Root abnormalities and nonsurgical management of generalized aggressive periodontitis

Da Lü1,2), Huanxin Meng1), Li Xu1), Xian’e Wang1), Li Zhang1), and Yu Tian1)

1)Department of Periodontology, Peking University School and Hospital of Stomatology, Beijing, P. R. China
2)Department of Stomatology, Peking University Shenzhen Hospital, Shenzhen, P. R. China

Abstract: To investigate long-term nonsurgical treatment outcomes in patients with generalized aggressive periodontitis (GAgP) and the impact of root abnormalities (RAs) and other patient-level factors in relation to GAgP progression. Patients (n = 64) from a GAgP cohort who completed active nonsurgical periodontal treatment and consented to re-evaluation after 3 to 11 (mean 5.3) years, were enrolled. RAs were identified using radiographs. Periodontal parameters (e.g., probing depths [PDs], and tooth loss [TL]) were investigated. Multivariate analysis was performed to identify factors contributing to TL and bone level alteration (∆BL). After treatment, the mean number of sites with PDs > 5 mm decreased from 54.3 to 17.2. Annual TL was 0.11/patient. Twenty-one patients (32.8%) had >4 teeth with root abnormalities (RA-teeth) and exhibited a higher risk for TL (univariate odds ration [OR] = 3.52, multivariate logistic OR = 6.57). Factors correlated to ∆BL were sites with residual PD > 5 mm (β = −0.400) and observation time (β = −0.210). Nonsurgical treatment provides beneficial outcomes in GAgP patients. Higher incidence of RAs and high prevalence of residual deep pockets have a negative impact on long-term outcomes. Practical implications: in cases of GAgP with residual deep pockets and high incidence of RAs, clinicians must emphasize that long-term outcomes of nonsurgical treatment may be compromised.

Keywords: aggressive periodontitis; risk factors; tooth abnormalities; nonsurgical periodontal therapy; tooth loss.

Introduction

Periodontitis is an inflammatory disease primarily caused by microorganisms in the bacterial plaque (1-3). Aggressive periodontitis (AgP) is recognized by rapid bone loss, which can further be divided into localized or generalized (4). The onset of AgP is linked to several risk factors and host susceptibilities (5,6). In periodontal practice, factors pertaining to the progression of AgP have been well described. Cumulative evidence highlights that factors such as age, smoking, prevalence of gingival bleeding, and interleukin-1 (IL-1) polymorphism have impeded the long-term prognosis of AgP patients (7-9). In order to improve the prognosis of AgP cases, intervention of risk factors and modified treatment plans based on risk assessment have been attempted (10-12).

Morphological abnormalities of tooth roots are common (13). Teeth with root abnormalities (RA-teeth) are often clinically involved in AgP (5). In a recent study (14), a method to identify RA-teeth by using maladjusted crown-root ratio (short-root), cone-root, curved-root, and syncretic-root of molars has been described. The study also indicated that the prevalence of RA was higher in AgP patients (14.3%) than in those with chronic periodontitis (CP) (5.0%) and periodontal healthy individuals (3.7%).

However, a limited number of published articles have indicated an association between RAs and prognosis of periodontitis. An early investigation (15) described that
an unfavorable crown-root ratio and root formation are related to poorer prognosis of periodontitis patients. Another study (16) suggested that a poor crown-root ratio (<1) is not related to tooth loss (TL). Further investigation is needed to determine whether a well-defined RA is a local risk factor for AgP progression.

The progression of periodontitis is a multifactorial phenomenon. Strong influential factors such as smoking, diabetes, and initial severe bone defects may veil the effects of weaker factors (IL-1 polymorphism and plaque index) (17-19). Thus, validation of putative factors should be conducted in restricted subjects with multivariate patterns. The primary aim of the present study was to assess long-term outcomes of generalized aggressive periodontitis (GAgP) treated using nonsurgical approaches. In addition, various factors related to TL and bone level alteration (∆BL) have been investigated, considering that RA is one of the concerned factors.

Materials and Methods

Study population
GAgP patients treated using nonsurgical periodontal approaches during the 1999-2008 period at the Department of Periodontology, Peking University School and Hospital of Stomatology, were recalled from June 2010 to March 2012 and re-evaluated 3 to 11 years after treatment. Patients who had any surgical intervention for periodontal disease were excluded. This program was approved by the Ethics Committee of the Peking University Health Science Center (IRB00001052-08010). All patients were informed of the study and written consent was obtained.

