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Aggressive periodontitis in children: a 14–19-year follow-up

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Abstract

Aim: The objective was to assess the recurrence of disease in subjects with a history of localized aggressive periodontitis (LAP).

Material and Methods: Initially, 11 children (7–13 years) with LAP were examined. Samples from the subgingival microbiota and soft tissue biopsies were obtained. Non-surgical periodontal therapy was performed and the affected deciduous and two permanent molars were extracted. The subjects were enrolled in a recall programme within the public dental services. Fourteen to 19 years after the initial examination, the subjects were recalled for clinical and radiographic examinations.

Results: The re-examination disclosed that two of the subjects exhibited recurrence of disease with probing pocket depth $\geqslant 6$ mm and bone loss of 3–4 mm at several teeth, while another two subjects presented a limited number of sites with the disease. While bleeding on probing was a general finding in the group, no further loss of attachment was detected in seven subjects. Advanced attachment loss at the deciduous teeth as well as the presence of *Aggregatibacter actinomycetemcomitans* and its specific clone JP at the initial examination did not correlate with the recurrence of disease at the 14–19-year follow-up.

Conclusion: Children treated for LAP do not always exhibit recurrence of periodontitis in the absence of supportive periodontal therapy over periods of 14–19 years.

Key words: aggressive periodontitis; bleeding on probing; bone loss; disease recurrence; radiographs

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Current classification of periodontitis distinguishes between the chronic and aggressive forms of the disease. Chronic periodontitis, which is the most common form, normally affects adults and is characterized by a slow or moderate rate of progression. Aggressive periodontitis, on the other hand, is in relative terms a rare disease with a prevalence varying between 0.1% and 0.5% (Albandar & Tinoco 2002). It is furthermore characterized by rapid attachment and bone loss at primary and permanent teeth in usually young subjects with a non-contributory medical history (Armitage 1999, Lang et al. 1999). The term ''aggressive'' in this context is used to characterize the disease regarding its early onset and rapid progression rather than specific histopathological features of the gingival lesion. In a review on adaptive host response in periodontitis, Berglundh & Donati (2005) concluded that the cellular composition in lesions of chronic and aggressive forms of periodontitis is similar. Nevertheless, because of the clinical features, subjects with aggressive periodontitis may represent a group of individuals with a unique susceptibility to periodontitis.

In a previous study from our laboratory, Berglundh et al. (2001) reported on local and systemic characteristics of host response in a group of 11 children with localized aggressive periodontitis (LAP). Gingival biopsies from diseased sites and blood samples were collected and evaluated in relation to data obtained from the analysis of samples from subjects with chronic periodontitis. In the current study, we report on the clinical and radiographic findings of periodontal conditions assessed in the subject group with aggressive periodontitis described by Berglundh et al. (2001) 14–19 years after the initial examination.

Conflict of interest and source of funding statement

The authors declare that there are no conflicts of interests.

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Material and Methods

The study protocol was approved by the regional ethical committee. Initially, 11 children (seven girls and four boys, aged 7–12 years; mean 9.5 ± 2.0) with signs of LAP were examined (for review see Berglundh et al. 2001). Ten of the children from four families had an African ethnic origin (Cape Verde Islands),

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while one subject was Caucasian (patient no. 7). The clinical and radiographic assessments included detection of sites that exhibited bleeding on probing (BoP) and a distance between the cemento-enamel junction (CEJ) and the bone crest (BC) exceeding 2 mm. Samples from the subgingival microbiota and soft tissue biopsies were obtained from the identified sites. Results from the different examinations including the presence of *Aggregatibacter actinomycetemcomitans* (A.a.) in the microbial samples were initially reported by Berglundh et al. (2001) and are also presented in Table 1. Further analyses of the A.a. samples regarding the specific JP2 clone were subsequently performed by Haubek et al. (1996). Subjects that were positive to this clone in the initial examination (patients 3, 4, 8, 9, 10 and 11) are described in Table 1.

Following examination, the subjects received non-surgical periodontal therapy including mechanical infection control. All affected deciduous molars and, in patient no. 10, two permanent teeth were extracted. The non-surgical therapy in the remaining sites resulted in pocket closure and resolution of inflammation. The subjects were enrolled in a regular recall programme within the home clinic of the public dental services. This programme consisted of annual controls with clinical and, when indicated, radiographic examinations, while no specific supportive periodontal therapy programme was provided to this group of subjects.

