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Periodontal management of an adolescent with Down's syndrome – a case report

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Summary. A case of periodontitis in a young adolescent Japanese girl with Down's syndrome is presented in this report. The patient received a monthly preventive course of dental care consisting of mechanical plaque control and oral hygiene instruction. After 2·5 years she recovered from progression of periodontal disease both clinically and microbiologically. The importance of clinical care for periodontitis in Down's syndrome patients is discussed.

Introduction

Down's syndrome is an autosomal disorder with an incidence of 1/700, caused by an extra chromosome 21. The syndrome is characterized by short stature, characteristic facial features with a protruding tongue, a wide range of learning difficulties, congenital heart disease, gastrointestinal disorders and other features. Dental characteristics have included abnormally rounded labial forms of tooth crown, missing teeth, delayed eruption, and malocclusions such as crowding, posterior crossbite and anterior open bite [1]. Periodontally, both the primary and the permanent dentitions are affected by a rapidly progressing and severe inflammation in more than 50 per cent of patients with Down's syndrome [2,3]. There are defects of chemotaxis and intracellular killing of polymorphonuclear and other phagocytes, which explains the high incidence of pocketing and marginal bone loss. The oral flora is not different from that of their siblings [4] while the periodontal breakdown is more pronounced [5]. It is also more severe than in matched patients with learning disabilities without Down's syndrome [6]. Meyle and Gonzales described the influence of Down's syndrome on periodontitis in children and adolescents [7]. It has been suggested that endogenous factors might contribute to the rapid progression of periodontal breakdown, e.g. inappropriate regulation of enzymes and T cell immunodeficiency [8] together with the functional defects of polymorphonuclear leucocytes and monocytes already highlighted [9]. These factors, together with the possibility of differences in collagen biosynthesis, abnormal capillary morphology [8] and hyperinnervation of the gingivae [10], may contribute to the rapid periodontal destruction observed in these patients.

A recent study has investigated the relationship between periodontopathic bacteria and early onset periodontitis in Down's syndrome [11]. Among 67 subjects with Down's syndrome, 28 individuals (41·8%: mean age $23{\cdot}75 \pm 4{\cdot}22$) had gingivitis (average probing depth $< 3{\cdot}5$ mm). No significant differences were observed in the bacterial profiles between the young adults with Down's syndrome and 41 age-matched, systemically healthy, individuals with other types of learning disabilities. Nonetheless Down's syndrome subjects generally develop an earlier and more extensive periodontal breakdown than those with other mental disabilities, suggesting that early onset periodontitis in Down's syndrome is mainly due to a compromised host status (including immunological deficiency, fragile periodontal tissues) and early senescence, rather than specific microbial agents. A longitudinal study of the progress of periodontal disease in 34 adolescents with Down's

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syndrome (mean age 16·6 ± 3·6 years) demonstrated that the frequency of periodontitis markedly increased in a 7-year follow-up period [12]. Although some longitudinal studies have investigated the development of periodontal disease in Down's syndrome [12–14], there have been almost no reports evaluating the effectiveness of a clinical preventive course for periodontitis in Down's syndrome patients. In one of few studies, carried out to determine the effectiveness of supragingival plaque control and oral hygiene instruction, clinical and microbiological parameters were monitored over a period of 12 weeks in 10 patients with Down's syndrome (aged 20–31 years; mean age 26·3 years) [14]. Subsequent to the initial examination and a professional tooth cleaning program, neither an improvement in the gingival condition (i.e. a significant alteration of the microbial composition) nor a reduction in the mean probing depth or the percentage of sites with probing depth > 4 mm could be detected.

In this study, an adolescent girl with Down's syndrome was diagnosed with periodontitis through mobility of her lower incisors. The aim of this report was to report on the periodontal management in the case of an adolescent with Down's syndrome.