The assessment criteria for GAgP were defined according to the classification proposed by the International Workshop for the Classification of Periodontal Diseases and Conditions (20,21). At baseline, patients fulfilled the following criteria for GAgP; i) at least six teeth with probing depths (PDs) ≥ 5 mm and attachment loss (AL) ≥ 3 mm with radiographic evidence of interproximal bone loss > 33.3%, ii) age 14-36 years and a minimum of 20 teeth present; third molars were not considered. Exclusion criteria were as follows; i) history of periodontal or antimicrobial therapy within the previous six months, ii) history of orthodontic therapy, iii) history of systemic diseases, pregnancy, or use of medications known to affect the periodontium.

Experimental design and nonsurgical therapy
At baseline therapy (T0), patients filled out required questionnaires that involved smoking status, general conditions, and medical and family history. Periodontal charting was recorded. Full-mouth periapical radiographs were taken using the bisecting angle technique. An experienced dental technician was consulted to assure consistent quality of the radiographs. Any radiograph with an inappropriate projection was re-taken at this stage. Nonsurgical periodontal treatment was initiated with oral health instructions (OHIs), followed by scaling and root planning (SRP) under local anesthesia. Patients were prescribed antibiotics (0.2 g of metronidazole and 0.5 g of amoxicillin, thrice a day for seven days) after SRP.

The periodontal charting record was updated at the first recall (2 to 6 months following nonsurgical therapy [T1]). Patients who were surgically managed because of residual deep pockets were excluded at this stage. For patients who remained in the study, supportive periodontal therapy (SPT) was suggested for two to four sessions/year. Certain patients with malocclusion received orthodontic treatment under strict SPT.

At the final recall (T2), at least three years after T1, patients were re-assessed by periodontal examination and full-mouth radiographs. Follow-up questionnaires containing the aforementioned items were recorded.

Clinical evaluation
Periodontal examination was performed at T0, T1, and T2 stages by three trained examiners (M.H., X.L., and Z.L.) using a Williams periodontal probe. For each available tooth, PD and AL were measured at six sites (mesial, distal, and middle sites of the buccal and lingual sides). AL was measured as the distance from the bottom of the pocket to the cementoenamel junction (CEJ). The highest bleeding index (BI) values (22) of buccal and lingual
surfaces were recorded. Third molars were excluded. TL during SPT was defined as SPT-TL.

Relative bone height
All scanned radiographs were assessed for bone height. Relative bone heights (RBHs) were calculated as the ratio of residual bone height of the interproximal site and full root length (23). ∆BL for each patient was calculated by subtracting average RBH in T0 from that in T2. 

$$\Delta BL = RBH_{T2} - RBH_{T0}$$

Root abnormality
Parameters of nonmolars measured on radiographs included the following (14):

Crown-root ratio (Fig. 1A); the tooth axis length was divided along the CEJ line; the ratio of these two parts was calculated.

Parameter of root width (Fig. 1B); median point of the line that joins the mesial (or distal) CEJ point and apex, was defined as M (or D). Line MD extended till the edge of root, forming a line segment M′D′. Parameter of root width = (M′D′ – MD) / 2.

Angle of the root (Fig. 1H); an angle formed by the long axis and apical third axis.

All measurements were solely performed by a trained examiner (L.D.) using a software (the Geometer’s Sketchpad, version 5.0, USA), and which were confirmed by an expert panel of three experienced periodontists (X.L., T.Y., and L.D.). Teeth with full-crown restorations or interproximal cervical defects that lost inaccurate markers were excluded from RA detection. Four types of RAs were identified (Fig. 1): i) A short-rooted tooth is an incisor or premolar that has a crown-root ratio greater than the “reference value” (Table 1). It was subsequently excluded when the crown was observed to be distorted owing to buccal/lingual malposition; ii) A cone-rooted tooth is an incisor or premolar that has a root width parameter less than the “reference value” (Table 1). It was subsequently excluded when it was observed to be either a torsiversion or a small-sized tooth that had a root with a maintained width from the cervical third to the middle third; iii) A curved-rooted tooth is an incisor or premolar with an angle of root >15° or with a root that exhibited an evident S-shape; iv) Syncretic-rooted molars have obvious triangular-shaped merged roots.

