

Treatment of Generalised Aggressive Periodontitis: A 4-year Follow-up Case Report

Rui Fang LU¹, Li XU¹, Huan Xin MENG¹, Xiang Hui FENG¹, Kai Ning LIU¹

Aggressive periodontitis comprises a group of rare, often severe, rapidly progressive forms of periodontitis mostly characterised by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families. This case report presents a 16-year-old female patient with clinical and radiographic evidence of severe attachment loss, whose mother was also a patient with severe periodontal destruction. The girl was diagnosed with generalised aggressive periodontitis and received full-mouth scaling and root planing, bone graft surgeries and guided tissue regeneration on intrabony defects mesial of the mandibular first molars. Microbiological and immunological tests were performed on five selected sites before and at 2 months after initial therapy. Clinical and radiographic findings reported up to 4 years postoperatively indicated good effects and stability of treatment outcome.

Key words: *aggressive periodontitis, periodontal initial therapy, bone graft surgery, guided tissue regeneration*

Aggressive periodontitis (AgP) is a group of infrequent types of periodontal diseases. It affects systemically healthy individuals less than 35 years old, although patients may be older¹. If the generalised interproximal attachment loss affects at least three permanent teeth other than first molars and incisors of patients with AgP, it is usually diagnosed as generalised aggressive periodontitis (GAgP). AgP may be universally distinguished from chronic periodontitis by the age of onset, the rapid rate of disease progression, the nature and composition of the associated subgingival microflora, alterations in the host's immune response and a familial aggregation of diseased individuals². Periodontal treatment of these patients includes scaling and root planing, alone or in conjunction with systematic antibiotics, as well as surgical and interdisciplinary approaches. How-

ever, there are only a few reports of long-term follow-up of patients with AgP in China. The aim of this paper is to describe the clinical features of a case with AgP in China and the long-term stability of the results obtained with periodontal therapy.

Case report

In 2006, a 16-year-old female patient was referred to our department for periodontal examination. Her chief complaints were increasing mobility of the right anterior mandibular teeth for 3 months and gum bleeding on brushing for 5 years (Fig 1a). The patient was systematically healthy and a non-smoker. The periodontal status of her mother was poor; she had lost all four central incisors, left maxillary lateral incisor and both mandibular first molars due to advanced periodontal destruction, and alveolar bone loss of all teeth was more than two-thirds of the root length, evaluated by radiographic examination (Fig 2). The periodontal status of the patient's father was not severe, even though he had smoked three to five cigarettes per day for over 10 years. Periodontal examination of the father revealed no pocket depth more than 5 mm and for all teeth alveolar bone loss was less

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

Corresponding author: Dr Li Xu, Department of Periodontology, Peking University School and Hospital of Stomatology, #22 Zhongguansun Nandajie, Haidian District, Beijing 10081, P.R. China. Tel: 86-10-82195368; Fax: 86 10 62173402; E-mail: xulihome@263.net



Fig 1 a) Clinical front view of the patient at initial examination, showing intense inflammation and oedema of the gingiva and extensive plaque and dental calculus. b) Clinical front view of the patient at 4 years after periodontal treatment, showing that the periodontal inflammation was significantly reduced and gingival recession in the mandibular incisors.