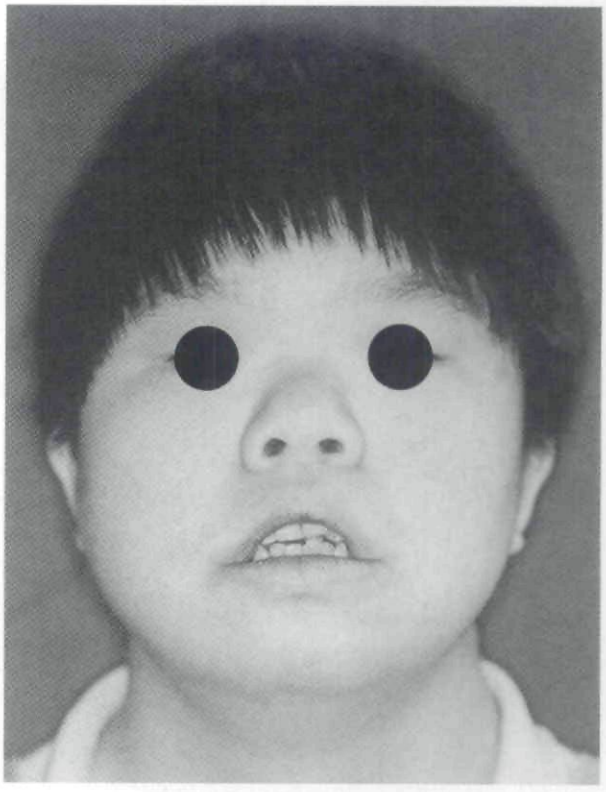

Fig. 1. Facial appearance of the patient demonstrating short neck, short nose and anterior open bite.

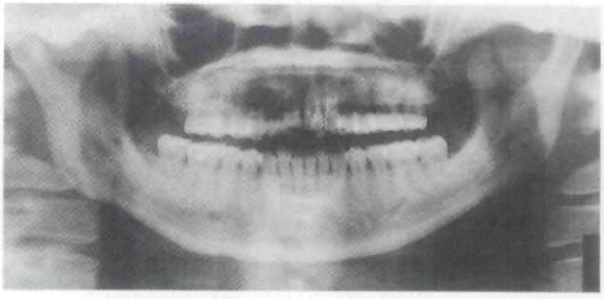

Fig. 2. Panoramic radiograph of the patient at age of 18 years. (at initial visit).

Case report

An 18-year-old Japanese girl with Down's syndrome was brought by her aunt, who was also her foster mother, to the Department of Pediatric Dentistry, Faculty of Dentistry, Kyushu University (Fig. 1). Her degree of intellectual disability was classified as 'moderate' by a simplified Intelligence Quotient (IQ) test. She had been initially referred to our hospital by a school dentist when she was 8 years old because of her malocclusion. By the time the study started in 1999, the patient had received regular dental care for 10 years, including examinations by a dentist at 1–3 months intervals and frequent visits for preventive care of individual caries, supragingival plaque removal and oral hygiene instruction. At the start of the study, the patient had a total of 27 teeth (upper right second molar and all four third molars were congenitally absent) (Fig. 2) with some resin restorations. This patient was chosen for assessment because, at 18 years of age, her periodontitis was clearly diagnosed as a result of her lower central incisors being so mobile that specific periodontal management was needed.

The patient had no medical disease (including heart disease) and had never shown an increased frequency of infections. She graduated from a school for disabled children and has attended a protective institution since graduating from high school. She took care of herself at home and brushed her teeth twice a day, once in the morning and once at night before going to bed, without assistance or supervision from her foster mother. She had never participated in any additional preventive-care programs.

The full mouth dental radiographs and the detailed periodontal chart at the initial visit are shown in