Reference values for each specified type have been previously determined (14). Any tooth fulfilling more than one RA criteria, was named according to the first one in the sequence (e.g., teeth with short, curved roots should be named short-rooted).

Data analysis
Data were processed using SPSS (version 13.0, Chicago IL, USA). Mean and standard deviation (SD) of parameters were calculated. Changes in periodontal parameters were analyzed using paired-sample t-test. Intergroup differences stratified by various profiles were evaluated using the Mann-Whitney/Kruskal-Wallis test for data with non-normal distribution (e.g., annual TL/patient), or the x²-test for dichotomous data (e.g., incidence of TL). Odds ratio (OR) and 95% confidence intervals (CIs) were analyzed using a univariate model reflecting the risk of TL ($P < 0.05$ considered statistically significant).

Logistic regression analyses were conducted to further quantify the association of each related factor with SPT-TL at the patient level. Categorical variables

---

**Table 1** Reference values and radiographic metric results of short- or cone-rooted incisors and premolars

|                        | n | Crown-root ratio | Parameter of root width (mm) |
|------------------------|---|------------------|-----------------------------|
|                        |   | Reference value | Median (min-max)           | Reference value | Median (min-max) |
| Upper central incisors |   |                 |                            |                |                  |
| Short root             | 18| >0.81           | 0.969 (0.813-1.388)        | <0.67          | 0.65 (0.63-0.66) |
| Cone root              | 4 |                 |                            |                |                  |
| Upper lateral incisors |   |                 |                            |                |                  |
| Short root             | 1 | >0.90           | 1.146 (1.146-1.146)        | <0.50          | 0.45 (0.36-0.49) |
| Cone root              | 7 |                 |                            |                |                  |
| Lower incisors         |   |                 |                            |                |                  |
| Short root             | 2 | >0.80           | 0.811 (0.810-0.811)        | <0.34          | 0.27 (0.13-0.33) |
| Cone root              | 30|                 |                            |                |                  |
| Upper premolars        |   |                 |                            |                |                  |
| Short root             | 32| >0.82           | 0.933 (0.822-1.165)        | <0.39          | 0.26 (0.05-0.38) |
| Cone root              | 32|                 |                            |                |                  |
| Lower premolars        |   |                 |                            |                |                  |
| Short root             | 0 | >0.82           | None                       | <0.42          | 0.30 (0.20-0.41) |
| Cone root              | 23|                 |                            |                |                  |
including gender and orthodontic treatment during SPT (yes/no) were entered. Continuous variables including age, RAs, mean RBH, percentage of BI > 2, sites with PD > 5 mm, and SPT session were switched to categorical data by using a threshold recommended in previous studies (23,24) or median/tertile of the current study population. The final model using the backward method illustrated selected factors \((P < 0.1)\) related to SPT-TL.

Stepwise linear regression was performed to analyze the association of mean ∆BL with the aforementioned parameters (e.g., age, sites with PD > 5 mm in T0, and annual SPT session). If a certain dichotomic parameter was positive or negative, one or zero was assigned. Factors were retained in the final model when \(P < 0.1\). Factors with a positive standardized coefficient (β) were recognized as protective factors for bone healing.

## Results

**Patients**

A total of 158 GAgP patients who joined our program and finished the nonsurgical treatment were recalled; of these 64 patients (19 men and 45 women) who responded and finished re-evaluation were enrolled. One male patient had a history of smoking (period >10 years; dose <20 cigarettes/day). Mean time of observation was 5.3 years (SD, 2.1). Mean age at stage T0 was 26.9 years (SD, 4.9). Seventeen patients (26.6%) complied with SPT at least once a year.

**Root abnormality**

According to assessment of 1,757 teeth (none of these had full-crown restorations), 229 teeth (13.0%) fell within the definition of RA; of these, 53 (23.1%) were short-rooted, 96 (41.9%) were cone-rooted, 54 (23.6%) were curved-rooted teeth, and 26 (11.4%) were syncretic-rooted molars. The radiographic metric assessment of incisors or premolars with RAs is illustrated in Table 1. At patient level, RA-teeth ranged from 0 to 15 (median, 4); 43 patients had ≤4 RA-teeth.

