Research Article

JPIS Journal of Periodontal & Implant Science

J Periodontal Implant Sci 2013;43:276-282 • http://dx.doi.org/10.5051/jpis.2013.43.6.276

Analysis of periodontal attachment loss in relation to root form abnormalities

Young-Mi Chung, Seong-Nyum Jeong*

Department of Periodontology, Daejeon Dental Hospital, Wonkwang University School of Dentistry, Daejeon, Korea

Purpose: The aim of this study was to explore root shape abnormalities, to investigate the influence of root form abnormalities on periodontal attachment loss, and to gather basic data to assist in the diagnosis and treatment of aggressive periodontitis.

Methods: From January 2010 to June 2012, a survey was conducted of all 3,284 periodontitis patients who visited the Department of Periodontology, Daejeon Dental Hospital, Wonkwang University School of Dentistry. Clinical parameters (probing depth, periodontal attachment loss, missing teeth) were measured and a radiographic examination was performed at the baseline. We classified the root shape abnormality of bicuspids and molars based on Meng classification.

Results: The periodontal attachment loss was the highest at the maxillary first molar (6.03 mm). The loss of the second molar was prominent. Type V deformity was shown to be the most common in the second maxillary and mandibular molars ($P<0.05$). Type V root shape was associated with the highest attachment loss ($P=0.01$).

Conclusions: Considering the small population and limited design of this study, definitive conclusions cannot be drawn. We suggest larger scale, methodologically more sophisticated studies that include normal controls and chronic periodontitis patients to clarify whether root form abnormalities are a potential risk factor for aggressive periodontitis.

Keywords: Aggressive periodontitis, Periodontal attachment loss, Tooth loss, Tooth root.

INTRODUCTION

Aggressive periodontitis is characterized by a rapid loss of periodontal attachment and alveolar bone. It commonly affects young adults [1-3]. As opposed to chronic periodontitis, the amount of biofilm and calculus accumulation in aggressive periodontitis is inconsistent with the severity and rate of progression of the periodontal destruction. These infections are subdivided into localized and generalized forms, according to the extent of the periodontal destruction [4]. The stringent age requirement used previously for diagnosis of early-onset periodontitis is no longer considered to be essential [5]. Even though there have been attempts to analyze aggressive periodontitis biochemically and microbiologically, there is no specific way to screen for the disease. Currently, early detection depends primarily on clinical and radiographic examination [1,5].

Many reports have discussed host susceptibility factors, including family aggregation, single nucleotide polymorphisms, polymorphonuclear neutrophils, antibodies to bacteria, smoking, stress, a local contributing factor (root morphology), and herpes virus infections [6]. Anatomic variations in teeth, such as cervical enamel projections, enamel pearls, intermediate bifurcation ridges, and root grooves have been regarded as etiologic cofactors in this destructive periodontal disease process [7]. It has been shown that molars are more vulnerable to attachment loss and are more frequently extracted [8-10]. Anatomical variations in molar root form may favor plaque

Received: Jun. 10, 2013; **Accepted:** Nov. 25, 2013
***Correspondence:** Seong-Nyum Jeong
Department of Periodontology, Daejeon Dental Hospital, Wonkwang University School of Dentistry, 77 Dunsan-ro, Seo-gu, Daejeon 302-120, Korea
E-mail: seongnyum@wonkwang.ac.kr, Tel: +82-42-366-1141, Fax: +82-42-366-1115

pISSN 2093-2278
eISSN 2093-2286

retention in these teeth and may contribute to an unfavorable crown-root ratio, resulting in increased susceptibility to loosening when they are subjected to heavy occlusal force [11]. Meng et al. [6] indicated that root shape abnormalities can be a susceptibility factor in the development of aggressive periodontitis and suggested root shape classification. Kim [12] reported that the ratio of root abnormalities was 1.76 times higher in aggressive periodontitis patients than in normal patients.