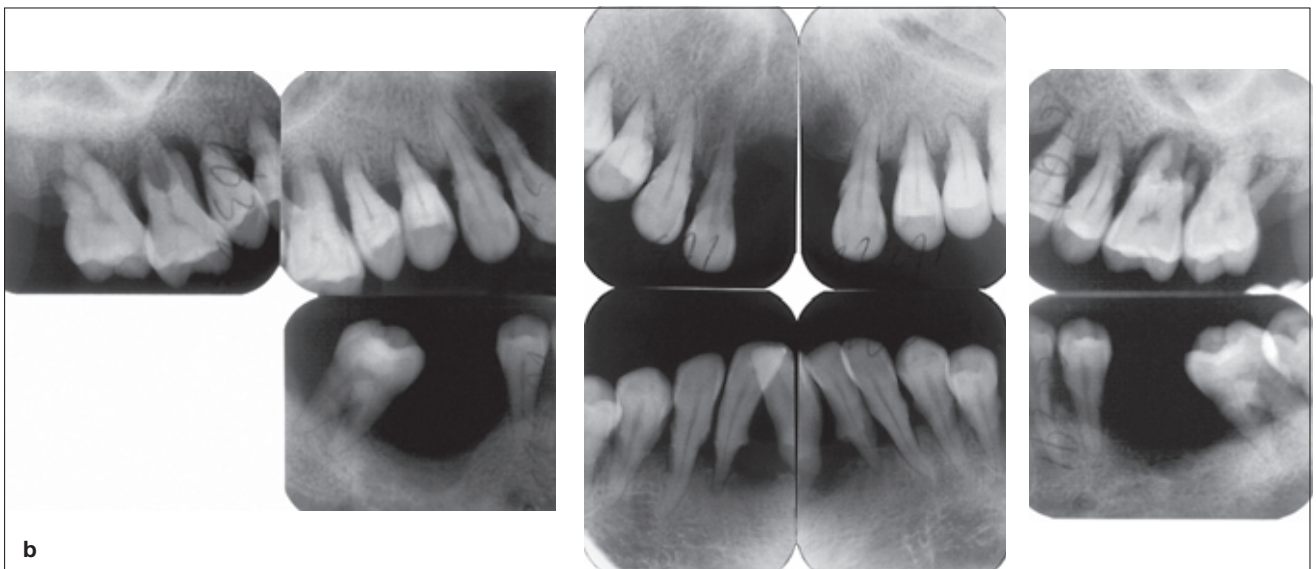


Fig 2 Clinical status of the patient's mother on first visit. a) Teeth 11, 21, 22, 31, 36, 41 and 46 had been lost and extensive dental calculus and gingival recession were observed. Clinical examination revealed severe attachment loss. b) Full-mouth periapical radiographs at initial examination revealed severe horizontal bone loss to the apical third of the root length. The prognosis for most of the teeth was hopeless.

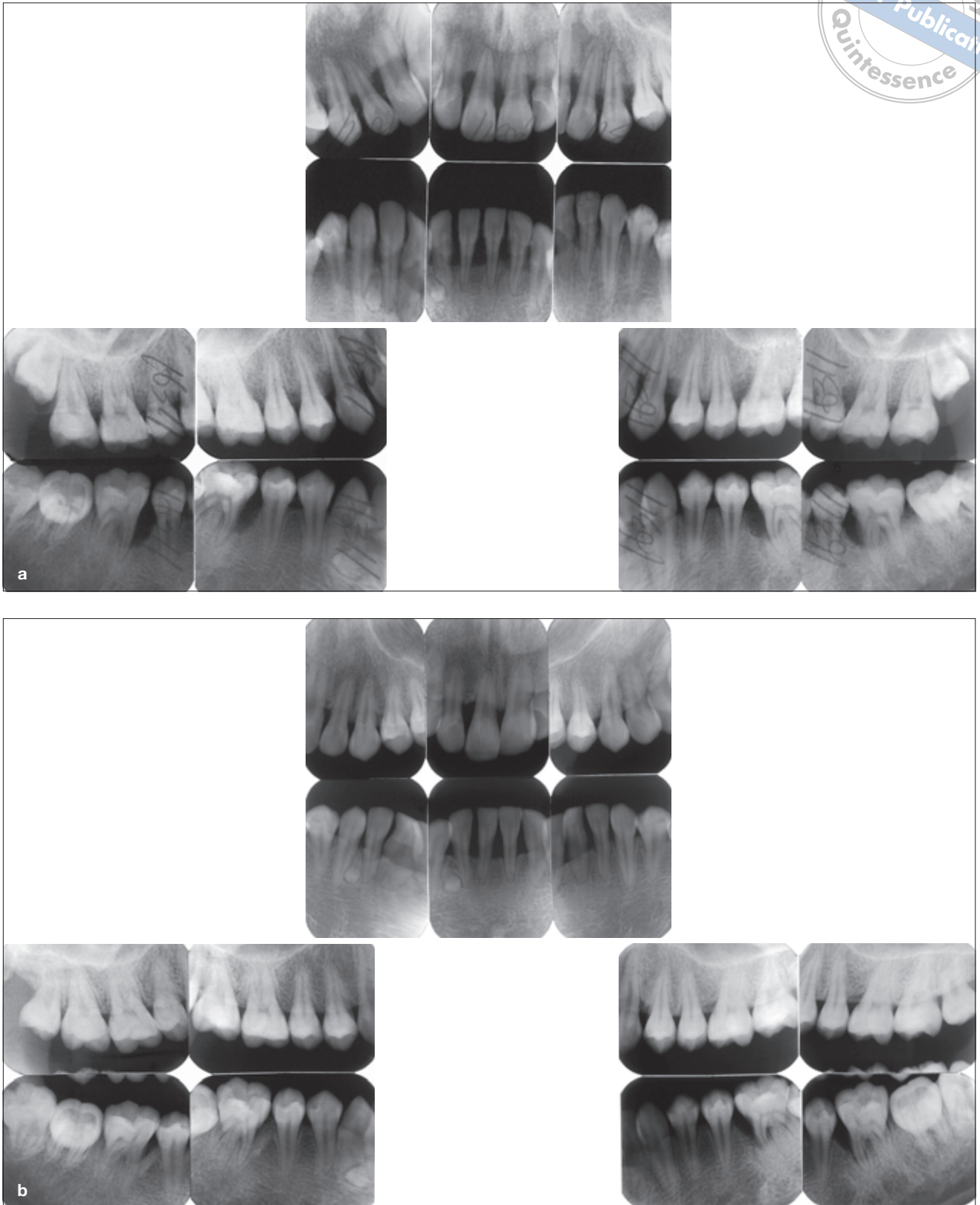
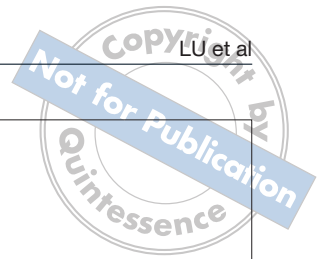