At 14–19 years after the initial examination and treatment, the 11 subjects were recalled for clinical and radiographic examinations at the Clinic of Periodontics, Public Dental Services, Gothenburg. One of the subjects (patient no. 7) did not attend the clinical examination but recently obtained radiographs were accessible. The age of the 10 remaining subjects (three males and seven females) at the follow-up examination varied between 21 and 32 years (mean 26 ± 3.2). Questionnaires and data from patient files were used to document the frequency and type of examinations and treatment performed during the 14–19-year period between the initial and the follow-up examinations.

The clinical follow-up examination included assessments of probing pocket depth (PPD) and BoP. Bitewing radiographs from pre-molar and molar segments were obtained. In the radiographs,

Table 1. Clinical, radiographic and microbiological data obtained at the initial and follow-up examinations.

Subject number	Initial examination						14–19-year follow-up examination						
	age at initial examination	no. of all sites BoP+ PPD $\geqslant$6 mm CEJ–BC $>$ 2 mm	no. of sites at permanent teeth	CEJ–BC mean(range) mm*	% a.a.	JP2	age at follow-up examination	BoP%	PPD $\geqslant$4 mm	PPD $\geqslant$6 mm	CEJ–BC $>$3 mm†	CEJ–BC $>$4 mm†	CEJ–BC mean(range) mm‡
1	8	12		4.23 (2.76–6.43)	0.1		23	25	29	10	3	3	7.40 (5.00–9.99)
2	11	4		2.83 (2.18–3.29)	0		27	57	17	8	11	7	6.58 (3.11–12.65)
3	9	3		2.70 (2.20–4.96)	50.9	+	25	53	29	0	0	0	
4	11	3		2.59 (2.31–2.97)	8.9	+	26	19	12	0	0	0	
5	7	5		2.67 (2.07–3.28)	0		23	51	45	0	0	0	
6	8	1		2.54 –	37.5		24	49	42	0	0	0	
7	8	8		3.48 (2.53–5.81)	40.1		25		Missing		0	0	
8	7	2		4.98 (3.93–6.02)	2.6	+	21	21	25	0	0	0	
9	12	1	1	2.37 –	54.5	+	30	50	26	2	1	0	3.63
10	12	3	2	3.67 (3.27–4.42)	18.2	+	27	59	34	8	4	0	3.25 (3.15–3.32)
11	12	1	1	2.87 –	22.7	+	32	29	0	0	1	0	3.24

*Mean values of sites CEJ–BC $>$ 2 mm (initial examination).
†Distance from CEJ–BC, number of sites.
‡Mean values of sites CEJ–BC $>$ 3 mm (follow-up examination).
BoP, bleeding on probing; CEJ, cemento-enamel junction; BC, bone crest; A.a., *Aggregatibacter actinomycetemcomitans*; PPD, probing pocket depth.

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the distance between the CEJ and the alveolar BC was determined. The radiographic measurements were performed using an Olympus SZH10 stereo macroscope (Olympus optical co. Ltd., Tokyo, Japan) with a Leica DFC280 camera connected to a computer with a Leica QWin software (Leica Imaging Systems Ltd., Cambridge, UK). The finding of BoP and a distance >3 mm between CEJ and BC in radiographs was considered as recurrence of disease.

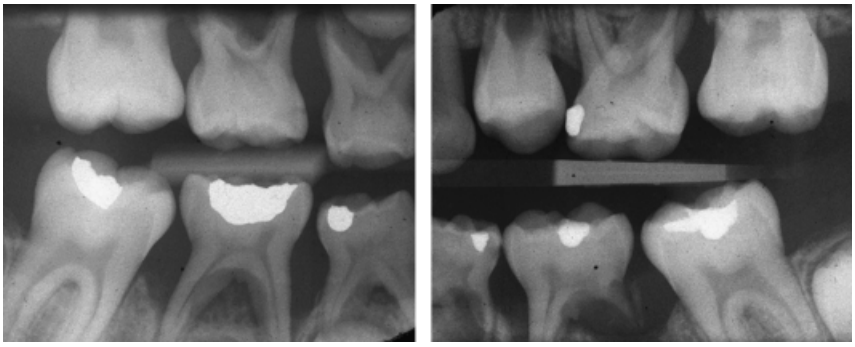

Fig. 1. Subject no. 1, 8 years old at the initial examination.