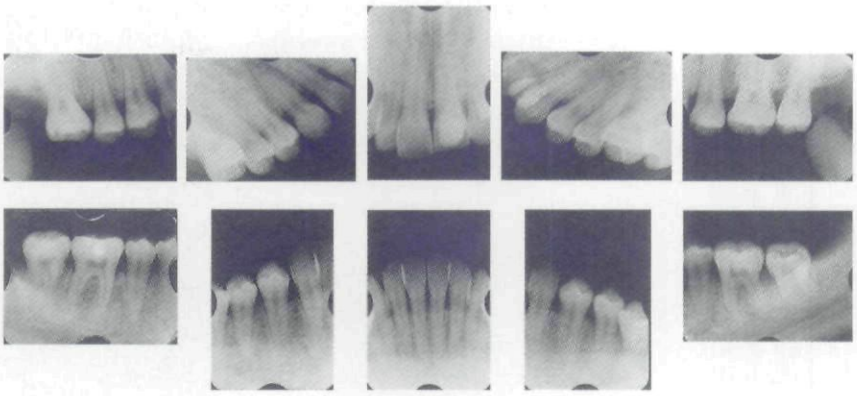

Fig. 3. Radiographic full mouse series at the initial visit. Radiographic findings indicate generalized mild to moderate horizontal alveolar bone loss with localized vertical bone loss in the molar areas.

Tooth	Periodontal pocket (mm) +:BOP						Tooth mobility
	DB	B	MB	DL	L	ML	
~~18~~							
~~17~~							
16	3+	2+	2+	4+	4+	5+	
15	2	2	3	3+	2+	2+	
14	2	2	2	3+	2+	3+	
13	4+	2+	5+	6+	4+	6+	
12	4+	2+	3+	6+	5+	6+	
11	3+	2+	4+	6	4	4	
21	2+	2+	4+	6+	6+	4+	
22	2+	4+	3+	2+	3+	3+	
23	2	2	2	2+	3+	6+	
24	2+	2+	2+	2+	2+	3+	
25	3+	2+	2+	2+	2+	3+	
26	2+	2+	2+	4+	2+	2+	
27	3+	2+	2+	4+	4+	3+	
~~28~~							
~~48~~							
47	3	2	2	3+	2+	2+	
46	3+	2+	2+	5+	4+	4+	
45	3+	2+	2+	3+	2+	3+	
44	2+	1+	3+	3+	2+	3+	
43	4+	2+	3+	3+	2+	2+	
42	3	2	2	3+	2+	3+	
41	3+	2+	4+	2+	2+	3+	I
31	3+	2+	2+	4+	2+	2+	I
32	2+	2+	2+	3+	2+	4+	
33	4	2	3	4+	2+	3+	
34	3+	1+	2+	3+	2+	3+	
35	3+	1+	3+	3+	3+	3+	
36	3	2	2	5+	6+	5+	
37	2+	2+	2+	6+	4+	3+	
~~38~~							

Fig. 4. Periodontal chart at the initial visit. DB: distobuccal, B: buccal, MB: mesiobuccal, DL: distolingual, L: Lingual, ML: mesiolingual, BOP: bleeding on probing.

Figs 3 and 4. The diagnosis of periodontitis was based on dental radiographs taken to estimate the degree of alveolar bone loss. The distance from the cement-enamel junction (CEJ) to the alveolar bone crest (AC) on the mesial and distal surfaces of the first permanent molars and the central incisors in the upper and lower jaws were measured, with the exception of the distal surfaces of upper central incisors because their images overlapped with those of the upper lateral incisors in the radiographs. The total number of sites measured was 14. The degree of alveolar bone loss was measured at the beginning of the study and 2·5 years later. Periodontitis was considered present when the CEJ to AC distance exceeded 2·0 mm on the radiographs [15]. The level of calculus on the proximal surfaces was determined from dental radiographs of 54 sites from 27 teeth.

The patient's oral health was evaluated over the following 2·5 years during which period monthly care consisted of professional tooth cleaning and combinations of scaling methods (i.e. ultrasonic scaling and hand scaling), oral hygiene instructions (to the patient), and counselling to the foster mother. The treatment plan was according to guidelines for periodontal therapy recommended by American Academy of Periodontology [16]. Briefly, initial periodontal treatment for the patient is undertaken in three phases: (i) patient education, training in personal oral hygiene, and counselling on control of risk factors; (ii) removal of supragingival and accessible subgingival bacterial plaque and calculus by periodontal scaling. Root surface irregularities and root surfaces altered by periodontal pathoses are treated by comprehensive periodontal root planing, and (iii) finishing procedures, which include post-treatment evaluation and review, and reinforcement of personal daily oral hygiene where appropriate.