**Therapeutic outcome**

Clinical parameters for T0, T1, and T2 stages are presented in Table 2. Before treatment (T0), a high prevalence of sites with BI > 2 (mean ± SD, 89.1 ± 22.3%) and with PD > 5 mm (mean ± SD, 54.3 ± 34.2 sites) were recorded. After periodontal therapy (T1), considerable improvement was observed (BI > 2: 43.3 ± 33.4%, \(P < 0.001\); PD > 5 mm sites: 13.5 ± 20.3, \(P < 0.001\)), although these parameters revealed ascent after several years of observation (T2); mean AL manifested a similar trend (T0: 4.1 ± 1.6 mm; T1: 3.2 ± 1.9 mm; and T2: 3.8 ± 1.9 mm). Mean RBH increased significantly from 0.64 ± 0.11 (T0) to 0.66 ± 0.12 (T2) \((P = 0.002)\).

**Tooth loss**

During SPT, 38 teeth in 15 patients were lost, either due to terminal loss of periodontal support or increased mobility. Seven (18.4%) of the lost teeth were RA-teeth. SPT-TL ranged 0-9, with an average of 0.11 TL/patient/year.

Table 3 describes the risk of SPT-TL when certain characteristics were positive in the univariate analysis. Patients with >4 RA-teeth had a higher incidence of SPT-TL \((OR = 3.52, 95\% CI: 1.06-11.76, P = 0.035)\) and annual SPT-TL \((n ± SD: 0.16 ± 0.26 vs. 0.09 ± 0.27, P = 0.041)\). The risk of SPT-TL was higher when the mean RBH in T0 was lower or when residual sites with PD > 5 mm were >9 or when sites with BI > 2 presented >25%.

In the multivariate logistic regression analysis (Table 4), the final model revealed that more RA-teeth (>4, \(OR = 6.57, P = 0.026\)) and more residual sites with BI > 5 mm (>25%, \(OR = 13.62, P = 0.012\)) significantly increased the risk of any SPT-TL, while a higher baseline RBH \((OR = 0.16 per grade, P = 0.005)\) and orthodontic treatment during SPT \((OR = 0.08, P = 0.103)\) were protective factors against SPT-TL.

**Alteration of bone height**

Univariate linear analysis revealed factors that were significantly associated with ∆BL (T2-T0), which included observation time, annual SPT sessions,
percentage of sites with BI > 2 in T1, and number of sites with PD > 5 mm in T1. According to multivariate linear regression results, these factors included observation time ($\beta = -0.210$, $P = 0.070$) and sites with PD > 5 mm in T1 ($\beta = -0.400$, $P = 0.001$) (Table 5).

### Discussion

The goal of periodontal treatment is to improve periodontal health and thus regain or maintain oral function. Our study emphasized the efficacy of periodontal treatment in terms of inducing noticeable alleviation of inflammation and achieving supplementary periodontal

| Table 3 | Univariate analysis of the incidence of any SPT-TL and annual SPT-TL in terms of different backgrounds or of periodontal characteristics |
|---------|-------------------------------------------------------------------------------------------------------------------|
| Total  | Incidence of any SPT-TL | Annual SPT-TL ($n \pm SD$) |
| Sex    | Patients (%) OR (95% CI) |                              |
| Male   | 19 5 (26.3) 0.80 (0.23-2.76) | 0.14 ± 0.32 |
| Female | 45 10 (22.2) | 0.10 ± 0.24 |
| Age in T0 | ≤25 years | >25 years |
| RA-teeth ≤4 | 44 7 (15.9) | 0.09 ± 0.27 |
| >4     | 20 8 (40.0) | 3.52 (1.06-11.76)* |
| Mean relative bone height (T0) | >0.69 | 0.03 ± 0.14 |
| 0.60-0.69 | 20 4 (20) | 0.08 ± 0.20 |
| 0.60     | 22 10 (45.5) | 17.50 (2.82-108.54) |
| % of sites with BI > 2 (T1) ≤25 | 24 2 (8.3) | 0.04 ± 0.13 |
| >25     | 40 13 (32.5) | 5.30 (1.08-26.01)* |
| n of sites with PD > 5 mm (T1) ≤9 | 41 6 (14.6) | 0.04 ± 0.11 |
| >9     | 23 9 (39.1) | 3.75 (1.12-12.51)* |
| Orthodontic treatment during SPT | No | 14 (24.6) | 0.12 ± 0.28 |
| Yes    | 7 1 (14.3) | 0.02 ± 0.06 |
| SPT session/year ≥1 | 17 2 (11.8) | Reference |
| 0-1 (0,1 excluded) | 21 6 (28.6) | 3.00 (0.52-17.32) |
| 0 26 7 (26.9) | 2.76 (0.50-15.30) |