Results

Examinations and treatment during the 14–19-year period

Information obtained from the subjects and the analysis of patient files revealed that the frequency of annual recall visits in their home clinics varied between 40% and 70% during the 14–19-year period between the two examinations. No subjects were smokers.

Two of the subjects (patient no. 1 and 2) exhibited recurrence of disease in several sites during the period between the two examinations and, in these siblings, tooth extractions were performed. In patient no. 1, tooth 36 was extracted about 2 years after the first examination, while 16 was extracted in conjunction with the second examination. Radiographs from subject no. 1 obtained during the follow-up period are presented in Figs 1–5. Patient no. 2 was referred for periodontal therapy immediately before the second examination and, in addition to non-surgical and surgical periodontal therapy, extractions of the teeth 26 and 36 were performed. In the majority of the remaining group of subjects, no data indicating recurrence of disease were found (Figs 6 and 7).

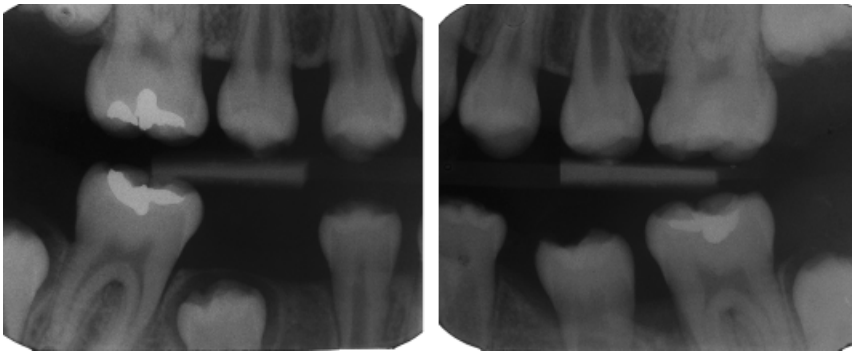

Fig. 2. Subject no. 1, 10 years old.

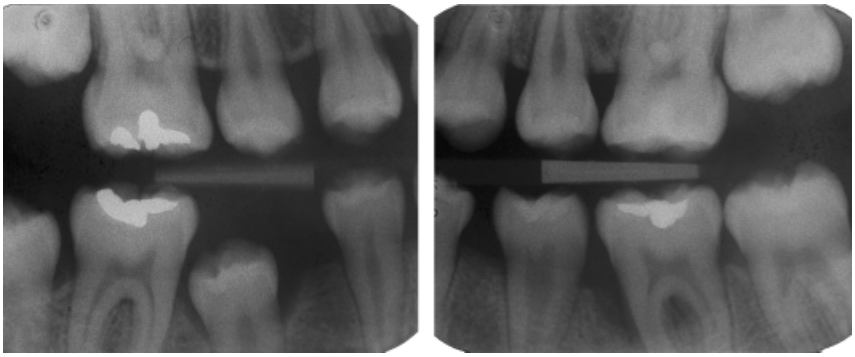

Fig. 3. Subject no. 1, 11 years old. Note the disease progression on tooth 36.

Clinical and radiographic re-examination

The results from the clinical and radiographic follow-up examination are presented in Table 1. The overall %BoP varied between 19% and 59% in the subjects. All but one had sites with PPD$\geqslant$4 mm, while sites with PPD$\geqslant$6 mm were detected in four of the subjects.

The number of pre-molar and molar sites with bone levels identified at >3 mm from the CEJ were found in five of the subjects, while only two subjects (patient no. 1 and 2) had tooth sites with bone levels of >4 mm.

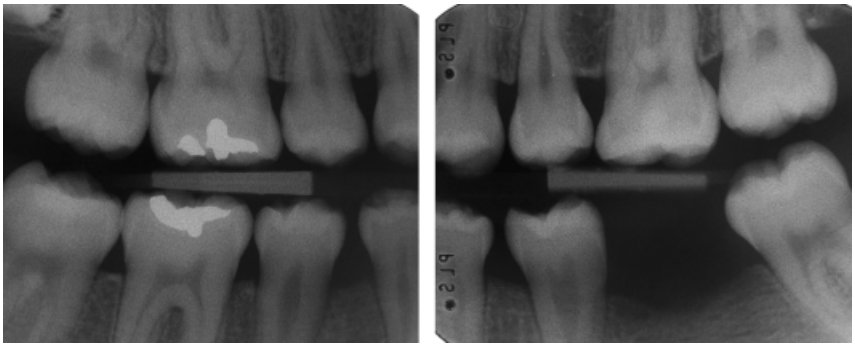

Fig. 4. Subject no. 1, 12 years old. Tooth 36 extracted.