The full mouth dental radiographs taken at the 2·5 year follow-up are shown in Fig. 5. The proportions of sites with CEJ to AC distance exceeding 2·0 mm at the beginning and end of the study were 57·1% (8/14) and 42·9% (6/14), respectively. There

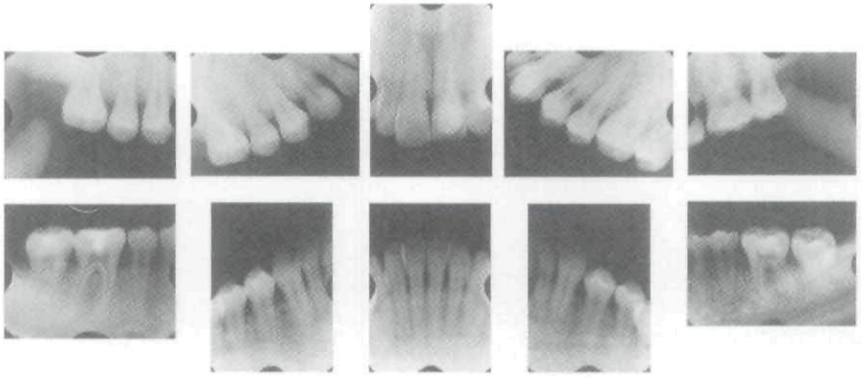

Fig. 5. Radiographic full mouse series at the 2·5 yr follow-up.

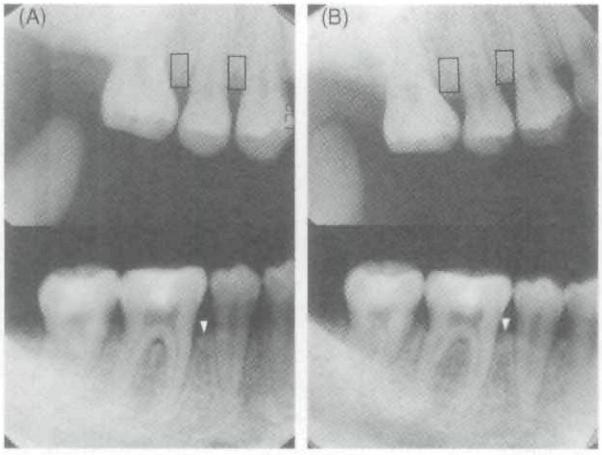

Fig. 6. Radiographs show the remodeling of the alveolar bone from the initial visit (A) to the 2·5 yr follow-up (B). Bone density increased (square grid) and the ridge of alveolar bone became distinct and projected higher (arrow heads).

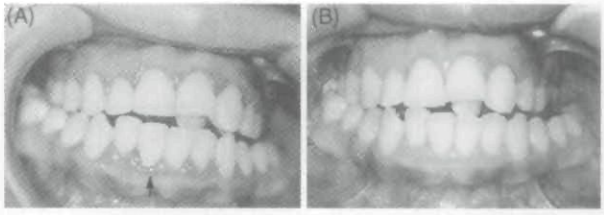

Fig. 7. Intraoral front view of the patient at the initial visit (A) and at the 2·5 yr follow-up (B) shows that gingival inflammation (arrow) improved during the practical course of preventive care.

Table 1. Change in percentage of sites with bleeding on probe (GBI%) and percentage of sites with probing depths of < 3 mm, 3–4 mm, and > 4 mm. Actual numbers/total observations are in parentheses.

	Initial visit	6 months	2·5 years
GBI	85·2 (138/162)	69·8 (113/162)	9·9 (16/162)
Probing depth			
< 3 mm	47·5 (77/162)	80·9 (131/162)	95·7 (155/162)
3–4 mm	42·6 (69/162)	19·1 (31/162)	4·3 (7/162)
> 4 mm	9·9 (16/162)	0 (0/162)	0 (0/162)

was no progression of alveolar bone loss between 1999 and 2002. Conversely, radiographic density and defective sites decreased, probably because of bone remodelling (Fig. 6) and at two sites (the mesial surfaces of the upper left and lower right first molars) alveolar bone loss decreased to less than 2 mm. No changes in bone loss were seen at other sites including those surrounding lower anterior teeth. Calculus was visible in 14·8% (8/54) of the sites in 1999 but at 0% (0/54) sites in 2002.