*Significant difference in the incidence of TL between groups ($x^2$-test, $P < 0.05$); **Negative correlation between the incidence of TL and mean relative bone height (T0) grade (trend $x^2$-test, $P = 0.001$); 1 Significance difference in annual TL between groups (Mann-Whitney test, $P < 0.05$); 2 Significant difference in annual TL among groups (Kruskal-Wallis test, $P = 0.008$); TL: tooth loss; RA: root abnormality; SPT: supportive periodontal therapy; SD: standard deviation; OR: odds ratio; CI: confidence interval.

| Table 4 | Logistic regression models for any tooth loss during SPT with putative risk factors: initial model (enter method) and final model (backward method) |
|---------|-------------------------------------------------------------------------------------------------------------------|
| Enter  | Final model |
| Gender (male vs. female) | 0.72 0.737 |
| Age in T0 (>25 years vs. ≤25 years) | 0.99 0.993 |
| RA-teeth (≥4 vs. ≤4) | 11.96 0.031 |
| Mean RBH in T0 (per grade*) | 0.077 0.006 |
| BI > 2 sites in T1 (>25% vs. ≤25%) | 15.05 0.076 |
| PD > 5 mm sites in T1 (>9 vs. ≤6) | 0.68 0.754 |
| Orthodontic treated during SPT | 0.01 0.079 |
| SPT session/year (vs. ≥1) ≥1 | 0.345 |
| 0-1 (0,1 excluded) | 7.82 0.158 |
| 0 | 2.34 0.505 |
| Constant | 0.193 |

*Grade 0: <0.60, Grade 1: 0.60-0.69, Grade 2: >0.69. RA: root abnormality; RBH: relative bone height; BI: bleeding index; PD: probing depth; SPT: supportive periodontal therapy; OR: odds ratio; CI: confidence interval.
support in the form of attachment gain (mean ∆AL: 0.9 mm) and bone increment (mean ∆RBH: 0.02). Our results are in agreement with a previous study, which was conducted on 11 patients with juvenile periodontitis; active treatment followed by a 5-year maintenance program resulted in approximately 2 mm of bone increment and approximately 3 mm of decrease in PD on the affected sites (25).

On the other hand, our follow-up results demonstrated recurrence after nonsurgical periodontal therapy compared to periodontal records from T1 to T2. An increasing prevalence of sites with BI > 2 (43.3-53.5%) and PD > 5 mm (mean: 13.5-17.2), additional AL (0.6 mm/teeth/patient), and tooth mortality (0.11 teeth/patient/year), were observed. These findings are similar to those reported in other studies that focused on AgP recurrence. A previous study which included 48 patients with early-onset periodontitis (EOP), reported an AL of 0.03-0.35 mm around the affected teeth after 4 years (26). AgP patients with a well-maintained therapy manifested 2.2% sites with an AL of ≥2 mm (27). The annual TL of treated AgP (or EOP) patients varied from 0.08-0.24 per patient (7,9).

TL has been regarded as a “true end point” relevant to treatment efficacy in several studies (19,28). However, this indicator could be influenced by the decisions of patients or clinicians on tooth extraction strategy, thus partially reflecting the progression of periodontal diseases. ∆BL during radiographic assessment is considered a more sensitive variable that demonstrates recovery of periodontal supporting tissues.