The aim of this study was to explore root shape abnormalities based on Meng classification, to investigate the influence of root form abnormalities on periodontal attachment loss and to gather basic data to assist in the diagnosis and treatment of aggressive periodontitis.

MATERIALS AND METHODS

Subjects

From January 2010 to June 2012, a survey was conducted of all 3,284 periodontitis patients who visited the Department of Periodontology, Daejeon Dental Hospital, Wonkwang University School of Dentistry. To qualify for inclusion in the survey, patients were required to display clinical and radiographic signs of aggressive periodontitis (based on the criteria of the American Academy of Periodontology International Classification of 1999) (Table 1). The patients were also required to be less than 35 years old at the time of the survey. Patients aged 35–40 years old who had suffered from periodontal disease since age twenty were also included. Of the 3,284 patients surveyed, 66 patients were selected as subjects for this study. The average age was 34.32 (±4.04). The numbers of males and females were 50 (2.2%) and 16 (1.6%), respectively, ($P>0.05$) (Table 2). Of the 66 subjects, 37 (56.1%) were smokers. To participate in the study, each subject was required to have a family history of destructive periodontitis back at least one generation. All of the subjects were thoroughly informed about the procedure and gave written consent for inclusion in the study. This study was approved by the Institutional Review Board of Wonkwang University Dental Hospital (IRB No. WKD IRB 20110201).

Table 1. Diagnostic criteria based on the 1999 AAP classification of periodontal diseases.

Localized aggressive periodontitis
Rapid attachment and bone loss in otherwise healthy patients
First molar-incisor presentation with no more than two other teeth affected
At least two permanent teeth affected where at least 1 is a first molar
Lifetime cumulative attachment loss ≥4 mm at the affected sites
Generalized aggressive periodontitis
Rapid attachment and bone loss in otherwise healthy patients
Generalized interproximal attachment loss affecting at least three teeth other than the first molars and incisors
Lifetime cumulative attachment loss ≥4 mm at the affected sites

AAP: American Academy of Periodontology.

Table 2. Number of patients by gender.

Gender	Periodontitis patients	Subjects
Female	1,032	16
Male	2,252	50
Total	3,284	66

Values are presented as number. $P<0.05$ was considered statistically significant. No statistically significant difference by gender ($P=0.108$).

Clinical examinations

Before clinical periodontal assessment, complete medical and dental histories were taken. The workup included clinical assessment and examination by a single examiner, including the probing depths, gingival recession (data not shown) at six points per tooth, and Löe's plaque index [13]. The probing pocket depth was measured from the free gingiva to the pocket base using a periodontal probe (PW, Hu-Friedy Manufacturing Co., Chicago, IL, USA). We measured the gingival recession from the cemento-enamel junction to the free gingiva with the same tool. We expressed periodontal attachment loss, defined as the length from the cemento-enamel junction to the depth of the pocket base, as the sum of the depth of the pocket and the gingival recession.

We classified roots according to the system published by Meng et al. [6]: cone root (type I), slender root (type II), curved root (type III), poor crown-root ratio (type IV), and syncretic root (type V) (Table 3, Fig. 1). We included the first and second

Table 3. The classification of root form by Meng [12].

Type	Type of root	Description
I	Cone root	Normal root
II	Slender root	Root morphology is too thin
III	Curved root	Root form is curved
IV	Maladjusted proportion of crown and root	Root form is abnormally short or long
V	Syncretic root	Two- or three-root fusion

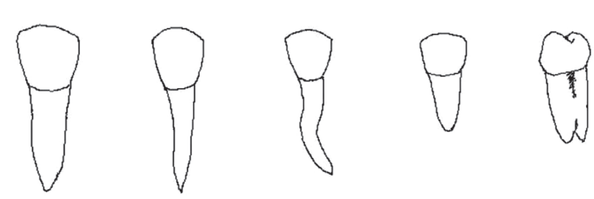

Figure 1. Root form of Meng classification [12].

premolars and the first and second molars of each arch in this root shape analysis study.