Gingival inflammation was assessed clinically by recording any bleeding following probing of the gingival sulcus, and the percentage of surfaces with gingivitis (GBI%) was estimated. Pocket depth was measured to the nearest 1 mm using a periodontal probe (type LM Instruments, Abo, Finland: pressure: 25–30 g). All measurements were performed from the peak of the gingival margin at six points (mesiobuccal, buccal, distobuccal, distolingual, lingual, and mesiolingual) around every tooth.

At the beginning of the study an intraoral frontal view of the patient showed moderate swelling especially in the incisal region (Fig. 7A). The detailed periodontal chart at the 2·5 year follow-up and the clinical findings throughout the study are presented in Fig. 8 and Table 1, respectively. Prior to the initiation of preventive care, gingival inflammation (expressed as GBI%) was 85·2%. The number of sites exhibiting pathological periodontal pockets (> 4 mm) and (4–3 mm) was 9·9% and 42·6%, respectively. During the course of the preventive care, both GBI% and probing depth improved. After 6 months, the

Tooth	Periodontal pocket (mm) +:BOP						Tooth mobility
	DB	B	MB	DL	L	ML	
~~18~~							
~~17~~							
16	1	1	1	2	1+	1	
15	1	1+	1	1	1	1	
14	1	1	1	1	1	1	
13	1	1	1	3	1	2	
12	2	1	1	3	1	2	
11	1	1	1	1	1	1	
21	1	1	1+	4	1+	3	
22	2	4	2	1	1	1	
23	2	1	2	2+	2	4	
24	2	2	2	2	2	2	
25	2+	1	2	2	2+	2	
26	2	2	2	2	2	2+	
27	3+	2	1	2	2	2+	
~~28~~							
~~48~~							
47	2	1	2	1	1	1	
46	1	1	1	1	1	1	
45	1	1	1	2	1	1	
44	1	1	1	1	1+	1	
43	1	1	1	2	1	1	
42	2	1	1	1	1	1+	
41	1	1	1	1	1+	1+	
31	1	1+	1	1	1	1	
32	1	1+	1	2	1	1	
33	2	2	2	1	1	1	
34	1	1	1	2	2	2	
35	1	1	1	1	1	1	
36	2	1	1	1	1	1	
37	2	2	1	2	2	2	
~~38~~							

Fig. 8. Periodontal chart at the 2·5 yr follow-up. DB: distobuccal, B: buccal, MB: mesiobuccal, DL: distolingual, L: Lingual, ML: mesiolingual, BOP: bleeding on probing.

GBI% was reduced to 69·8%, and pathological periodontal pockets of (> 4 mm) and (4–3 mm) were reduced to 0% and 19·1%, with an increase in the sites of < 3 mm to 80·9%. At the 2·5 year follow-up, sites with bleeding on probe and pathological pockets were markedly reduced (Table 1). Mean probing depth was reduced from 2·9 mm at the initial visit to 2·1 mm after 6 months follow-up and to 1·3 mm after 2·5 years. An intraoral frontal view of the patient at the 2·5 year follow-up showed an improvement in the degree of gingival swelling (Fig. 7B). The upper left anterior teeth still had gingival swelling because of a relapse in oral hygiene.

To detect the presence of *Actinobacillus actinomycetemcomitans*, *Bacteroides forsythus*, *Camphylobacter rectus*, *Eikenella corrodens* and *Porphyromonas gingivalis*, subgingival plaque samples were obtained from the surface with the deepest probing depth at each of the four first molars. In this study, the polymerase chain reaction (PCR) method was used to investigate the putative periodontal pathogens harboured in the subgingival plaque [17], and the conditions of PCR for the indicated DNA fragments are shown in Table 2 [18–20]. Samples were amplified for 35 cycles (95°, 60 s; 62°, 60 s; 72°, 60 s). After treatment of the fragment with primers, DNA samples were analysed by electrophoresis using 1·8% (w/v) agarose gel. Current techniques for periodontal microbial identification include culture, nucleic acid and immunologically-based diagnostic methods. Polymerase chain reaction (PCR) method used in our study allows the specific amplification of target bacterial DNA, and offers a highly sensitive and specific detection method for bacteria in biological samples [21]. This detection method is more sensitive than culture, and seemed to have less cross-reactivity than DNA probe detection [18]. PCR has been used to detect the presence of less than 100 specific bacteria in biological samples and can be used to rapidly determine the presence of an extremely low number of putative pathogens in multiple sites from the

Table 2. Primer pairs and PCR conditions for bacteria.