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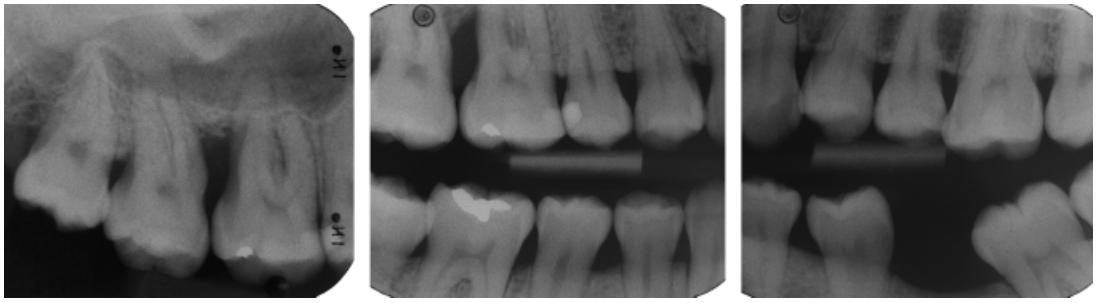

Fig. 5. Subject no. 1, 23 years old at the follow-up examination.

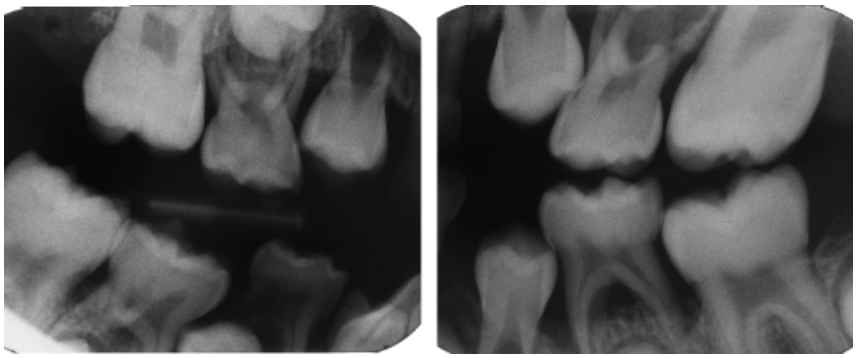

Fig. 6. Subject no. 8, 7 years old at the initial examination.

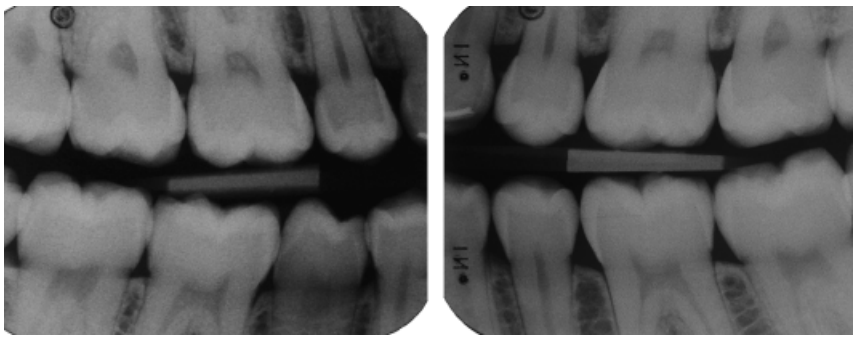

Fig. 7. Subject no. 8, 21 years old at the follow-up examination.

Discussion

The present report presents data from a 14–19-year follow-up of 11 subjects all of whom had a history of LAP at deciduous and permanent teeth. Re-examination disclosed that two of the subjects exhibited signs of recurrence of disease with PPD $\geqslant$ 6 mm and bone loss of 3–4 mm at several teeth, while another two subjects presented a limited number of diseased sites with a smaller amount of bone loss. While BoP was a general finding in the group, no further loss of attachment was detected in seven subjects. Advanced attachment loss at deciduous teeth as well as the presence of A.a. and its specific JP2 clone at the initial examination did not correlate with recurrence of disease at the 14–19-year follow-up. It is suggested that children treated for LAP do not always exhibit recurrence of periodontitis in the absence of supportive periodontal therapy over periods of 14–19 years.