Tooth mortality was defined as the value obtained by adding the numbers of hopeless and missing teeth. The evaluation criterion for the hopeless category was an insufficient attachment level to maintain health, convenience, and function [14,15]. Following the clinical examination, full-mouth periapical radiographs were taken and analyzed for bone level, root deformity, or missing teeth using PiView STAR software (Infinitt Co., Seoul, Korea).

Statistical analysis

Data management procedures employed SPSS ver. 14.0. (SPSS Inc., Chicago, IL, USA). The tooth mortality and root form distribution were analyzed using the chi-square test. Periodontal probing depth and clinical attachment loss by root form was analyzed using one-way analysis of variance with Tukey or Dunnett T3 post hoc test.

Table 4. Smoking status of subjects.

Smoker, n (%)	Nonsmoker, n (%)	*P*-value
37 (56.1)	29 (43.9)	0.325[a)]

Nonsmoker means previous smoker and never-smoker. $P<0.05$ was considered statistically significant.
[a)]No statistically significant difference by smoking status.

Table 5. Periodontal pocket depth of representative teeth.

	First premolar	Second premolar	First molar	Second molar	*P*-value
Maxilla	4.0±1.61	4.38±1.54[c)]	5.16±2.26[a)]	5.06±2.20	0.000
Mandible	3.83±1.45	3.92±1.49[c)]	4.8±1.63	5.02±2.08[b)]	0.000
P-value	0.433	0.029	0.183	0.875	

Values are presented as mean±standard deviation (mm). $P<0.05$ was considered statistically significant.
[a)]Statistically significant difference in the maxilla group. [b)]Statistically significant difference in the mandible group. [c)]Statistically significant difference between the maxilla group and mandible group.

Table 6. Periodontal attachment loss of representative teeth.

	First premolar	Second premolar	First molar	Second molar	*P*-value
Maxilla	4.37±1.84	4.68±1.80[c)]	6.03±2.71[a,c)]	5.40±2.36	0.000
Mandible	4.11±1.58	4.16±1.59[c)]	5.14±1.86[b,c)]	5.02±2.08	0.000
P-value	0.266	0.028	0.005	0.594	

Values are presented as mean±standard deviation (mm). $P<0.05$ was considered statistically significant.
[a)]Statistically significant difference in the maxillary group. [b)]Statistically significant difference in the mandible group. [c)]Statistically significant difference between the maxilla group and mandible group.

RESULTS

There was no significant difference in prevalence of disease among smokers and nonsmokers ($P>0.05$) (Table 4). The plaque index averaged 1.7 (data was not shown).

The probing pocket depth was the deepest at the maxillary first molar and at the mandibular second molar (Table 5). The periodontal attachment loss was the highest at the first molar in both the maxilla and mandible (Table 6). The average number of missing teeth was 1.35 per subject. The tooth mortality was the highest among the maxillary first molars and the mandibular second molars (Table 7).

The results of root shape deformity analysis of bicuspids and molars showed type V deformity to be the highest in the second maxillary and mandibular molars. Type IV was the most common in the first mandibular molar ($P<0.05$). All five types of root shape deformity were seen in the maxillary second premolar only (Table 8). In a comparison of frequency of root form abnormalities, the maxillary incidence (16.7%) was higher than the mandibular incidence (10.9%), showing a significant difference ($P=0.008$) (Table 9). We analyzed the periodontal attachment loss of each tooth according to root form. In the second maxillary and mandibular molars, the type V root anomaly correlated with the highest periodontal attachment loss, 6.2 mm and 6.0 mm, respectively, among the deformity types, but not to a significant extent. There were

Table 7. Tooth mortality rate by tooth type.