RAs are often observed in patients with periodontitis, particularly AgP (5). Early longitudinal studies underscored that RA was a local risk factor for periodontal breakdown (15). To the best of our knowledge, lucid definition and category of RAs was not introduced until Xu et al. (14) initiated a series of geometric measurements to identify RAs. The available surface area of the root is critical to reinforce the strength of periodontal attachment. Reduced root surface attachment area due to a congenital abnormality or disease process may compromise periodontal support. It is argued by our authors that conditions such as short roots, conical roots, and syncretic roots possess relatively minor surface areas of periodontal ligament compared to normally shaped roots; hence, overloading of such teeth may accelerate the process of periodontal destruction. In addition, curved roots transfer adverse lateral force during physiological functioning (mastication); these lateral forces may lead to localized stress shielding and accelerated breakdown of the periodontal ligament. Mathematically, in experiencing the same amount of bone destruction, short roots lose higher proportions of periodontal support than do long roots. Following the pilot study (14), we attempted to apply previous metrical data on new clinical subjects and explore a standardized method to detect RAs. This process requires prudence and does not solely rely on quantified criteria for two reasons. First, observers need to recognize if the metrical result is distorted because of the inappropriate radiographic projection on malpositioned teeth. Second, the abnormal parameter of the root width could be a result of tooth size variation other than a cone-shaped abnormality. In patient-level analysis, RA-teeth > 4 remained in the final logistic regression model, exhibiting the significant predictive value of TL following nonsurgical treatment (OR = 6.57). This result suggests that number of RAs is a risk indicator for further TL. RAs could be relevant to reduced periodontal support and inappropriate occlusive force, which leads to rapid periodontal breakdown. Further investigation of this risk indicator is warranted.

Periodontal tissue damage is well considered to be initiated by microorganisms present in the dental plaque biofilm (29) and is associated with host inflammatory responses to microbial challenges (30). Several studies have suggested that plaque control and gingival index

| Table 5 Univariate and multivariate linear regression for correlation of bone level alteration (∆BL) and patient-level factors |
|---------------------------------------------------------------|---------------------------------------------------------------|
| Univariate analysis Multivariate regression | Univariate analysis Multivariate regression |
| | β | P value | β | P value |
| Age | −0.161 | 0.205 | −0.210 | 0.070 |
| Observation time | −0.261 | 0.038 | | |
| RA-teeth | 0.070 | 0.580 | | |
| Mean RBH | 0.071 | 0.578 | | |
| Percentage of BI > 2 sites in T1 | −0.349 | 0.005 | −0.400 | 0.001 |
| n of PD > 5 mm sites in T1 | −0.426 | <0.001 | −0.400 | 0.001 |
| SPT session/year | 0.283 | 0.023 | | |
| (constant) | 3.902 | <0.001 | | |
are significantly correlated to marginal bone differences (MBDs) and TL (19,31,32). Since plaque index is associated with supragingival microbiota, and gingival index reflects an unbalanced interaction between host and subgingival pathogens to a certain extent, our study focused more on the BI of affected teeth than on plaque control. Following nonsurgical debridement at T1, a high prevalence of sites with BI ≥ 2 significantly increased the odds of TL and correlated to ABL with a negative effect; although it was not selected in the multivariate linear model because of colinearity with other factors (residual PD ≥ 5 mm sites). Jansson et al. (31) reported similar findings in a prospective study of over 20 years in 513 individuals, wherein the Ainamo-Bay BI at baseline correlated with MBDs (r = 0.17, P < 0.001). Matulienė et al. (32) reported that bleeding on probing increased the odds of TL (OR = 1.9) in a retrospective study.

PD is another critical clinical measurement that indicates the severity of gingival swelling and depth of ecological niches for pathogenesis. As observed in the present study, subjects with a higher prevalence of a residual PD of > 5 mm experienced a higher risk of SPT-TL. PD > 5 mm (sites) remained a significant predictor in multivariate linear regression of ABL, indicating a negative impact on bone healing. It could be explained that incompletely treated sites with residual deep pockets resulted in gingival irritation. In addition, deep pockets are more likely to harbor anaerobic pathogens relevant to host immune defense (33,34). Our findings are in line with certain surveys. For instance, a study with a longer duration (mean, 11.3 years) suggested that the presence of sites with PD ≥ 6 mm significantly contributed to the risk of TL (OR = 1.7) and progression (teeth with ≥ 3 mm of proximal AL) during SPT at patient level. These authors discussed that PD ≥ 6 mm represented an incomplete treatment outcome that required additional therapy (32). Another study conducted on 128 Indonesian inhabitants reported that the number of sites with PD ≥ 5 mm significantly correlated with the amount of AL over 15 years (35).

Several studies have emphasized the efficacy of regular SPT in maintaining long-term positive outcomes (19,36,37). In our study, 17 patients (26.6%), who complied with the annual SPT, experienced lower SPT-TL (0.05 teeth/patient/year) and positive bone healing. These results are similar to those of a previous study conducted on an AgP cohort, wherein regular compliers consisted 28.6% who lost 0.08 teeth/year and irregular ones who lost 0.15 teeth/year (9).

