Maintenance after a Complex Ortho-Perio Treatment in a Case of Generalized Aggressive Periodontitis: 7-Year Result

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Abstract

Background: Generalized aggressive periodontitis (GAgP) encompasses a distinct type of periodontal disease exhibiting much more rapid periodontal tissue destruction than chronic periodontitis. The best method for management of GAgP may include the use of both regenerative periodontal techniques and the administration of systemic antibiotics. **Methods:** The treatment of a case of GAgP over a period of 6.7 years is presented in this case report. Initial periodontal therapy (week 1- 32) consisted of supragingival plaque control and three appointments of scaling and root planing. Based on the periodontal pathogens isolated (5 species), the patient also received metronidazole plus amoxicillin for one week, followed 10 weeks later by metronidazole plus amoxicillin/clavulanate for one week. The patient was put on regular supportive periodontal therapy (SPT) thereafter. Orthodontic treatment was performed after completion of the initial therapy for 96 weeks. Measurements of clinical attachment level, bleeding on probing and plaque index were obtained at every examination. **Results:** Antimicrobial and mechanical treatment resulted in eradication of all periopathogens and significantly improved all clinical parameters. During orthodontic treatment and active maintenance, there was no relapse of GAgP. The patient participated in SPT for 194 weeks and thereafter decided to discontinue SPT. Twenty-four months later a relapse of GAgP was diagnosed and all teeth had to be extracted. **Conclusions:** These results indicate that a combined mechanical and antimicrobial treatment approach can lead to consistent resolution of GAgP. Further studies including a larger number of cases are warranted to validate these findings.

Key words: Periodontal disease, generalized aggressive periodontitis, microbiology/diagnosis, orthodontic treatment

Introduction

Generalized aggressive periodontitis (GAgP) is characterized by rapid progression and destruction of periodontal tissues, and is often associated with certain periodontal pathogens and a high risk of disease relapse (Hoffmann *et al.*, 2007; American Academy of Periodontology, 2000; Armitage, 1999). The elimination or significant reduction of periopathogens is one of the major treatment goals (Liu, 2003). However, the etiology and role of periodontal pathogens in GAgP are not completely understood (Mombelli *et al.*, 2002). The combination of a compromised remaining dentition and the risk of later tooth loss due to GAgP relapse presents challenges in designing an appropriate treatment plan, including an appropriate supportive periodontal treatment schedule, use of adjunctive antimicrobial therapy as well as the prosthetic rehabilitation of patients with the disease. Long-term clinical success has been reported in the treatment of GAgP after regenerative periodontal treatment and/or implant surgery if additional emphasis was placed on control of the infection (Hoffmann *et al.*, 2007; Mengel *et al.,* 2007; Miliauskaite *et al.,* 2007). However, for patients with GAgP conventional mechanical debridement together with oral hygiene may not be sufficient (Buchmann *et al.,* 2002). Previous studies of this condition have shown beneficial effects of the adjunctive use of antibiotic therapy and have led to

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acceptance of this treatment approach for this type of periodontitis (Haffajee *et al.,* 2003; Herrera *et al.,* 2002; Purucker *et al.,* 2001; van Winkelhoff *et al.,* 1992). Nevertheless, additional attachment loss may occur at one or more sites, despite well-executed therapeutic efforts to stop disease progression (Kamma and Baehni, 2003).

This report presents a case of GAgP and argues for the importance of infection control during treatment and maintenance procedures. The results indicate that the long-term stability of periodontal tissues and retention of dentition rely largely on infection control.

Case

History

The 37-year-old female patient, a non-smoker, presented in February 1993 for periodontal treatment at the private practice of one of the authors (GGZ, Duesseldorf, Germany). The patient was in good general health and had not taken any medication. She reported more than 10 years of periodontal problems, including movement of teeth, gingival bleeding, and inflammation with a suppurative exudate and pain. Several teeth had already been extracted (#15, 16, 17, 18, 31 and 32).

Examinations

Generalized inflammation of the gingiva with recessions leading to partial loss of the curved contours and absence of stippling as well as halitosis was present. The interdental papillae were edematous, intensely red and bled spontaneously. Radiographs (*Figure 1F*) showed generalized horizontal and vertical bone loss with vertical defects over 2/3 of the root length. The restorations of the remaining dentition were insufficient.

At the initial examination (E1), neither probing measurements nor photographic documentation could be obtained due to the patient's discomfort. Tooth #1 was extracted at this point of time.

Subgingival plaque samples were taken with sterile paper points from the deepest pocket of each quadrant. These samples were pooled, placed in a transportation vial and sent to a commercial microbiological laboratory for polymerase chain reaction (PCR) analysis.

Clinical attachment level (CAL), bleeding on probing (BOP) and plaque index (PI) (O'Leary *et al.,* 1972) measurements were recorded at four sites per tooth. The cemento-enamel junction (CEJ) was used as the reference point for CAL measurements. In cases where the CEJ was not visible due to existing restorations their margin was used. All periodontal measurements were taken with the same periodontal probe (UNCP-15, Hu Friedy, Chicago, IL, USA).

Treatment

Preliminary treatment consisted of oral hygiene instruction, supragingival debridement, polishing of the teeth, and subgingival irrigations with chlorhexidine 0.1% (Chlorhexamed Fluid, GlaxoSmithKline, Buehl, Germany) every three days. Initial treatment was performed over a period of two weeks (*Table 1*).

Mechanical treatment: Scaling and root planing (SRP) of all quadrants was performed by means of Gracey curettes (Hu-Friedy, Leimen, Germany) in one session.