Fig 3 a) Full-mouth periapical radiographs at initial examination revealed horizontal bone loss up to half of the root length in incisors, angular bony defects mesial of the left and right mandibular first molars extending to about two-thirds of the root length, and fuzziness and a break in the continuity of the bone crest in the remaining teeth. b) Full-mouth periapical radiographs at 4 years after initial periodontal therapy. Note that the crests of interdental septa are clear and there is obvious bone fill mesial of the left maxillary first molar and both mandibular first molars.

than one-third of the root length. Informed consents for periodontal therapy and clinical sample collection were obtained from the patient and her parents.

Examinations of the patient included an intraoral examination, full-mouth probing depth (PD) and attachment loss (AL) at six sites around each tooth, tooth mobility, furcation involvements, full-mouth periapical radiographs and a complete blood count and biochemical analysis. The data collected revealed that the average PD of the full mouth was 4.8 ± 1.5 mm and average AL was 3.0 ± 0.9 mm. There was deep PD of 9 to 10 mm mesial of the left and right mandibular first molars, with grade I furcation involvement on the buccal side. Mandibular incisors demonstrated grade I to II mobility with PD of 5 to 9 mm. Radiographic examination revealed that the average relative bone height for all teeth was 76.6% [(bottom of defect mesial and distal to root apex / cemento-enamel junction (CEJ) to root apex) \times 100%]. The radiographs also revealed intrabony defects mesial of the left and right mandibular first molars, and that horizontal bone loss in the maxillary and mandibular incisors was about half the root length. Fuzziness and a break in the continuity of the lamina dura at the bone crest of the rest of the teeth were observed (Fig 3a). The blood test revealed a low albumin/globulin ratio, while blood glucose, alkaline phosphatase and other liver function tests produced normal results. Based on the medical history and examinations, the patient was diagnosed as GAgP.

The patient received periodontal initial therapy, including instruction in tooth brushing using the Bass method and the use of interdental brushes, and scaling and root planing of all teeth under local anaesthesia. The patient was recalled for maintenance care every 2 months and received prophylaxis scaling. If necessary, the patient was re-motivated and re-instructed in oral hygiene procedures.

