<0.001
Maxilla	0.065	0.022	2.926	0.003
b) Dependent variable: RAL-V change; 59 patients/9,560 sites				
Intercept	0.113	0.048	2.337	0.020
Adjunctive antibiotics[a]	0.213	0.065	3.256	0.001
Baseline PPD≥6 mm	0.825	0.074	11.165	<0.001
Baseline bleeding on probing	0.183	0.053	3.443	0.001

[a] Only in cases with baseline detection of *A. actinomycetemcomitans*

different baseline PPD strata, overall PPD reductions and attachment gains observed in this study are favorable. This may be explained by FMD (used for all patients) and systemic adjunctive antibiotics (used in 22/59 patients, 37 %).

Multilevel regressions support these interpretations. In general, deeper pockets (PPD≥6 mm) respond with better PPD reduction and RAL-V gain. Furthermore, systemic antibiotics are associated with better PPD reduction and RAL-V gain. AgP is associated with less favorable PD reductions in shallow and moderate pockets, but not in deep pockets. Adjunctive antibiotics are associated with better PD reductions in moderate and deep pockets but not in shallow pockets. It is likely that shallow pockets do not harbor periopathogenic bacteria in an amount that require antibiotics to be suppressed substantially. Baseline detection of some bacteria is associated with more favorable clinical results (*P. gingivalis* in shallow and moderate pockets and *T. denticola* in deep pockets). Overall categories of baseline pockets multirooted teeth respond less favorably despite RAL-V gain in shallow pockets. In baseline shallow pockets, only small RAL-V gains were observed and severe furcation involvement is less likely and less favorable PPD reduction may be due to posterior position than furcation involvement. RAL-V changes in the PPD>6 mm subgroup are better for AgP (1.5 mm) than ChP (1.0 mm). However, this may be due to the effect of antibiotic therapy (Table 7b). There were more patients receiving antibiotics in the AgP than the ChP group.

FMD results in AgP with less favorable PPD reduction than in ChP. It has been shown that patients with AgP hyperrespond

Table 5 Multilevel regression analysis of all sites with baseline PPD 1.0–3.4 mm: dependent variable: a) change of probing pocket depths (PPD) and b) relative vertical attachment level (RAL-V) 12 weeks after baseline

	Estimate	Standard error	*Z* value	*p* value
a) Dependent variable: PPD change; 59 patients/5,384 sites				
Intercept	0.404	0.102	3.955	<0.001
Aggressive periodontitis	−0.052	0.024	−2.155	0.031
Baseline bleeding on probing	0.080	0.018	4.367	<0.001
Baseline *P. gingivalis*	0.050	0.015	3.313	0.001
Baseline *T. forsythia*	−0.057	0.021	−2.725	0.006
Female sex	0.058	0.024	2.452	0.014
Multirooted tooth	−0.101	0.019	−5.438	<0.001
Tooth mobility	0.033	0.014	2.338	0.019
b) Dependent variable: RAL-V change; 59 patients/5,383 sites				
Intercept	0.176	0.065	2.714	0.007
Adjunctive antibiotics[a]	0.177	0.086	2.054	0.040
Interproximal site	−0.149	0.072	−2.051	0.040

[a] Only in cases with baseline detection of *A. actinomycetemcomitans*

Table 6 Multilevel regression analysis of all sites with baseline PPD 3.6–5.8 mm: dependent variable: a) change of probing pocket depths (PPD) and b) relative vertical attachment level (RAL-V) 12 weeks after baseline

	Estimate	Standard error	*Z* value	*p* value
a) Dependent variable: PPD change; 59 patients/2,713 sites				
Intercept	1.551	0.127	12.213	<0.001
Aggressive periodontitis	−0.079	0.039	−2.008	0.045
Adjunctive antibiotics[a]	0.189	0.050	3.753	<0.001
Female sex	0.136	0.044	3.079	0.002
Baseline *P. gingivalis*	0.067	0.022	3.070	0.002
Multi-rooted tooth	−0.438	0.037	−11.818	<0.001
Interproximal site	−0.317	0.049	−6.437	<0.001
b) Dependent variable: RAL-V change; 59 patients/2,713 sites				
Intercept	1.146	0.190	6.028	<0.001
Baseline *P. gingivalis*	0.061	0.027	2.237	0.025
Interproximal site	−0.445	0.071	−6.267	<0.001
Multirooted tooth	−0.278	0.054	−5.182	<0.001
Maxilla	−0.141	0.053	−2.671	0.032
Tooth mobility	−0.084	0.039	−2.146	0.032

[a] Only in cases with baseline detection of *A. actinomycetemcomitans*

to inflammatory agents (i.e., subgingival biofilm). In untreated AgP, higher serum levels of elastase and C-reactive protein are observed than in ChP [10]. This hyperresponsiveness may cause higher levels of inflammation even after removal of subgingival biofilm and, thus, maintain pocketing. Hence, therapeutical considerations for AgP may be to take advantage from more frequent supportive periodontal treatment. Furthermore, additional anti-inflammatory therapy (e.g., subantimicro bial dose doxycycline) may be used [29, 30].

Within the limitations of the present study, the following conclusions may be drawn:

1. Regarding PPD reduction, AgP responded overall less favorably to nonsurgical treatment than ChP. However, regarding mean attachment gains both groups responded similarly.
2. Further factors influencing PPD reductions positively on the patient level were adjunctive antibiotics and on the

Table 7 Multilevel regression analysis of all sites with baseline PPD≥6 mm: dependent variable: a) change of probing pocket depths (PPD) and b) relative vertical attachment level (RAL-V) 12 weeks after baseline

	Estimate	Standard error	*Z* value	*p* value
a) Dependent variable: PPD change; 59 patients/1,473 sites				
Intercept	0.258	0.396	0.653	0.514
Adjunctive antibiotics[a]	0.593	0.168	3.536	<0.001
Baseline *T. denticola*	0.174	0.048	3.589	<0.001
Smoker	−0.372	0.179	−2.076	0.038
Female sex	−0.629	0.031	−20.430	<0.001
Baseline PPD	0.389	0.031	12.485	<0.001
Maxilla	−0.194	0.075	−2.600	0.009
Interproximal site	−0.553	0.119	−4.646	<0.001
Multi-rooted tooth	−0.764	0.075	−10.180	<0.001
b) Dependent variable: RAL-V change; 59 patients/1,472 sites				
Intercept	0.240	0.322	0.745	0.456
Adjunctive antibiotics[a]	0.595	0.190	3.133	0.002
Baseline PPD	0.206	0.039	5.324	<0.001
Multirooted tooth	−0.290	0.093	−3.103	0.002
Interproximal site	−0.623	0.148	−4.194	<0.001

[a] Only in cases with baseline detection of *A. actinomycetemcomitans*

tooth level were with initial PPD≥6 mm, BOP, increased baseline tooth mobility and maxillary teeth.

3. Further factors influencing PPD reductions negatively on patient level were female sex and on tooth level were multirooted teeth.

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