Systemic medication: Depending on the detected periodontal pathogens, the following systemic medications were prescribed immediately after SRP (Berglundh *et al.,* 1998): 1) For initial infection by *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*) alone or with other pathogens: 800 mg/d metronidazole (MTNZ) plus 1000 mg/d amoxicillin (AMO) for one week; 2) for continued or repeated infection by *A. actinomycetemcomitans* alone or with other pathogens: 800 mg/d MTNZ plus 1000 mg/d amoxicillin/clavulanate potassium (AUG) for one week (*Table 1*).

The first examination (E1) occurred directly after completion of preliminary treatment. The following pathogens were detected in the subgingival plaque (*Table 1*): *A. actinomycetemcomitans* (5×10^4); *Tannerella forsythia* (formerly *Bacteroides forsythus*; 5×10^4); *Campylobacter rectus* (5×10^3) and *Fusobacterium nucleatum* (5×10^3); *Porphyromonas gingivalis* (5×10^3); and *Treponema denticola* (1×10^4). SRP was performed and MTNZ and AMO were prescribed (*Table 1*).

The second examination (E2) occurred 10 weeks after E1 and revealed an improved clinical condition. The gingival tissues had regained their pink color and firm texture. Microbiological analysis showed the continued presence of *A. actinomycetemcomitans* (5×10^3), *T. forsythia* (5×10^3), and *P. gingivalis* (5×10^3). A second SRP was performed, and MTNZ and AUG were administered according to the above-mentioned schedule (*Table 1*).

The third examination (E3) was performed 10 weeks after E2, revealing a much improved clinical condition. However, the continued presence of *A. actinomycetemcomitans* (4×10^3) was detected. A third SRP was performed, and MTNZ and AUG were prescribed (*Table 1*).

Ten weeks after E3 (i.e., at E4), clinical and laboratory examinations did not detect periodontal pathogens. At E4, orthodontic treatment was started (*Figure 1 A-E*). The orthodontic examination revealed a skeletal Class II relationship with an overjet of 15 mm, lip incompetence (ANB 10°; *Figure 1A-E*) and mandibular retrognathia of 7 mm. Tooth #12 was extracted. Teeth #8 and 9 protruded labially, with increased interdental spaces between the central and lateral incisors. All mandibular teeth were mesially inclined, and the mandibular incisors were super-erupted without touching the protruding maxillary teeth. The anterior maxillary teeth showed advanced loss of periodontal attachment and were subject to non-physiological pressure from the lower lip. A straight-wire appliance with a 0.022 inch slot

Table 1. Clinical course of the patient.

	Examination								
	Initial Examination	1	2	3	4	5	6	7	8
CAL (mm) Mean ± SD (Min, max)	NR	10.04 ± 1.65 (8, 13)	9.90 ± 1.69 (8, 13.5)	9.92 ± 1.73 (8, 13.5)	9.31 ± 1.94*† (7, 14)	8.94 ± 1.92†‡ (7, 14)	8.94 ± 1.92‡ (7, 14)	8.16 ± 1.06†‡ (7, 10)	NR
BOP (%)	100	79.17	50	32.29*	32.29*	18.75*	27.08*	36.36*	100[@]
PI (%)	100	65.63	42.71	32.29*	32.29*	27.08*	27.08%*	38.64[§]	69.32
Pathogens	Aa, Cr, Fn, Pg, Tf, Td		Aa, Pg, Tf	Aa	ND	ND	ND	ND	NA
Treatment (Duration)	Initial treatment (2 weeks)	SRP; MTNZ + AMOX (1 week)	SRP; MTNZ + AUG (1 week)	SRP; MTNZ + AUG (1 week)	Start of orthodontic treatment (96 weeks)	End of orthodontic treatment and start of maintenance (every 2 mo)	Maintenance (every 2 mo)	Last maintenance session	Extractions, dentures
Time between Examinations		directly after initial treatment	10 weeks after exam. 1	10 weeks after exam. 2	10 weeks after exam. 3	96 weeks after exam. 4	16 weeks after exam. 5	178 weeks after exam. 6	96 weeks after exam. 7

Notes: CAL, clinical attachment level; BOP, bleeding on probing; PI, plaque index; NR, not recorded; ND, not detectable; NA, not applicable. Pathogens: Aa, *A. actinomycetemcomitans*; Cr, *C. rectus*; Fn, *F. nucleatum*; Pg, *P. gingivalis*; Tf, *T. forsythia*; Td, *T. denticola*; SRP, scaling and root planing; MTNZ, metronidazole; AMOX; amoxicillin; AUG; amoxicillin/clavulanate potassium. *$p < 0.01$ vs. examination 1, †$p < 0.001$ versus previous examination, ‡$p < 0.001$ versus examination 1, §$p < 0.01$ versus previous examination, @$p < 0.05$ vs. examination 1.