	Missing teeth	Hopeless	Tooth mortality[a)]
Maxilla			
Central incisor	10 (7.56)	3	13 (9.85)
Lateral incisor	6 (4.55)	0	6 (4.55)
Canine	4 (3.03)	1	5 (3.79)
First premolar	9 (6.82)	9	18 (13.64)
Second premolar	3 (2.27)	11	14 (10.61)
First molar	12 (9.09)	16	28 (21.21)
Second molar	10 (7.56)	16	26 (19.67)
Total	54 (5.84)	56	110 (11.90)
Mandible			
Central incisor	5 (3.79)	7	12 (9.09)
Lateral incisor	2 (1.52)	5	7 (5.30)
Canine	0 (0)	0	0 (0)
First premolar	3 (2.27)	3	6 (4.55)
Second premolar	6 (4.55)	4	10 (7.56)
First molar	8 (6.06)	6	14 (10.61)
Second molar	11 (8.33)	9	20 (15.15)
Total	35 (3.79)	34	69 (7.47)

Values are presented as number (%). $P<0.05$ was considered statistically significant.
[a)]Statistically significant difference in the maxilla and mandible group.

Table 8. Analysis of root form deformity by tooth.

	Type I	Type II	Type III	Type IV	Type V	Total	*P*-value
Maxilla							
First premolar	90 (89.1)	2 (2.0)	6 (5.9)	3 (3.0)	0 (0)	101 (100)	0.000[a]
Second premolar	90 (85.7)	2 (1.9)	3 (5.7)	5 (4.8)	2 (1.9)	105 (100)	0.000[a]
First molar	89 (91.8)	0 (0)	0 (0)	3 (3.1)	5 (1.0)	97 (100)	0.000[a]
Second molar	65 (66.3)	0 (0)	0 (0)	5 (5.1)	28 (28.6)	98 (100)	0.000[a]
Mandible							
First premolar	101 (98.1)	0 (0)	0 (0)	2 (0.4)	0 (0)	103 (100)	0.000[b]
Second premolar	94 (94.0)	0 (0)	0 (0)	1 (1.0)	0 (0)	100 (100)	0.000[b]
First molar	95 (94.1)	0 (0)	0 (0)	6 (5.9)	0 (0)	101 (100)	0.000[b]
Second molar	68 (69.4)	0 (0)	0 (0)	12 (12.2)	18 (18.4)	98 (100)	0.000[b]

Values are presented as frequency (%). $P<0.05$ was considered statistically significant.
[a]Statistically significant difference between each tooth of the maxilla. [b]Statistically significant difference between each tooth of the mandible.

Table 9. Composition of root form by jaw type.

	Type I	Type II	Type III	Type IV	Type V	Total	*P*-value
Maxilla	334 (83.3)	4 (1.0)	12 (3.0)	16 (4.0)	35 (8.7)	401 (100)	0.000[a]
Mandible	358 (89.1)	0 (0)	5 (1.2)	21 (5.2)	18 (4.5)	402 (100)	0.000[b]
Total	692 (81.6)	4 (0.5)	17 (2.0)	37 (4.6)	53 (6.6)	803 (100)	

Values are presented as frequency (%). $P<0.05$ was considered statistically significant.
[a]Statistically significant difference in the maxilla. [b]Statistically significant difference in the mandible.

Table 10. Periodontal attachment loss of each tooth by root form.

Root type	First premolar	Second premolar	First molar	Second molar
Maxilla				
I	4.5±1.49	4.8±1.78	7.0±2.04	5.7±1.89
II	3.6±0.35	3.5±0.28	-	-
III	5.7±3.46	4.5±1.98	-	-
IV	3.4±0.96	5.7±0.60	7.1±0.74	5.7±2.11
V	-	2.8±0.07	8.1±2.90	6.2±1.98
P-value	0.177	0.256	0.245	0.583
Mandible				
I	4.2±1.49	4.4±1.28	5.5±1.46	5.5±1.58
II	-	-	-	-
III	-	4.3±0.78	-	-
IV	2.3±0.00	4.3±0.00	4.5±1.96	5.8±2.19
V	-	-	-	6.0±1.68
P-value	0.073	0.961	0.119	0.499

Values are presented as mean±standard deviation (mm). $P<0.05$ was considered statistically significant. There was no statistically significant difference in periodontal attachment loss of any of the types of teeth by root form.

no significant differences in attachment loss between the different deformities detected in the first and second premolars and first molars (Table 10). In the comparison between root shape abnormality and periodontal attachment loss regardless of tooth type, the type V root shape was associated with the highest periodontal attachment loss at 6.09±2.11 ($P=0.01$) (Table 11).