Nevertheless, the present study has certain limitations. For example, inaccurately projected RA-teeth might have escaped detection upon radiographic examination. The long-cone projection technique and cone-beam computed tomography might provide more accurate information on root shape and size. However, such techniques expose patients to a higher dose of unnecessary radiation and elicit ethical concerns. The bisecting angle technique is a popular technique and was favored in the present study owing to its benefits such as ease of manipulation and significantly lower radiation exposure. Teeth restored using either full-crown, interproximal, or cervical restorations lost metric markers and this may lead to an inaccurate evaluation. Such conditions were rarely observed in our study. Moreover, further research is warranted to confirm whether orthodontic treatment under strict SPT is beneficial for patients with AgP.

In conclusion, beneficial outcomes can be achieved using nonsurgical treatment strategies for patients with GAgP. A higher incidence of RAs is associated with an increased risk of further periodontal deterioration and TL. Moreover, a high prevalence of incompletely treated sites in the form of residual deep pockets and gingival inflammation has a negative impact on bone healing.

Acknowledgments
This study was funded by the National Natural Science Foundation of China (30271411, 30471882, and 30973319); the National Key Project of Scientific and Technical Supporting Programs of China (2002AA217091, 2007BAZ18B02); and the Clinical Research Fund, Ministry of Health P. R. China. The authors are grateful for the help of Ms. Song Yi (statistical consultant).

Conflict of interest
The authors declare no conflict of interest with this work.