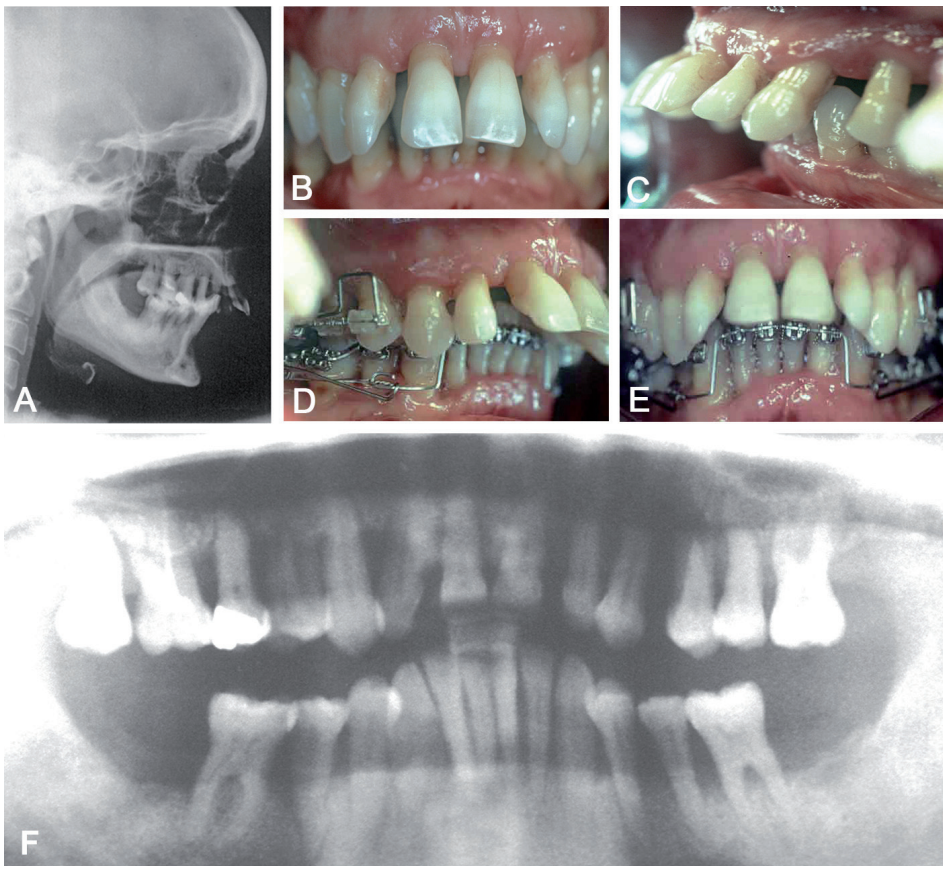

Figure 1. A: Lateral cephalogram before orthodontic treatment; B, C: Clinical situation at examination 4; D, E: Clinical situation during orthodontic treatment; F: Orthopantomogram at initial examination.

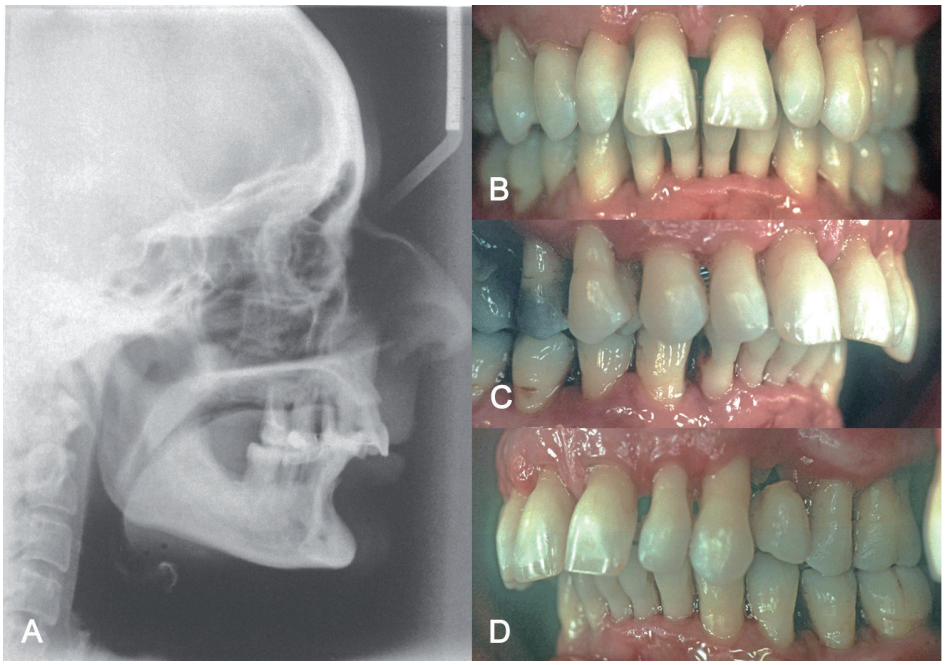

Figure 2. A: Lateral cephalogram after orthodontic treatment; B, C, D: Clinical situation after orthodontic treatment (examination 5).

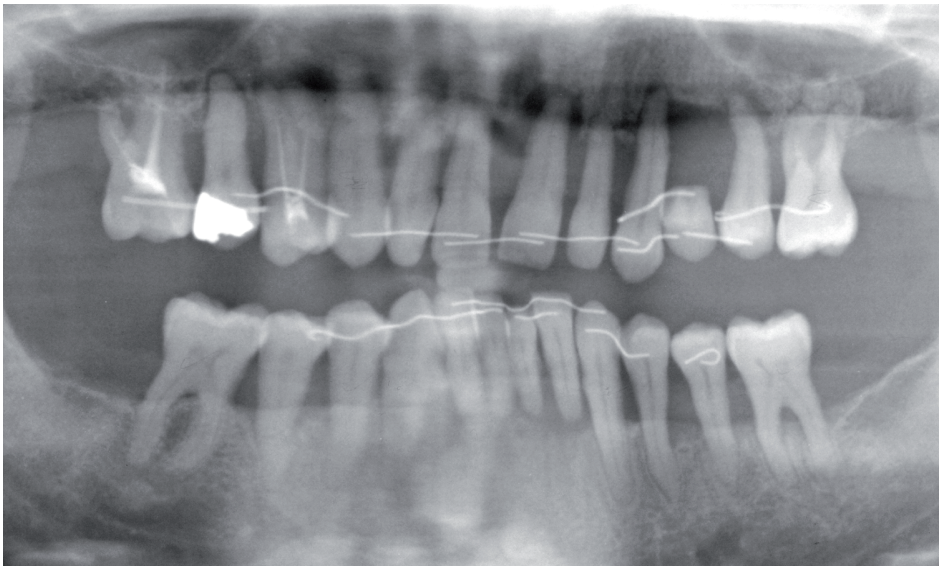

Figure 3. Orthopantomograph 6.7 years after initial examination, before extractions.