Table 11. Analysis of periodontal attachment loss by root form.

Type I	Type II	Type III	Type IV	Type V	*P*-value
5.07±1.05	3.53±0.26	5.05±2.30	5.35±2.15	6.09±2.11	0.01[a]

Values are presented as mean±standard deviation (mm). $P<0.05$ was considered statistically significant.
[a]Statistically significant difference in periodontal attachment loss between each deformity group.

DISCUSSION

Although we did our best to identify aggressive periodontitis patients based on clinical and radiographic evidence, we could not confirm the subjects of this study to be aggressive periodontitis patients. Classification of periodontal disease and the criteria for distinction between the two major forms of periodontitis are listed in the Consensus Reports of the 1999 International Workshop. However, distinguishing between the two forms in clinical practice remains problematic. For example, we looked for the primary criteria of rapid attachment and bone loss to diagnose aggressive periodontitis. However, any evaluation of disease progression requires some inference of the condition's severity with respect to patient age. According to current classification principles, age should

JPIS Journal of Periodontal & Implant Science

no longer constitute a primary determinant of diagnosis. Familial aggregation is also largely unhelpful [16]. A recent systematic review of the literature concluded that there is no evidence that the subgingival etiological factors in chronic periodontitis are substantially different from those of corresponding lesions in aggressive periodontitis [17]. Instead, it is generally accepted that the variation in susceptibility to periodontitis and types of disease are largely due to host factors [18]. In treating patients with aggressive periodontitis, therefore, early and aggressive treatment measures are more important than an investigation of etiology.

A review of the published literature suggests that the prevalence of aggressive periodontitis may vary significantly among countries and ethnicities. Albandar and Tinoco [19] reported that the prevalence of aggressive periodontitis was 10% in African-Americans, 5% in Hispanics, and 1.3% in white United States adolescents between 13 and 17 years of age. Low prevalence rates ranging between 0.1% and 0.2% have been reported in Europe [20]. In Japan, Kowashi [21] reported a 0.47% prevalence of aggressive periodontal disease in subjects between 19 and 28 years of age. The data from China showed the prevalence of aggressive periodontitis to vary between 0.12% and 0.47% in different areas of the country [22]. Of the surveyed initial 3,284 patients, 66 patients were provisionally assumed to have aggressive periodontitis (2.01% prevalence) with a mean age of 34.32, which was somewhat higher than that found in previous clinical studies [23]. It seems that this is because the subjects were patients who presented to the hospital rather than youths without symptoms. Considering an asymptomatic progression period and delay in patients visiting the hospital, we applied less strict criteria for age, especially since age is not a critical diagnostic criterion for aggressive periodontitis.

The gender ratio was male:female 3.13:1, but this was not a statistically significant difference. Studies are contradictory as to the predilection of periodontal disease according to gender. Differences in periodontal disease risk by sex have not been clearly demonstrated [24]. Baer [25] estimated a female:male ratio of about 3:1. Hormand and Frandsen [26] concluded that the disease affects females more often than males with a ratio of 2.5:1. Albandar and Rams [27] denied the existence of a relationship between aggressive periodontitis and gender. More studies are required to determine the gender ratio in aggressive periodontitis prevalence in Koreans.

In the present study, marked attachment loss over 5 mm was observed at the first molars. This result is consistent with our previous study [23]. It is assumed that the first molars might be affected earlier by periodontitis. Aggressive periodontitis developed as a localized form may progress into a generalized form with the involvement of more teeth with advancing age.