Six months after the initial phase of periodontal therapy, a re-evaluation of the periodontal status revealed that oral hygiene was good, PD of most sites was 1 to 3 mm and the average PD of the mouth was 3.0 ± 0.6 mm. Deep pockets of 7 to 9 mm persisted mesial of both mandibular first molars (see Fig 5a). Flap surgeries in conjunction with bone grafts were deemed necessary for both mandibular first molars.

The left and right mandibular first molars were treated separately, according to the principles of bone graft surgery. Under local anaesthesia, full-thickness mucoperiosteal flaps were raised facially and lingually from the mandibular second premolars to the second molars and all granulation tissue was removed from the defects. A two to three-wall combination intrabony

defect was found mesial of both first molars. In the right first molar, the distance between the CEJ and the bottom of the defect was 8 mm and the distance between the CEJ and the approximal bone defect was 2 mm (Fig 4a). The root surfaces were conditioned with tetracycline for 3 minutes and subsequently rinsed thoroughly with sterile saline. The bone defects were filled with Bio-Oss Collagen® (Geistlich, Switzerland) to the alveolar crest (Fig 4b). Flaps were repositioned and closed with vertical internal mattress sutures and covered by periodontal dressings. The patient was advised to rinse with 0.12% chlorhexidine solution twice a day for 4 weeks and to take amoxicillin 0.5 g three times a day for 7 days. The sutures were removed after 14 days.

Recall appointments were once a week during the first month, and once every month during the following 5 months. Re-evaluation at 6 months after surgery showed that PD at the mesial sites of both mandibular first molars was 5 mm with bleeding on probing, and radiographic examination revealed only partially filled intrabony defects (Figs 5a and 5b). A second round of surgical therapy was planned, comprising bone graft surgery and guided tissue regeneration (GTR) for the left and right mandibular first molars, respectively. After full-thickness mucoperiosteal flap elevation and debridement, an intrabony defect was observed mesial of both teeth. In the right first molar, the distance between the CEJ and the bottom of the defect was 5 mm (Fig 4c), which was then completely filled with blood moistened Bio-Oss® and covered by Bio-Gide® membrane (Geistlich, Switzerland) (Fig 4d), the wound and membrane were securely covered with the mucoperiosteal flap. Radiographic examination at 6 months post second surgical therapy showed almost completely bone fill, and the original PD of 8 to 9 mm was reduced to 4 mm (Fig 5c).

The patient was then put into regular periodontal maintenance of every 2 to 3 months. Four years after initial therapy, periodontal examination revealed that the average PD was 2.9 ± 0.6 mm, the average AL was 3.4 ± 0.8 mm, with no PD greater than 4 mm (Fig 1b). A full-mouth series of radiographs showed that bone level was stable and obvious bone fill mesial of the left maxillary first molar and both mandibular first molars. The average relative bone height for all teeth was 88.2% (Fig 3b), with an increase in bone height of 8.4% (88.2% vs. 76.6%) compared with the radiograph taken before treatment (Fig 3a).

Gingival crevicular fluid (GCF) was obtained from the mesiobuccal sites of the right maxillary central incisor and first premolar, the left maxillary first molar and the left and right mandibular first molars before and at