size was used (Ormco Co., Orange, CA, USA) (Roth, 1976; 1987) along with the segmented arch technology (Burstone, 1982; Manhartsberger *et al.,* 1989). For the segment formation, a 0.017 x 0.025 inch stainless steel arch (Ormco Co.) was used and the intrusion springs were curved out of a 0.016 x 0.022 inch TMA arch (Ormco Co.). The torque for setting up the lateral tooth segments totaled approx. 15-24 Newton/mm (N/mm); the intrusion force in the front tooth segments was 0.5 to 0.8 N. Fixed orthodontic appliances were placed to level and align the posterior maxillary and mandibular teeth. Simultaneously, the anterior maxillary teeth were progressively intruded and retruded until the interdental spaces were closed. All teeth were stabilized with single arch wires at 96 weeks of treatment; 5 months after this, all orthodontic appliances were removed and teeth #20 to 29 were splinted *(Figure 2).*

At E5 orthodontic treatment was completed. The subgingival plaque analysis showed no presence of periodontal pathogens, and the patient was enrolled in supportive periodontal therapy (SPT, *Table 1*).

Supportive periodontal therapy

Oral hygiene recall sessions were scheduled once a week between E1 and E3, once every three weeks between E3 and E4, and once every two months from E4 to E7 (during and after the orthodontic treatment). During recall sessions, the patient received oral hygiene instructions, supragingival scaling and polishing of all teeth and subgingival irrigations with chlorhexidine digluconate solution 0.1% (Chlorhexamed Fluid, GlaxoSmithKline). CAL, BOP, and PI were recorded and subgingival plaque samples were taken at E1-7. After E7 the patient cancelled all further appointments due to her relocation.

Data analysis

Measurements for CAL at each examination are presented as mean ± SD and range (*Table 1*). Bleeding on probing and PI were calculated as the percentage of sites with bleeding on probing or with plaque accumulation, respectively (*Table 1*). From E2-7, tooth sites were also grouped according to CAL changes (gain, loss, or no change) compared with E1 and the previous examination, and CAL, BOP, and PI outcomes were compared.

In this report, the tooth sites of only one patient were the statistical units. Standard statistical analysis to investigate significant differences is therefore not possible. Cohen's d effect sizes (ES) were calculated to validate the observed CAL gains and to estimate differences in CAL between maxilla and mandible at E1, E4 and E7 and between E1 and E4, E1 and E7, and E4 and E7, separated by jaw and as totals. Cohen's d was also calculated to estimate the CAL difference at E1, E4 and E7 between single- and multi-rooted teeth for the maxilla and mandible. Furthermore, Cohen's d was calculated to evaluate CAL changes for these groups of teeth between E1 and E4, E1 and E7, and E4 and E7. ES was defined as small (d = 0.2), medium (d = 0.5) or large (d = 0.8). Commercial software was used for all statistical analyses (SPSS for Windows, 16/2007, SPSS Inc., Chicago, IL, USA).

Results

This case study follows the treatment and maintenance of a patient with GAgP over 6.7 years. During the study period, no adverse reactions to prescribed medication or local anesthetics were observed. Stability of the clinical situation was obtained at the end of the initial treatment phase and was maintained during supportive periodontal therapy (E1-E7).

One tooth (#1) was extracted at the initial examination and a second tooth (#12) was extracted during the orthodontic treatment phase (E1-E4, number of teeth = 25, E5-E7, N = 24).

The patient participated in supportive periodontal therapy for 194 weeks and thereafter decided to discontinue the therapy. Twenty-four months later a relapse of GAgP was diagnosed and all teeth were extracted (*Figure 3, Table 1*).

The total duration of periodontal and orthodontic treatment (IE-E5) was 32.2 months. The period of supportive periodontal therapy (E4-E7) was 72.5 months. Treatment from E1-E4 resulted in the eradication of all periodontal pathogens (*Table 1*). During the time between E4 and E5, orthodontic treatment was performed and no relapse of GAgP was observed (*Table 1*). E8 occurred 24 months after E7 and showed recurrence of GAgP and tooth loss (*Table 1*, *Figure 3)*. From E1-E7, a nearly linear CAL gain was observed from 10.42 mm to 8.16 mm (*Tables 1 and 3, Figures 4 and 5*). At E4-E7, CAL measurements were lower than at E1 and/or the immediately preceding examination (*Tables 1 and 3*). This pattern was reversed at E8.

Bleeding on probing decreased from 79.17% (E1) to 18.75% (E5). Thereafter, BOP increased, from 36.36% (E7) to 100% (E8). The PI followed a pattern similar to BOP (65.63% at E1, 27.08% at E6). The PI then increased to 38.64% (E7) and to 69.32% at E8 (*Tables 1 and 3*).

Sites were grouped according to CAL gain or loss over the total observation time and records at each examination are presented as differences from E1 and the previous examination (*Table 2*). Sites showing CAL loss had the lowest differences (e.g., highest CAL measurements) in comparison to the previous examination and E1 at almost all time points (*Table 2*). Further, at sites with CAL loss, BOP and PI varied from visit to visit but had generally decreased by E7. Sites showing CAL gain had less inter-visit variation in BOP and PI, but showed declines at E7 similar to those at sites showing CAL loss. In contrast, sites showing no change in CAL showed stable to increased BOP and PI outcomes. For all groups, the lowest BOP and PI were observed between E4-E6 (*Table 2*).

Analysis of CAL by location and type demonstrates higher CAL measurements over time for all maxillary teeth than for mandibular teeth (*Table 3*). CAL measurements were higher for multi-rooted teeth than for single-rooted teeth in both the maxilla and mandible (*Table 3*).