The average number of missing teeth was 1.35 at the first visit, but if we include the teeth diagnosed as hopeless, it doubled to 2.71. The most frequently missing teeth in the current study were the maxillary first molars (21.21%) and the mandibular second molars (15.15%). In the evaluation of tooth mortality, a high attachment loss of molars seems to lead to high tooth loss. We can assume that the high percentage of molar tooth loss might be because the molars had additional susceptibility factors such as occlusal interference, hygienic difficulty, and abnormal root form. Further epidemiologic studies of larger populations, comparing them with normal and chronic periodontitis subjects, are needed to assess the exact prevalence and pattern of aggressive periodontitis in Korea.

The shape, length, and spread of molar roots are important factors in tooth prognosis, as they can affect the anchorage and stability of molars. Molar root fusion is one of the most common anomalies in root morphological development [8,11]. Root fusion (type V) was the commonest anomaly in both the maxilla and mandible, and showed the highest prevalence in the second molars in this study. Ross and Evanchik [11] reported that a relatively high proportion of Europeans (70%) had one or more maxillary molars and 54% had one or more mandibular molars with fused roots. In the studies of Choi et al. [28] and Ryu et al. [29], root fusion demonstrated higher prevalence in the maxilla than mandible, and was the highest in the second maxillary molar. When we analyzed the degree of periodontal tissue destruction according to root shape, the second molars with root fusion (type V) had a greater risk of increased probing depth and periodontal attachment loss. In the correlation of root fusion and local inflammation, Choi et al. [28] reported a significance increase in clinical index–probing depth, gingival index, and tooth mobility in both the maxilla and mandible. Likewise, Hou and Tsai [8,30] described the correlation between root fusion and periodontal disease in a Chinese population. Negative characteristics of molars with root fusion, such as an unfavorable crown-to-root ratio, short root length, and a taper-shaped root, may offer less resistance to heavy occlusal loads and/or torque forces. This evidence suggests that molar root fusion may ultimately accelerate periodontal tissue destruction. However, Hou and Tsai's study was not conducted in patients with aggressive periodontitis. Given the similar results of the present study, we can cautiously infer that a fused root can be a contributing factor in disease progression of aggressive periodontitis as well as of chronic periodontitis.

In summary, periodontal attachment loss was the highest at the first molar in both the maxilla and mandible. Root fusion (type V) was the most prevalent among root abnormality types in both the maxilla and mandible, demonstrating the highest prevalence in the second molars. In the comparison between root shape abnormality and periodontal attachment loss regardless of tooth type, the type V root shape was associated with the highest attachment loss. Studies on the impact of root shape in patients with aggressive and chronic periodontitis are rare. Considering the small population and limited design of this study, definitive conclusions cannot be drawn. We suggest more large-scale, methodologically sophisticated studies that include normal controls and chronic periodontitis patients to clarify whether root form abnormalities are a potential risk factor for aggressive periodontitis.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

ACKNOWLEDGEMENTS

This study was supported by a 2012 Grant from Wonkwang University.