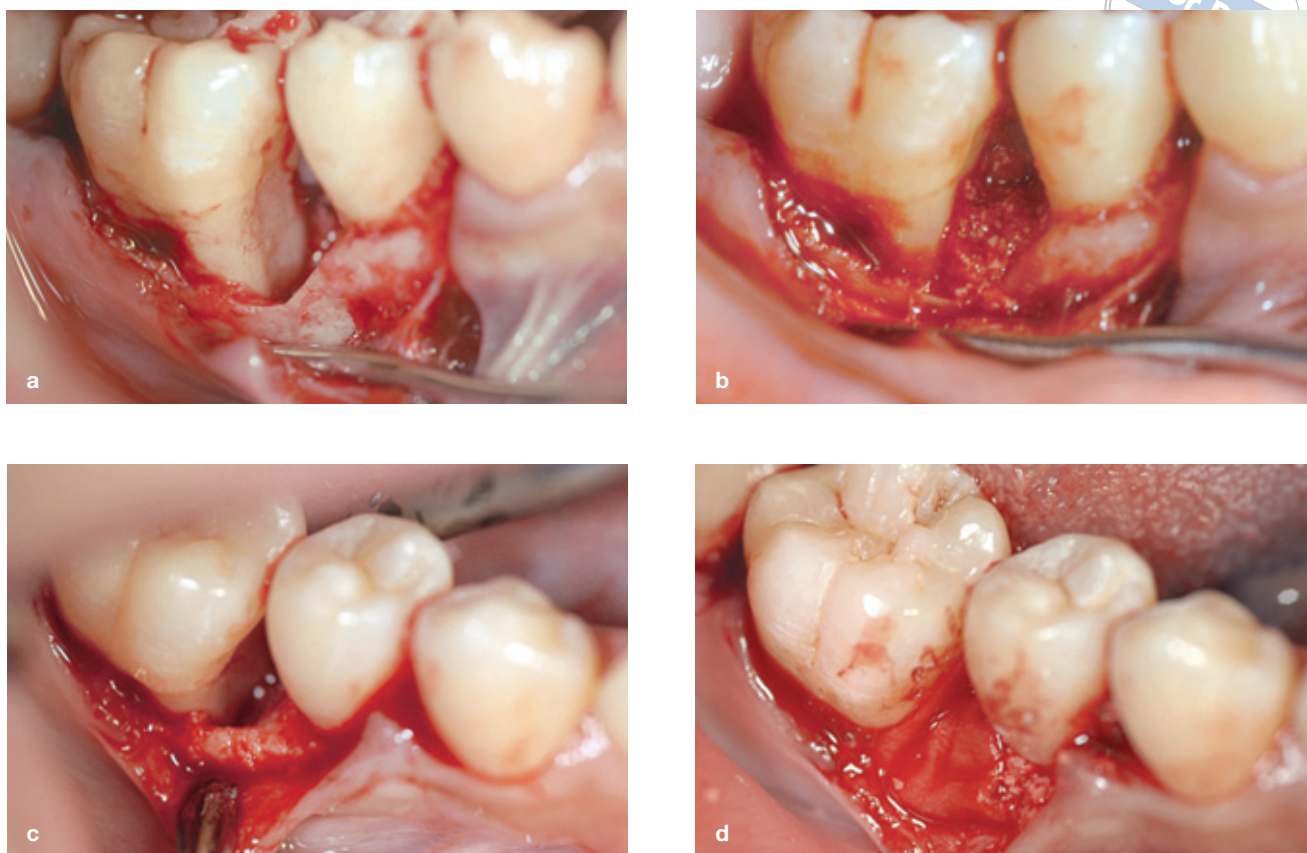


Fig 4 a) Clinical intraoperative view of the right mandibular first molar. b) The mesial intrabony defect was filled with Bio-Oss Collagen. c) Clinical intraoperative view at second surgical therapy (6 months after the first bone graft surgery). The mesial intrabony defect, which was narrower and shallower than at the first surgery (a), was filled with Bio-Oss and covered by Bio-Gide membrane (d).