A comparison of CAL outcomes by use of Cohen's d at E1 and E4 showed a small ES (d = 0.4) for all teeth and for maxillary teeth, and a medium ES (d = 0.5) for mandibular teeth. Large ES were found between E1 and E7 (all teeth: d = 1.3; maxilla: d = 1.7; mandible: d = 1.3). Comparing E4 and E7, the mandibular teeth and all teeth show a medium ES (mandible: d = 0.6; all teeth: d = 0.7), and the maxillary teeth show a large ES (d = 0.9, *Figure 5*).

The patient's teeth were also grouped into single- and multi-rooted categories and CAL outcomes were compared by use of Cohen's d (*Figure 5*). The maxillary single-rooted teeth had a medium ES (d = 0.7) between E1 and E4, and a large ES between E1 and E7 (d = 1.5) as well as between E4 and E7 (d = 0.8). The maxillary multi-rooted teeth had a medium ES (d = 0.2) between E1 and E4, and large ES between E1 and E7 (d = 1.8) as well as between E4 and E7 (d = 1.1). Mandibular single-rooted teeth had a small ES (d = 0.4) between E1 and E4, a large ES (d = 1.0) between E1 and E7 and a medium ES (d = 0.5) between E4 and E7. Mandibular multi-rooted teeth had a medium ES (d = 0.5) between E1 and E4, a large ES (d = 1.8) between E1 and E7 and a medium ES (d = 0.7) between E4 and E7.

Comparison among all single- and multi-rooted teeth based on Cohen's d showed that the maxilla at E1 had a medium negative ES (CAL loss, d = -0.6) and the mandible had a small negative ES (d = -0.4). The same results were found for the mandible and maxilla at E4. At E7, a small positive ES (CAL gain) is shown for both the maxilla (d = 0.2) and the mandible (d = 0.1).

Discussion

In the presented case, a patient with generalized aggressive periodontitis was treated by scaling and root planing and systematic administration of amoxicillin and metronidazole three times over a period of 8.2 months. After the initial treatment phase, inflammation-free periodontal tissues were maintained for a period of 72.5 months. During this time, orthodontic therapy was performed despite the severe attachment and bone loss.

During the entire course of treatment and maintenance (80.7 months, or 6.7 yrs), intensive oral hygiene was practiced and regular control of the presence and/or recolonization of the subgingival plaque by periodontal pathogens was performed. The time from the patient's cessation of periodontal maintenance and supportive therapy to the loss of all teeth was 24 months.

In a recent study of prognostic factors in the treatment of GAgP a wide range of variation in initial outcomes following non-surgical treatment was reported (Hughes *et al.,* 2006). In this study it was concluded that clinical parameters such as plaque, bleeding on probing and initial pocket depth were poor predictors of treatment outcome at both the site-specific and the patient levels. In the case presented here, PI outcomes did not necessarily indicate insufficient oral hygiene. A major disadvantage of this index lies in the fact that, after discoloration, any visible staining is recorded as a positive count irrespective of the amount of plaque present. The use of a more differentiated index would

have been better, but was not possible due to limitations of patient treatment in a private practice setting.

A microbiological analysis representing the subgingival microflora of the entire oral cavity is relevant for adjunctive systemic antibiotic treatment of GAgP (Haffajee *et al.,* 2003; Buchmann *et al.*, 2002; Herrera *et al.*, 2002; Purucker *et al.*, 2001; van Winkelhoff *et al.*, 1992). Because of economic reasons, analysis of pooled plaque sampled from several sites was used in this study (Krigar *et al.*, 2007).

It is accepted that the use of MTNZ + AMO is advantageous in rapidly lowering periodontal pathogen counts when accompanied by SRP (Feres *et al.,* 2001; Winkel *et al.,* 2001). In a recently published study, the administration of MTNZ + AMO was argued to be the only adjunctive treatment that resulted in statistically significant reductions in levels of *A. actinomycetemcomitans, P. gingivalis, T. denticola* and *T. forsythia* (Xajigeorgiou *et al.*, 2006). Although MTNZ + AMO appears to be an effective choice when *A. actinomycetemcomitans* is involved, a 12-month study by Flemming *et al.* (1998) has shown persistence of *P. gingivalis*. In another recent study, the timing of systemic adjuvant MTNZ + AMO administration has been investigated (Kaner *et al.,* 2007). It was found that the administration of MTNZ + AMO immediately after SRP produced significant improvements in all clinical periodontal parameters and provided more attachment gain. There is no direct evidence to recommend specific protocol for the use of adjunctive systemic antimicrobials with non-surgical mechanical debridement. However, indirect evidence suggests that the antibiotic uptake should start on the day of debridement completion; debridement should be completed within a short time (Herrera *et al.,* 2008).

The results of the present case agree with the above-mentioned studies and demonstrate the efficacy of combined SRP and MTNZ + AMO treatment in cases of GAgP.

Table 2. Attachment changes (mm ± SD) compared to previous examination or examination 1.