Gene	Primer pairs	Size of product (bp)
Universal primers for positive control [20]	F:5′-CAGGATTAGATACCCTGGTAGTCCACGC-3′ R:5′-GACGGGCGGTGTGTACAAGGCCCGGGAACG-3′	625
A. actinomycetemcomitans [20]	F:5′-GGAATTCCTAGGTATTGCGAAACAATTTGATC-3′ R:5′-GGAATTCCTGAAATTAAGCTGGTAATC-3′	262
B. forsythus [18]	F:5′-GCGTATGTAACCTGCCCGCA-3′ R:5′-TGCTTCAGTGTCAGTTATACCT-3′	641
C. rectus [18]	F:5′-TTTCGGAGCGTAAACTCCTTTTC-3′ R:5′-TTTCTGCAAGCAGACACTCTT-3′	598
E. corrodens [18]	F:5′-CTAATACCGCATACGTCCTAAG-3′ R:5′-CTACTAAGCAATCAAGTTGCCC-3′	688
P. gingivalis [19]	F:5′-ATAATGGAGAACAGCAGGAA-3′ R:5′-TCTTGCCAACCAGTTCCATTGC-3′	131

Table 3. Change in bacterial presence.

	Initial visit	6 months	2·5 years
A. actinomycetemcomitans	+	–	–
B. forsythus	+	–	–
C. rectus	+	+	+
E. corrodens	+	+	+
P. gingivalis	+	–	–

+, presence; –, absence.

same patient and, more importantly, can be used to determine changes in pathogen number in the same site over time [19]. *C. rectus*, *B. forsythus*, *E. corrodens*, *P. gingivalis* and *A. actinomycetemcomitance* were assessed in our study as the minimum number of strains of the periodontal pathogens needing to be investigated. There are sufficient data to consider *A. actinomycetemcomitance*, *B. forsythus*, and *P. gingivalis* as aetiologic agents in various forms of periodontal disease; *A. actinomycetemcomitance* is most often found in early onset periodontitis, whereas *P. gingivalis* and *B. forsythus* are found as or more frequently in adult onset periodontitis [22]. *C. rectus* and *E. corrodens* were chosen to assess the gingivitis as these have been frequently detected not only in gingivitis in adults and subjects with advanced periodontitis but also in child patients with gingivitis [18].

The bacterial findings throughout the study are presented in Table 3. Prior to initiation of the course of treatment, all five periodontal pathogens included in the study were present at one or more sites in the patient. The occurrence of *A. actinomycetemcomitance*, *B. forsythus* and *P. gingivalis* diminished at the 6-month follow-up. At the 2·5 year follow-up *C. rectus* and *E. corrodens* were still present, despite a significant decrease in GBI% and probing depth over the 2·5 years of treatment.

Discussion

A clinical and radiographic examination of 80 Swedish patients with Down's syndrome between 10 and 19 years of age showed that early periodontitis is frequently seen in patients with Down's syndrome as early as 11 years of age, and that the lesions are first diagnosed in the anterior mandibular region [15]. The patient in our study had not developed mobility of the mandibular incisors until 18 years of age. Mobility diminished clinically and the occurrence of alveolar bone loss did not increase during her subsequent 2·5 years of treatment. However, our follow-up period for radiographic evaluation of alveolar bone height was shorter than in previous studies [12,13]. Although not detected at the time, some radiographic evidence of alveolar bone loss was already present in this girl's lower incisors at 15 years when these were reviewed (data not shown). Progression to periodontitis in this patient might have been stopped sooner by starting a preventive course of treatment earlier. Probing depth decreased markedly during the first 6 months of preventive care, while GBI% decreased markedly after the 6 month follow-up. It appears that the index of probing depth as a measure in evaluating prevention of periodontitis is more sensitive than that of GBI%, suggesting that gingival inflammation expressed as GBI% might take longer to recover compared with probing depth.