References
1. Genco RJ (1996) Current view of risk factors for periodontal diseases. J Periodontol 67, 1041-1049.
2. Demmer RT, Papapanou PN (2010) Epidemiologic patterns of chronic and aggressive periodontitis. Periodontol 2000 53, 28-44.
3. Stabholz A, Soskolne WA, Shapira L (2010) Genetic and environmental risk factors for chronic periodontitis and aggressive periodontitis. Periodontol 2000 53, 138-153.
4. Lang N, Bartold PM, Cullian M, Jeffcoat M, Mombelli A, Murakami S et al. (1999) Consensus report: aggressive periodontitis. Ann Periodontol 4, 53.
5. Meng H, Xu L, Li Q, Han J, Zhao Y (2007) Determinants of host susceptibility in aggressive periodontitis. Periodontol 2000 43, 133-159.
6. Meng H, Ren X, Tian Y, Feng X, Xu L, Zhang L et al. (2011) Genetic study of families affected with aggressive periodontitis. Periodontol 2000 56, 87-101.
7. Kamma JJ, Buehni PC (2003) Five-year maintenance follow-
up of early-onset periodontitis patients. J Clin Periodontol 30, 562-572.
8. Hughes FJ, Syed M, Koshy B, Bostanci N, McKay IJ, Curtis MA et al. (2006) Prognostic factors in the treatment of generalized aggressive periodontitis: II. Effects of smoking on initial outcome. J Clin Periodontol 33, 671-676.
9. Baumer A, El Sayed N, Kim TS, Reitmeir P, Eickholz P, Pretzl B (2011) Patient-related risk factors for tooth loss in aggressive periodontitis after active periodontal therapy. J Clin Periodontol 38, 347-354.
10. Van Dyke TE, Sheilesh D (2005) Risk factors for periodontitis. J Int Acad Periodontol 7, 3-7.
11. Garcia RI, Nunn ME, Dietrich T (2009) Risk calculation and periodontal outcomes. Periodontol 2000 50, 65-77.
12. Matuliene G, Studer R, Lang NP, Schmidlin K, Pjetursson BE, Salvi GE et al. (2010) Significance of periodontal risk assessment in the recurrence of periodontitis and tooth loss. J Clin Periodontol 37, 191-199.
13. McNamara CM, Garvey MT, Winter GB (1998) Root abnormalities, talon cusps, dens invaginatus with reduced alveolar bone levels: case report. Int J Paediatr Dent 8, 41-45.
14. Xu L, Meng H, Tian Y, Zhang L, Feng X, Zhang G (2009) Evaluation of root abnormality in patients with aggressive periodontitis. Zhonghua Kou Qiang Yi Xue Za Zhi 44, 266-269.
15. McGuire MK, Nunn ME (1996) Prognosis versus actual outcome. II. The effectiveness of clinical parameters in developing an accurate prognosis. J Periodontol 67, 658-665.
16. Ekuni D, Yamamoto T, Takeuchi N (2009) Retrospective study of teeth with a poor prognosis following non-surgical periodontal treatment. J Clin Periodontol 36, 343-348.
17. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA (2002) Periodontitis-systemic disease associations in the presence of smoking--causal or coincidental? Periodontol 2000 30, 51-60.
18. Faggion CM Jr., Petersilka G, Lange DE, Gerss J, Flemming TF (2007) Prognostic model for tooth survival in patients treated for periodontitis. J Clin Periodontol 34, 226-231.
19. Eickholz P, Kaltschmitt J, Berbig J, Reitmeir P, Pretzl B (2008) Tooth loss after active periodontal therapy. I: patient-related factors for risk, prognosis, and quality of outcome. J Clin Periodontol 35, 165-174.
20. Armitage GC (1999) Development of a classification system for periodontal diseases and conditions. Ann Periodontol 4, 1-6.
21. Armitage GC (2004) Periodontal diagnoses and classification of periodontal diseases. Periodontol 2000 34, 9-21.
22. Mazza JE, Newman MG, Sims TN (1981) Clinical and antimicrobial effect of stannous fluoride on periodontitis. J Clin Periodontol 8, 203-212.
23. Lü D, Meng H, Xu L, Lu R, Zhang L, Chen Z et al. (2013) New attempts to modify periodontal risk assessment for generalized aggressive periodontitis: a retrospective study. J Periodontol 84, 1536-1545.
24. Lang NP, Tonetti MS (2003) Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). Oral Health Prev Dent 1, 7-16.
25. Wennstrom A, Wennstrom J, Lindhe J (1986) Healing following surgical and non-surgical treatment of juvenile periodontitis. A 5-year longitudinal study. J Clin Periodontol 13, 869-882.
26. Gunsolley JC, Califano JV, Koertge TE, Burmeister JA, Cooper LC, Schenkein HA (1995) Longitudinal assessment of early onset periodontitis. J Periodontol 66, 321-328.
27. Buchmann R, Nunn ME, Van Dyke TE, Lange DE (2002) Aggressive periodontitis: 5-year follow-up of treatment. J Periodontol 73, 675-683.
28. Tonetti MS, Steffen P, Muller-Campanile V, Suvan J, Lang NP (2000) Initial extractions and tooth loss during supportive care in a periodontal population seeking comprehensive care. J Clin Periodontol 27, 824-831.
29. Page RC, Offenbacher S, Schroeder HE, Seymour GJ, Kornman KS (1997) Advances in the pathogenesis of periodontitis: summary of developments, clinical implications and future directions. Periodontol 2000 14, 216-248.
30. Armitage GC (2010) Comparison of the microbiological features of chronic and aggressive periodontitis. Periodontol 2000 53, 70-88.
31. Jansson L, Lavstedt S, Zimmerman M (2002) Prediction of marginal bone loss and tooth loss—a prospective study over 20 years. J Clin Periodontol 29, 672-678.
32. Matuliene G, Pjetursson BE, Salvi GE, Schmidlin K, Bragger U, Zwahlen M et al. (2008) Influence of residual pockets on progression of periodontitis and tooth loss: results after 11 years of maintenance. J Clin Periodontol 35, 685-695.
33. Socransky SS, Haflajic AD, Cugini MA, Smith C, Kent RL Jr. (1998) Microbial complexes in subgingival plaque. J Clin Periodontol 25, 134-144.
34. Feng X, Zhang L, Xu L, Meng H, Lu R, Chen Z et al. (2014) Detection of eight periodontal microorganisms and distribution of Porphyromonas gingivalis fimA genotypes in Chinese patients with aggressive periodontitis. J Periodontol 85, 150-159.
35. Van der Velden U, Abbas F, Armstead S, Loos BG, Timmerman MF, Van der Weijden GA et al. (2006) Java project on periodontal diseases. The natural development of periodontitis: risk factors, risk predictors and risk determinants. J Clin Periodontol 33, 540-548.
36. Checchi L, Montevecchi M, Gatto MR, Trombelli L (2002) Retrospective study of tooth loss in 92 treated periodontal patients. J Clin Periodontol 29, 651-656.
37. Ng MC, Ong MM, Lim LP, Koh CG, Chan YH (2011) Tooth loss in compliant and non-compliant periodontally treated patients: 7 years after active periodontal therapy. J Clin Periodontol 38, 499-508.