	Examinations					
Grouped Sites	2 ‡	3 ‡	4 ‡	5 ‡	6 ‡	7 §
CAL loss (n‡ = 16, n§ = 8)						
*X ± SD (min, max)	-1.1 ± 0.9 (-2.5 - 0.5)	-0.1 ± 0.3 (-0.5 - 0)	-0.3 ± 0.6 (-1 - 05)	0.3 ± 0.5 (0, 1)	0.0 ± 0.0 (0, 0)	0.8 ± 0.4 (0.5 - 1.0)
*%BOP (MD)	-12.5	-25	0	-25	0	12.5
*%PI (MD)	0	0	0	0	0	12.5
†X ± SD (min, max)	-1.1 ± 0.9 (-2.5 - 0.5)	-1.3 ± 0.9 (-2.5 - 0.5)	-1.5 ± 0.6 (-2 - -1)	-1.3 ± 0.5 (-2 - -1)	-1.3 ± 0.5 (-2 - -1)	-0.8 ± 0.4 (-1 - -0.5)
†%BOP (MD)	-12.5	-37.5	-37.5	-50	-50	-37.5
†%PI (MD)	0	-12.5	-12.5	-12.5	-12.5	12.5
CAL gain (n = 76)						
*X ± SD (min, max)	0.5 ± 0.8 (-1.5 - 2)	0.0 (0-0)	0.8 ± 0.3 (0.5 - 1.5)	0.4 ± 0.4 (0 - 1)	0.0 ± 0.0 (0 - 0)	0.3 ± 0.4 (0 - 1)
*%BOP (MD)	-50	0	0	0	0	0
*%PI (MD)	-25	0	0	0	0	0
†X ± SD (min, max)	0.5 ± 0.8 (-1.5 - 2)	0.5 ± 0.8 (-1.5 - 2)	0.3 ± 0.8 (-0.5 - 2.5)	1.7 ± 0.7 (0 - 3)	1.7 ± 0.7 (0 - 3)	2.0 ± 0.8 (0.5 - 3.5)
†%BOP (MD)	-50	-50	-50	-75	-50	-50
†%PI (MD)	-25	-25	-25	-50	-50	-25
No change (n = 4)						
*CAL	-1.5	0.0	0.5	0.0	0.0	1.0
*%BOP	0	-50	0	0	0	0
%PI*	0	0	0	0	0	0
†CAL	-1.5	-1.5	-1.0	-1.0	-1.0	0.0
†%BOP,	0	-50	-50	-50	-50	-50
†PI	0	0	0	0	0	0

* vs. previous examination; † vs. examination 1; ‡ 25 teeth (100 sites) present; §24 teeth (96 sites) present; MD, median; CAL, clinical attachment level; BOP, bleeding on probing; PI, plaque index

Table 3. Clinical attachment level (CAL) measurements. Teeth grouped by form.