Our management for this patient with Down's syndrome could not be compared to previous studies from the literature, as previous longitudinal investigations have reported only statistical averages, and little has been published on specific measures for Down's syndrome with periodontal disease [12–14]. Only one study [12] has shown an improvement of the gingival condition, which is probably due to the fact that more patients participated in preventive dental care at the follow-up compared to baseline during the 7-year period. The follow-up period in our study is limited, but the results suggest that clinical prevention by physical plaque control is possible in Down's syndrome patients and is effective not only for gingival inflammation but also for periodontal disease. Our treatment did not include chemotherapeutic agents and resective procedures, because all the periodontal pockets measured tended to decrease and bacterial profiles changed. If the bacterial examination had detected abnormal plaque status in spite of follow-up, other measures might well have been taken.

It is important to know which factors affect development of periodontitis in the patient in order to make plans for the periodontal management in patients with Down's syndrome. The factors that might enhance periodontitis include tooth morphological features such as short roots and an unfavourable crown/root ratio, dental crowding and inadequate oral hygiene. In the patient in our study, the upper left lateral incisor, which in this patient had translated completely to the palatal side of the left central incisor, had a pathological probing depth of 4 mm with bleeding even after 2·5 years of treatment. It would be difficult for probing depth adjacent to this translated

tooth to improve because almost no alveolar bone is present between this tooth and upper left central incisor. It was considered that ideally the tooth should be extracted as the best measure of periodontal management. However, the patient was happy with this tooth's position and it was therefore retained at the patient's request. Every effort needs to be made to prevent the progress of periodontitis in such a translated tooth through the supra- and subgingival scaling and oral hygiene instruction and the tooth retained as long as the status of the surrounding periodontium does not get worse. Periodic examination including occlusal assessment should be taken.

The improved condition of the gingivae, the significantly greater frequency of shallow pockets (commensurate with reduction in deep pockets), and the reduction in probing depth during the 2·5 year follow-up demonstrated the efficacy of subgingival plaque control in this patient, suggesting the possibility that subgingival plaque could be a critical factor in the progress of periodontitis in patients with Down's syndrome.

Patients with Down's syndrome have gingival inflammation associated with a high amount of plaque [14]. Lesser efficacy in supragingival plaque control may be a result of lesser degrees of patient cooperation. The degree of intellectual disability in the patient was moderate enough to allow her to understand oral hygiene instructions effectively, which is not always the case. Furthermore, her chances for effective plaque control were improved by living at home. Severity of periodontitis is higher in subjects living in institutions than in those living at home as a consequence of the better degree of plaque control achieved at home [23]. Other factors that might have enhanced periodontitis in the patient include traumatic occlusion in relation to an oversized mandible with a protrusive dentition, and mouth breathing with an open mouth and a protrusive, fissured tongue. These features are characteristic in patients with Down's syndrome, and in addition to the defective host immune responses, may indirectly contribute to the progress of periodontitis.