Studies on long-term follow-up of children with a history of localized aggressive (or formerly pre-pubertal) periodontitis appear to be absent, whereas few reports exist on adolescents who were monitored after treatment of the former diagnosis juvenile periodontitis. Thus, in a 5-year follow-up, Lindhe & Liljenberg (1984) reported that treatment of localized juvenile periodontitis (LJP) in 16 subjects, 14–18 years of age, resulted in resolution of gingival inflammation, clinical attachment gain and radiographic bone fill in angular bone defects. The treatment included systemic antibiotics, non-surgical and surgical periodontal therapy and professional tooth cleaning every third month during the 5-year monitoring period. Wennström et al. (1986), in a similar 5-year study on 16 young subjects with LJP, reported that surgical or non-surgical periodontal therapy without systemic antibiotics together with a maintenance care programme of recall visits every 6 months during the first 2 years resulted in improved periodontal conditions with clinical attachment gain and reduced PPD. In a retrospective study, Saxén et al. (1986) reported on the treatment outcome of juvenile periodontitis in 20 subjects, aged 15–29 years. The treatment consisted of non-surgical and surgical periodontal therapy followed by a maintenance programme including professional tooth cleaning at intervals of 2–10 months. Clinical examinations made at 6–12 years after periodontal therapy revealed that 18 out 584 teeth were lost and that periodontal conditions, as described by PPD, % BoP and bone loss scores, were markedly improved. Although the subject sample was considerably older, the results of the re-examination in the study reported by Saxén et al. (1986) are in agreement with data presented in the current study. Thus, among the subjects in the present material, few teeth were lost during the 14–19 years of follow-up and obvious signs of recurrence of periodontitis were restricted to two of the 10 subjects. While the subjects in the studies by Lindhe & Liljenberg (1984), Wennström et al. (1986) and Saxén et al. (1986) received a comprehensive maintenance care programme after the initial periodontal therapy, no similar strategy

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was provided to the children in the present material. This difference in maintenance during the follow-up period is reflected by the overall large % of BoP at the re-examination of the current study on the one hand, but not by the limited recurrence of periodontitis on the other.

The finding in the present study that only few subjects with LAP exhibited signs of recurrence of periodontitis during the 14–19-year follow-up period without supportive periodontal therapy is in contrast to the data presented by Brown et al. (1996). They followed 91 adolescents who at baseline were 13–20 years old and were classified into LJP, generalized juvenile periodontitis or incidental attachment loss categories. The re-examination performed after 6 years revealed that the severity and extent of periodontitis had increased. Thus, the number of affected teeth was greater than before and sites with previously detected disease exhibited further loss of attachment. Oliveira Costa et al. (2007) reported on periodontal disease progression in 44 children, aged 8–15 years, during a 52-month period with no dental care. Similar to the study by Brown et al. (1996) the re-examination demonstrated an increase in the number of sites with loss of clinical attachment and bone. While the studies by Brown et al. (1996), and Oliveira Costa et al. (2007) illustrate disease progression in untreated study samples, the children in the current material exhibited limited recurrence of disease following initial but no supportive periodontal therapy.

The fact that aggressive periodontitis is associated with the presence of A.a. is well known. Recent studies, however, revealed a particular strong association between the specific JP2 clone of A.a. and this disease in adolescents of an African origin (Haubek et al. 2007). In this context, it is interesting to note that the initial examination of the subjects in the current study included sampling of the subgingival microflora of affected teeth and the detection of A.a. As outlined in Table 1, only six of the 11 subjects showed samples positive for the JP2 clone. In addition, samples obtained from the two subjects exhibiting recurrence of disease at the 14–19-year re-examination had no or only small fractions of A.a. and were, accordingly, negative regarding the JP2 clone.

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Clinical Relevance

Scientific rationale for the study: Children with aggressive periodontitis are regarded as highly susceptible to periodontitis. It is not known, however, if such subjects also present recurrence of periodontitis after periods of 14–19 years of follow-up without supportive periodontal therapy.
Principal findings: Out of 11 initially identified children with LAP, only two subjects exhibited obvious signs of recurrence of disease, while another two subjects exhibited minor signs of disease. Considering that only mechanical infection control and extraction therapy was carried out initially, it is interesting to note that advanced attachment loss at deciduous teeth as well as the presence of A.a. and its specific JP2 clone at the initial examination did not correlate with the recurrence of disease at the 14–19-year follow-up.
Practical implications: None.

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