		EXAMINATIONS						
X ± SD, median (min-max)	Tooth Group	1	2	3	4	5	6	7
Maxilla								
	Molars	10.5 ± 2.1, 10.5 (9-12)	9.8 ± 2.5, 9.8 (8-11.5)	9.8 ± 2.5, 9.8 (8-11.5)	8.8 ± 2.5, 8.8 (7-10.5)	8.5 ± 2.1, 8.5 (7-10)	8.5 ± 2.1, 8.5 (7-10)	8.3 ± 1.8, 8.3 (7-9.5)
	Premolars	11.5 ± 1.3, 11.5 (10-13)	11.5 ± 1.8, 11.5 (9.5-13.5)	11.6 ± 1.9, 11.8 (9.5-13.5)	11.3 ± 2.6, 11.0 (9-14)	10.8 ± 3.1, 10.8 (8-14)	10.8 ± 3.1, 10.8 (8-14)	7.8 ± 0.4, 7.8 (7.5-8)
	Incisors + Canines	10.7 ± 1.8, 10.5 (8-13)	10.2 ± 1.8, 10.0 (8-12.5)	10.2 ± 1.8, 10.0 (8-12.5)	9.8 ± 1.7, 10.0 (7.5-12)	9.3 ± 1.5, 10.0 (7-11)	9.3 ± 1.5, 10.0 (7-11)	8.8 ± 1.2, 9.0 (7-10)
	Single root	10.6 ± 1.5, 10.5 (8-13)	10.1 ± 1.5, 10.0 (8-12.5)	10.1 ± 1.5, 10.0 (8-12.5)	9.6 ± 1.5, 9.5 (7.5-12)	9.1 ± 1.4, 9.3 (7-11)	9.1 ± 1.4, 9.3 (7-11)	8.6 ± 1.1, 8.5 (7-10)
	Multiple roots	11.5 ± 1.7, 12.0 (9-13)	11.4 ± 2.4, 12.0 (8-13.5)	11.5 ± 2.5, 12.3 (8-13.5)	11.1 ± 3.1, 11.8 (7-14)	11.0 ± 3.2, 11.5 (7-14)	11.0 ± 3.2, 11.5 (7-14)	8.3 ± 1.8, 8.3 (7-9.5)
	Total	10.9 ± 1.6, 11.0 (8-13)	10.5 ± 1.8, 10.8 (8-13.5)	10.6 ± 1.9, 10.8 (8-13.5)	10.1 ± 2.2, 10.0 (7-14)	9.7 ± 2.2, 10.0 (7-14)	9.7 ± 2.2, 10.0 (7-14)	8.5 ± 1.2, 8.5 (7-10)
Mandible								
	Molars	9.5 ± 0.7, 9.5 (9-10)	9.3 ± 1.8, 9.3 (8-10.5)	9.3 ± 1.8, 9.3 (8-10.5)	8.8 ± 1.8, 8.8 (7.5-10)	8.0 ± 1.4, 8.0 (7-9)	8.0 ± 1.4, 8.0 (7-9)	7.8 ± 1.1, 7.8 (7-8.5)
	Premolars	8.8 ± 0.5, 9.0 (8-9)	9.3 ± 1.4, 9.3 (8-10.5)	9.3 ± 1.4, 9.3 (8-10.5)	8.5 ± 1.5, 8.5 (7-10)	8.0 ± 1.2, 8.0 (7-9)	8.0 ± 1.2, 8.0 (7-9)	7.8 ± 0.9, 7.8 (7-8.5)
	Incisors + Canines	9.3 ± 1.8, 8.5 (8-12)	9.3 ± 1.4, 9.3 (8-10.5)	9.3 ± 1.4, 9.3 (8-10.5)	8.5 ± 1.5, 8.5 (7-10)	8.3 ± 1.5, 8.0 (7-10)	8.3 ± 1.5, 8.0 (7-10)	8.0 ± 1.1, 8.0 (7-9)
	Single root	9.1 ± 1.4, 9.0 (8-12)	9.3 ± 1.3, 9.3 (8-10.5)	9.3 ± 1.3, 9.3 (8-10.5)	8.5 ± 1.4, 8.5 (7-10)	8.2 ± 1.3, 8.0 (7-10)	8.2 ± 1.3, 8.0 (7-10)	7.9 ± 1.0, 7.8 (7-9)
	Multiple roots	9.5 ± 0.7, 9.5 (9-10)	9.3 ± 1.8, 9.3 (8-10.5)	9.3 ± 1.8, 9.3 (8-10.5)	8.8 ± 1.8, 8.8 (7.5-10.0)	8.0 ± 1.4, 8.0 (7-9)	8.0 ± 1.4, 8.0 (7-9)	7.8 ± 1.1, 7.8 (7-8.5)
	Total	9.2 ± 1.3, 9.0 (8-12)	9.3 ± 1.3, 9.3 (8-10.5)	9.3 ± 1.3, 9.3 (8-10.5)	8.5 ± 1.4, 8.5 (7-10)	8.2 ± 1.3, 8.0 (7-10)	8.2 ± 1.3, 8.0 (7-10)	7.8 ± 0.9, 7.8 (7-9)
Maxilla & Mandible								
	Molars	10.0 ± 1.4, 9.5 (9-12)	9.5 ± 1.8, 9.3 (8-11.5)	9.5 ± 1.8, 9.3 (8-11.5)	8.8 ± 1.8, 8.8 (7-10.5)	8.3 ± 1.5, 8.0 (7-10)	8.3 ± 1.5, 8.0 (7-10)	8.0 ± 1.2, 7.8 (7-9.5)
	Premolars	10.1 ± 1.7, 9.5 (8-13)	10.4 ± 1.9, 10.5 (8-13.5)	10.4 ± 2.0, 10.5 (8-13.5)	9.9 ± 2.5, 9.3 (7-14)	9.4 ± 2.6, 8.8 (7-14)	9.4 ± 2.6, 8.8 (7-14)	7.8 ± 0.7, 7.8 (7-8.5)
	Incisors + Canines	10.0 ± 1.8, 10.0 (8-13)	9.7 ± 1.6, 9.8 (8-12.5)	9.7 ± 1.6, 9.8 (8-12.5)	9.1 ± 1.7, 9.8 (7-12)	8.8 ± 1.5, 9.5 (7-11)	8.8 ± 1.5, 9.5 (7-11)	8.4 ± 1.2, 9.0 (7-10)
	Single root	9.8 ± 1.6, 9.5 (8-13)	9.6 ± 1.4, 10.0 (8-12.5)	9.6 ± 1.4, 10.0 (8-12.5)	9.0 ± 1.5, 9.3 (7-12)	8.6 ± 1.4, 8.8 (7-11)	8.6 ± 1.4, 8.8 (7-11)	8.2 ± 1.1, 8.3 (7-10)
	Multiple roots	10.8 ± 1.7, 11.0 (9-13)	10.7 ± 2.3, 11.0 (8-13.5)	10.8 ± 2.4, 11.0 (8-13.5)	10.3 ± 2.8, 10.3 (7-14)	10.0 ± 3.0, 9.5 (7-14)	10.0 ± 3.0, 9.5 (7-14)	8.0 ± 1.2, 7.8 (7-9.5)
	Total	10.0 ± 1.7, 10.0 (8-13)	9.9 ± 1.7, 10.5 (8-13.5)	9.9 ± 1.7, 10.5 (8-13.5)	9.3 ± 1.9, 9.5 (7-14)	8.9 ± 1.9, 9.0 (7-14)	8.9 ± 1.9, 9.0 (7-14)	8.2 ± 1.1, 8.3 (7-10)