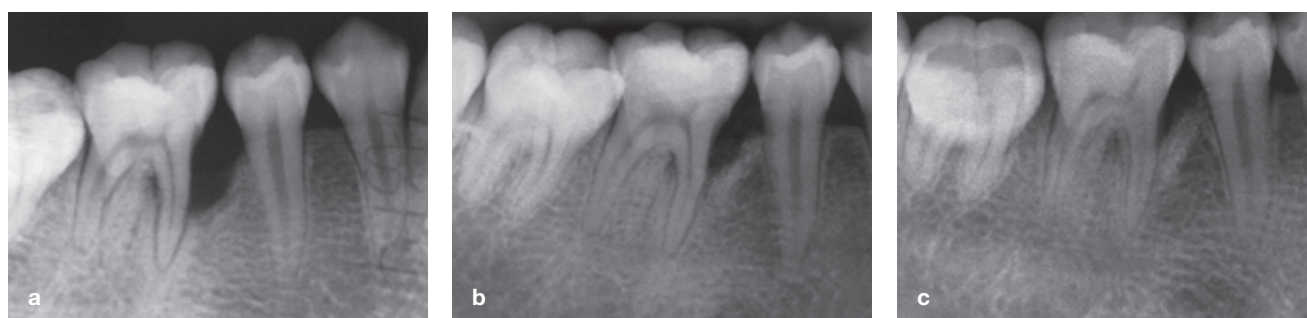


Fig 5 Long-cone paralleling technique. a) Radiograph of the right mandibular first molar before first surgery. b) Radiograph at 6 months after the first surgical bone graft procedure. c) Radiograph at 6 months after the second surgical bone graft and GTR procedure. Compared with the original situation (a), pronounced new bone formation mesial of the tooth can be observed.

2 months after periodontal initial therapy. Polymerase chain reaction amplification of *Porphyromonas gingivalis* (*Pg*), *Tannerella forsythia* (*Tf*), *Treponema denticola* (*Td*) and *Aggregatibacter actinomycetemcomitans* (*Aa*) was performed in the samples of GCF from the

right maxillary first premolar, the left maxillary first molar and the right mandibular first molar. Butyric acid, propionic acid, 25-hydroxyvitamin D₃ (25(OH)D₃) and interleukin-6 (IL-6) concentrations were analysed from the GCF supernatant (Tables 1 and 2).

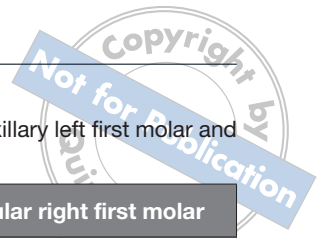


Table 1 The clinical and microbiological results for mesiobuccal sites of maxillary right first premolar, maxillary left first molar and mandibular right first molar at baseline and 2 months after periodontal initial therapy

	Maxillary right first premolar		Maxillary left first molar		Mandibular right first molar	
	Baseline	2 months	Baseline	2 months	Baseline	2 months
PLI	3	2	3	1	2	1
BI	4	4	4	2	4	1
AL (mm)	7	7	5	4	5	4
Pg	+	+	+	+	+	+
Tf	+	+	+	-	+	-
Td	+	+	+	-	+	-
Aa	-	+	-	-	+	-
Butyric acid (mmol/L)	2.45	2.41	5.65	0.71	3.5	0
Propionic acid (mmol/L)	8.76	11.39	10.89	3.54	13.53	0

PLI, plaque index; PD, probing depth; BI, bleeding index; AL, attachment loss; Pg, Porphyromonas gingivali; Tf, Tannerella forsythia; Td, Treponema denticola; Aa, Aggregatibacter actinomycetemcomitans.

Table 2 The clinical and immunological results for mesiobuccal sites of mandibular left first molar and maxillary right central incisor at baseline and 2 months after periodontal initial therapy

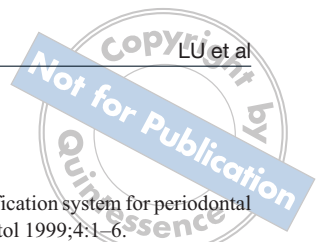
	Mandibular left first molar		Maxillary right central incisor	
	Baseline	2 months	Baseline	2 months
PLI	3	2	2	1
PD (mm)	8	7	5	3
BI	4	3	3	1
AL (mm)	7	7	4	3
25(OH)D ₃ (mmol/L)	6870	3093	20050	515
IL-6 (mmol/L)	3645	2591	10451	2926

PLI, plaque index; PD, probing depth; BI, bleeding index; AL, attachment loss; 25(OH)D₃, 25-hydroxyvitaminD₃; IL-6, interleukin 6.

Deep pockets in the mesiobuccal sites of the right maxillary central incisor and first premolar and left maxillary first molar reduced to no more than 4 mm at 2 months after initial therapy, meanwhile the microbiological testing showed that *Aa*, *Tf* and *Td* were negative in these sites (these periodontal pathogens had been positive before treatment). The concentration of butyric acid, propionic acid, 25(OH)D₃ and IL-6 also showed significant decrease in these sites (Tables 1 and 2).