Because detection of putative pathogens is critical for delineating the aetiology and progression of periodontitis [24], eliminating periodontal pathogens was believed to be a valuable enhancement factor in our study. A decrease in DNA-positive pathogens during treatment in our study suggests that periodontal pathogens might be controlled by means of plaque control in Down's syndrome patients. This result is not consistent with findings in a previous report demonstrating that supragingival plaque control by itself could not produce any profound subgingival microbiological response. In this investigation, oral hygiene improved markedly after therapy lasting 4–12 weeks in a group of 11 patients with cerebral palsy (mean age: 36 years, with a range from 23 to 53 years) [14]. For our patient, professional subgingival plaque control as well as supragingival plaque control was carried out monthly, suggesting the possibility that this method may have controlled bacteria for a period lasting several months or more. After 6 months, *A. actinomycetemcomitans*, *B. forsythus* and *P. gingivalis* were no longer detected, but the other two pathogens, *C. rectus* and *E. corrodens*, remained at their initial levels even after 2·5 years. This was despite the fact that gingival inflammation, expressed as bleeding on probing, markedly improved between 6 months and 2·5 years. There are two possible explanations. Firstly, *C. rectus* and *E. corrodens* might more commonly occur in paediatric and adult gingivitis [18]. Secondly, *A. actinomycetemcomitance*, *B. forsythus* and *P. gingivalis* may be more sensitive to an improvement of periodontitis in Down's syndrome than the other two species. There is evidence that *B. forsythus* and *P. gingivalis* are detected more frequently in adult periodontitis, while *A. actinomycetemcomitance* is the primary pathogen in juvenile periodontitis [26], meaning that these three bacterial pathogens decrease when the intraoral environment changes as a consequence of preventive treatment such as subgingival scaling.

The early periodontitis seen in patients with Down's syndrome should be distinguished from early onset periodontitis (EOP), because the term 'early onset periodontitis' was coined to describe periodontal disease in young individuals who are otherwise healthy, eliminating systemic disease as being associated with the pathogenesis of periodontitis [26]. However, as far as the progression and severity of destruction is concerned, the oral manifestation of Down's syndrome is consistent with the Juvenile periodontitis (JP) disease pattern [27]. The patient's clinical features, involving rapid and severe bone loss surrounding several permanent teeth at the start of the study, resembled the generalized form of juvenile periodontitis (GJP). Defects in neutrophil chemotaxis are common findings in this condition, which most often occur during late teenage years or young adulthood [26]. The aetiology in GJP is still unclear, but *Porphyromonas gingivalis* has been

implicated as a putative pathogen [26]. The patient in our study had *Porphyromonas gingivalis* at the initial visit. However, the bacterial contribution in Down's syndrome may be different from that in GJP, as *Porphyromonas gingivalis* was not detected in 50% of patients with gingivitis and 23·1% of those with periodontitis in patients with Down's syndrome in a previous investigation [11]. In contrast to the clinical manifestations of generalized early onset periodontitis including GJP with the absence of plaque and the apparent lack of inflammation [28], the patient in our study demonstrated moderate inflammation of the gingivae. That may be a clinical characteristic in patients with Down's syndrome.

Summary/Conclusion

Our report emphasizes that preventive oral hygiene including subgingival plaque control must be considered of particular importance for children and young people with Down's syndrome.

Acknowledgements

We would like to express our sincere thanks and appreciation to Dr Mitoshi Kushiyama and Dr Kiyotaka Tomihisa for their help in the clinic and with the PCR experiments.

Résumé. Cet article décrit un cas de parodontite chez une jeune adolescente japonaise porteuse de trisomie 21. La patiente a reçu une série mensuelle de soins dentaires consistant en un contrôle mécanique de plaque et un enseignement de l'hygiène buccale. Après 2,5 ans la progression de la maladie parodontale a été arrêtée cliniquement et d'un point de vue microbiologique. La discussion de cet article aborde l'importance des soins cliniques lors de parodontites chez le patient porteur de trisomie 21.

Zusammenfassung. Ein Fall von Parodontitis bei einer japanischen Jugendlichen mit Down Syndrom wird vorgestellt. Die Patientin erhielt eine monatliche professionelle Zahnreinigung sowie Mundhygieneinstruktionen. Nach 2.5 Jahren war die Progression der Parodontalerkrankuung klinisch und mikrobiologisch zum Stillstand gekommen. Die Bedeutung der klinischen parodontologischen Betreuung von Patienten mit Down Syndrom wird diskutiert.

Resumen. En este informe se presenta un caso de periodontitis en una adolescente japonesa con síndrome de Down. La paciente recibía mensualamente un curso preventivo de cuidados dentales consistente en control mecánico de la placa e instrucción de la higiene oral. Después de 2,5 años se recuperó de la progresión de la enfermedad periodontal tanto clínica como microbiológicamente. Se discute la importancia del cuidado clínico de la periodontitis en los pacientes con síndrome de Down.

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