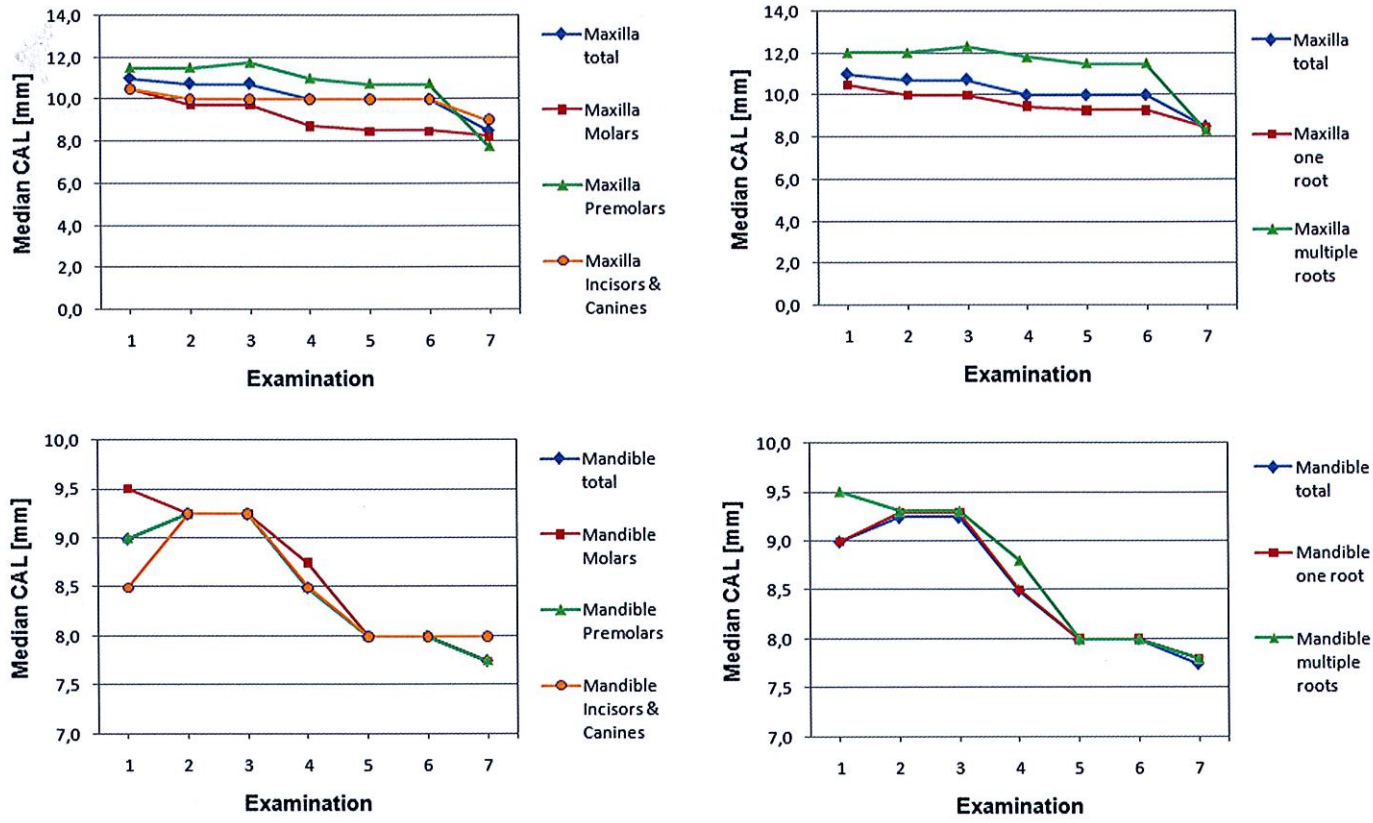

Figure 4. Median clinical attachment loss. A: Overall; B: In the mandibular and maxillary teeth, by type of tooth in the maxilla (C) and mandible (D); by number of roots in the maxilla (E) and mandible (F).

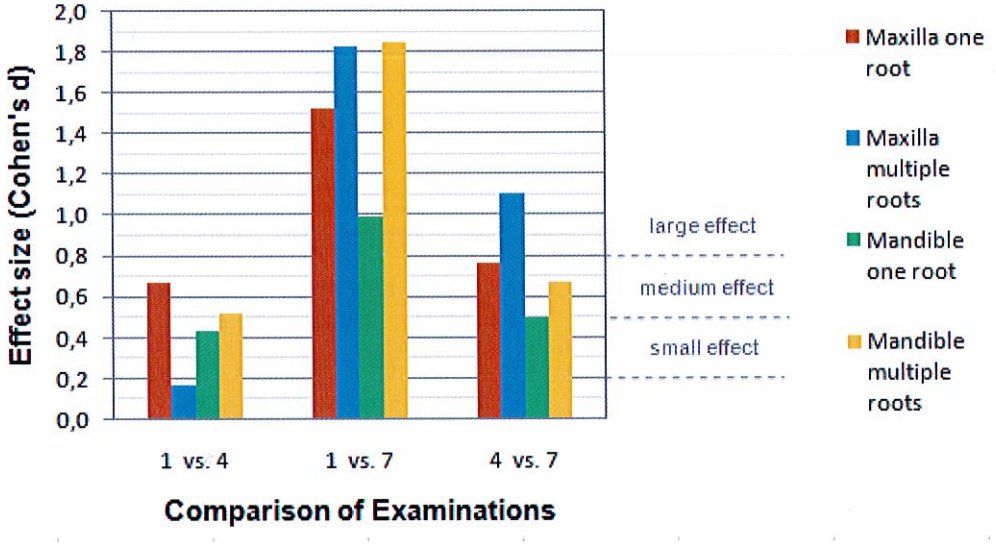

Figure 5. Effect size - Cohen´s d. Teeth were grouped into single- and multi-rooted categories and clinical attachment level outcomes were compared.

In this study, some sites showed attachment loss over the duration of treatment and maintenance. These sites initially showed the most extensive periodontal tissue destruction and disease progression could not be stopped. This finding is in agreement with previously published data (Kamma and Baehni, 2003). Sites showing CAL gain had less inter-visit variation in BOP and PI. In contrast, sites showing no change in CAL had stable to increased BOP and PI outcomes. In both cases, the lowest BOP and PI were observed after active periodontal treatment (E4) and after 72.5 months of maintenance (E7); however, higher attachment gain was observed in single-rooted teeth. At sites with CAL loss, BOP and PI varied from visit to visit but generally decreased by E7. Over the study duration all maxillary teeth, single- and multi-rooted, showed higher CAL measurements than mandibular teeth. This could be due to the anatomic characteristics of the maxilla and to its greater spongiosa component. In the case of multi-rooted teeth, it could be reasoned that their morphology creates niches more difficult to instrument for the eradication of plaque.

In the case presented, no new modalities for the treatment of GAgP were used. The aim of this case report is to demonstrate the possibility of maintenance of a stable periodontal condition as well as the ability of the periodontal tissues to tolerate controlled tooth movements, as long as the periodontal infection is controlled.

The frequency and type of combined mechanical and antibiotic treatment was not based on a fixed schedule, but was developed on the basis of clinical and laboratory findings. The obtained results agree with those reported by Sigusch *et al.* (2001) who found that adjunctive administration of antibiotics with SRP leads to significantly greater CAL gain and pocket depth reduction compared to SRP alone.

Conclusions

The results of the present study support the hypothesis that a consistent resolution of GAgP could be achieved utilizing a combined mechanical and antimicrobial treatment followed by periodontal maintenance and microbiological monitoring.

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