Deep pockets in the mesiobuccal sites of the left and right mandibular first molars, which had intrabony

defects, did not reduce obviously after initial therapy. In the right first molar, *Pg*, *Td* and *Tf* were all detected before and at 2 months after initial therapy; *Aa* was negative before treatment, but was positive at 2 months after initial therapy. There was also no obvious reduction of butyric acid, 25(OH)D₃ and IL-6 concentrations in the GCF supernatant, but the propionic acid level increased for the left mandibular first molar at 2 months after treatment (Tables 1 and 2).



Discussion

This young female presented with the major common features of AgP, including non-contributory medical history, rapid attachment loss, bone destruction and familial aggregation³. Due to the lack of knowledge in oral healthcare, there was extensive plaque and dental calculus, much of which was very severe, which is quite characteristic of patients with AgP in China.

There may be some genetic or immunological factors that have not been found and so we can not change in patients with AgP⁴. Active periodontal therapy and periodontal maintenance at 3-month or shorter intervals are therefore essential for preventing further progression of the tissue destructive process⁵. GTR and bone graft procedures in periodontitis lesions represent exciting therapeutic modalities and have been shown to maintain stability for a long time, which was in accordance with other reports⁶.

It has been reported that many indicators, including putative periodontal pathogens⁷, cytokines⁸ and bacteria metabolic acids⁹, are associated with periodontal destruction. In this case, we chose some of these indicators and tested for them both before and after initial therapy. The results showed that the changes in the potential indicators were in correspondence with the clinical changes. In sites that responded well to the initial therapy, the indicators tested showed obvious reduction, while in sites that did not respond well to the initial therapy, the indicators tested showed no obvious reduction or even increased in concentration. What was interesting was that in the intrabony defect mesial of the right mandibular first molar, *Aa* recolonised after the initial periodontal treatment. This might indicate a recolonisation of *Aa* from other pocket sites or from the saliva after treatment¹⁰.

Within the limitation of this case report, no obvious reduction in the indicators was found to mean a relatively poor prognosis and that more active treatment may be needed, including flap surgery, adjunctive antibiotics and more frequent recalls. Active periodontal therapy and maintenance can lead to long-term success in patients with AgP.

References

1. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999;4:1–6.
2. Armitage GC, Cullinan MP. Comparison of the clinical features of chronic and aggressive periodontitis. *Periodontol* 2000 2010;53:12–27.
3. Novok KF, Novok MJ. Aggressive periodontitis. In: Crazza's Clinical Periodontology, ed 10. Missouri: Elsevier, 2006:506–512.
4. Meng H, Xu L, Li Q et al. Determinants of host susceptibility in aggressive periodontitis. *Periodontol* 2000 2007;43:133–159.
5. Rosalem W, Rescala B, Teles RP et al. Effect of non-surgical treatment on chronic and aggressive periodontitis: Clinical, immunologic, and microbiologic findings. *J Periodontol* 2011;82:979–989.
6. Sant'Ana AC, Passanezi E, Todescan SM et al. A combined regenerative approach for the treatment of aggressive periodontitis: Long-term follow-up of a familial case. *Int J Periodontics Restorative Dent* 2009;29:69–79.
7. Teles RP, Gursky LC, Favari M et al. Relationships between subgingival microbiota and GCF biomarkers in generalized aggressive periodontitis. *J Clin Periodontol* 2010;37:313–323.
8. Liu K, Meng H, Lu R et al. Initial periodontal therapy reduced systemic and local 25-hydroxy vitamin D(3) and interleukin-1beta in patients with aggressive periodontitis. *J Periodontol* 2010;81:260–266.
9. Tsuda H, Ochiai K, Suzuki N et al. Butyrate, a bacterial metabolite, induces apoptosis and autophagic cell death in gingival epithelial cells. *J Periodontol Res* 2010;45:626–634.
10. Johnson JD, Chen R, Lenton PA et al. Persistence of extracrevicular bacterial reservoirs after treatment of aggressive periodontitis. *J Periodontol* 2008;79:2305–2312.