REFERENCES

1. Albandar JM, Brown LJ, Loe H. Clinical features of early-onset periodontitis. J Am Dent Assoc 1997;128:1393-9.
2. Tonetti MS, Mombelli A. Early-onset periodontitis. Ann Periodontol 1999;4:39-53.
3. Ranney RR. Classification of periodontal diseases. Periodontol 2000 1993;2:13-25.
4. Hansen BF, Gjermo P, Bergwitz-Larsen KR. Periodontal bone loss in 15-year-old Norwegians. J Clin Periodontol 1984;11:125-31.
5. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4:1-6.
6. Meng H, Xu L, Li Q, Han J, Zhao Y. Determinants of host susceptibility in aggressive periodontitis. Periodontol 2000 2007;43:133-59.
7. Leknes KN, Lie T, Selvig KA. Root grooves: a risk factor in periodontal attachment loss. J Periodontol 1994;65:859-63.
8. Hou GL, Tsai CC, Huang JS. Relationship between molar root fusion and localized periodontitis. J Periodontol 1997;68:313-9.
9. Dunlap RM, Gher ME. Root surface measurements of the mandibular first molar. J Periodontol 1985;56:234-8.
10. Gher ME, Vernino AR. Root morphology: clinical significance in pathogenesis and treatment of periodontal disease. J Am Dent Assoc 1980;101:627-33.
11. Ross IF, Evanchik PA. Root fusion in molars: incidence and sex linkage. J Periodontol 1981;52:663-7.
12. Kim SJ. A study on root morphology of anterior and premolars in Korean generalized aggressive periodontitis patients [dissertation]. Gwangju: Chonnam National University; 2009.
13. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967;38:610-6.
14. McGuire MK, Nunn ME. Prognosis versus actual outcome. II. The effectiveness of clinical parameters in developing an accurate prognosis. J Periodontol 1996;67:658-65.
15. Karen FN, Takei HH. Determination of prognosis. In: Newman MG, Takei HT, Klokkevold PR, Carranza FA. Carranza's clinical periodontology. 11st ed. St. Louis: Saunder Elsevier; 2012. p.373-83.
16. Picolos DK, Lerche-Sehm J, Abron A, Fine JB, Papapanou PN. Infection patterns in chronic and aggressive periodontitis. J Clin Periodontol 2005;32:1055-61.
17. Mombelli A, Casagni F, Madianos PN. Can presence or absence of periodontal pathogens distinguish between subjects with chronic and aggressive periodontitis? A systematic review. J Clin Periodontol 2002;29 Suppl 3:10-21.
18. Page RC, Offenbacher S, Schroeder HE, Seymour GJ, Kornman KS. Advances in the pathogenesis of periodontitis: summary of developments, clinical implications and future directions. Periodontol 2000 1997;14:216-48.
19. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. Periodontol 2000 2002;29:153-76.
20. Kronauer E, Borsa G, Lang NP. Prevalence of incipient juvenile periodontitis at age 16 years in Switzerland. J Clin Periodontol 1986;13:103-8.
21. Kowashi Y. Prevalence of juvenile periodontitis among students at Nagasaki University. Adv Dent Res 1988;2:395-6.
22. Wang ZL, Qu XQ, Yang SH, Li XY. Prevalence study of juvenile periodontitis. J Modern Stomatol 1990;4:171-2.
23. Cho CM, You HK, Jeong SN. The clinical assessment of aggressive periodontitis patients. J Periodontal Implant Sci 2011;41:143-8.
24. Van der Velden U, Abbas F, Armand S, Loos BG, Timmerman MF, Van der Weijden GA, et al. Java project on periodontal diseases. The natural development of periodontitis: risk factors, risk predictors and risk determinants. J Clin Periodontol 2006;33:540-8.
25. Baer PN. The case for periodontosis as a clinical entity. J Periodontol 1971;42:516-20.
26. Hormand J, Frandsen A. Juvenile periodontitis: localization of bone loss in relation to age, sex, and teeth. J Clin Periodontol 1979;6:407-16.

27. Albandar JM, Rams TE. Risk factors for periodontitis in children and young persons. Periodontol 2000 2002;29:207-22.
28. Choi BK, Hong KS, Chung CH, Lim SB. A study of distribution, prevalence and relationship of the localized periodontitis of first and second molar root fusion. J Korean Acad Periodontol 2006;36:503-13.
29. Ryu SH, Heo SR, Lee SJ, Chang MT, Kim HS. Distribution, prevalence and sex linkage of molar root fusion. J Korean Acad Periodontol 2002;32:61-8.
30. Hou GL, Tsai CC. The morphology of root fusion in Chinese adults (I): grades, types, location and distribution. J Clin Periodontol 1994;21:260-4.