

**Introduction**

Aggressive periodontitis (AP) is a rare form of periodontitis that may be localised (lAP) or generalised (gAP) depending on the clinical and laboratory findings. According to the 1999 international classification workshop, a case may be diagnosed as AP if it presents with the following major common features:

- non-contributory medical history;
- rapid attachment loss and bone destruction;
- familial aggregation of cases.

The secondary features that are generally present but may not be seen in a case with AP include:

- amount of microbial deposits inconsistent with the severity of periodontal destruction;
- elevated proportions of *Actinobacillus actinomyces*comitans (Aa);
• phagocyte abnormalities;
• hyper-responsive macrophages, producing increased PGE\(_2\) and IL\(_1\)β;
• in some cases, self-arresting disease progression.

gAP represents the most heterogeneous group and includes the most severe forms of periodontitis. Earlier described as generalised juvenile periodontitis, gAP may be diagnosed based on the following findings:\(^2\):

- usually affecting persons under 30 years of age (however, may be older);
- generalised interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors;
- pronounced episodic nature of periodontal destruction;
- poor serum antibody response to infecting agents.

Some patients of gAP may present with systemic manifestations such as weight loss, depression and general malaise\(^3\).

In most of the cases of gAP there may be severe acute gingival inflammation associated with bleeding on probing; gingival tissues that appear pink, free of inflammation and occasionally with some degree of stippling may be noticed\(^4\). Gingival enlargement is a rare finding in a case of gAP. The following report will present a case of gAP with gingival enlargement and will discuss its periodontal management.

### Case presentation

The patient was a 15-year-old girl who consulted for the treatment of recurrent gingival enlargement, increased spacing and mobility in her teeth. Although she complained of malaise, weakness and gastric upset, her medical history revealed no documented systemic problems. There was no history of drug intake. She did not smoke or drink alcohol and her family history did not reveal any significant information related to her present condition. Episodes of gingival enlargement had occurred in the last year but resolved after professional oral prophylactic treatment without complication. On clinical examination, the patient looked weak and anxious. There was no evidence of jaundice or cyanosis although mild pallor was noticed on examining her sclera. A complete haematological examination was advised, which revealed no abnormality. Increased Aa antibody titres are anticipated for cases of AP but were absent in this case. Oral evaluation revealed generalised gingival enlargement, the enlarged tissue was firm, resilient and pink with mild signs of inflammation. The enlargement was greater in the lower anterior region of the mouth. A peculiar finding was that the gingiva on the lingual aspect of her teeth was relatively normal. Dental examination revealed marked spacing between anterior teeth in both maxilla and mandible, with Grade-II mobility in all
the teeth except lower central incisors, which were Grade-III mobile. Probing depth (PD) ranged from 5 to 12 mm and was larger in molar areas and the mandibular anterior region of the mouth. Periodic panoramic radiographic examination revealed progressive generalised bone loss throughout the entire dentition. Microbiological examination revealed elevated levels of Aa.

### Treatment

Periodontal treatment planning, including gingivectomy for the gingival enlargement, was explained to the patient. The patient was given overall instructions in oral hygiene techniques. A session of scaling and root planing was performed for the entire mouth. After the initial treatment, gingivectomy was performed under local anaesthesia, along with extraction of her mobile mandibular incisors, which were later replaced using a removable partial denture. Histopathological examination of excised gingival tissue revealed hyperplastic-stratified squamous epithelium with underlying fibrocollagenous tissue infiltrated by dense lymphoplasmacytic infiltrate. These findings, when correlated with the age and clinical findings, strongly supported the diagnosis of pubertal gingival enlargement associated with AP. Two weeks after gingivectomy was performed, the patient was put on a weekly recall visit for another 4 weeks. In each visit thorough scaling and root planing was carried out, with emphasis on maintenance of strict oral hygiene. After 6 weeks the case was reassessed and there were still deep pockets ranging from 8 to 10 mm. The decision was made to gain access to deep lesions with a modified flap operation and adjunctive antimicrobial therapy was started with a combination of metronidazole and amoxicillin. The therapy consisted of 250 mg metronidazole plus 375 mg amoxicillin, both 3 times a day, for a period of 7 days. One week after surgery, sutures were removed and the patient was asked to return at 3-month intervals for the maintenance phase.

After one year of starting the treatment, marked improvement in the patient's condition was noticed. The pocket depths ranged from 2 to 3 mm and gums were in a healthy condition.
Discussion

This paper reports a case of gingival enlargement seen in a patient with gAP. Gingival enlargement may be associated with many conditions, including pregnancy, puberty, vitamin C deficiency, plasma cell gingivitis, and non-specific gingival enlargement (granuloma pyogenicum). Other types of enlargements include inflammatory (chronic/acute), drug-induced, leukaemic, granulomatous, neoplastic, and idiopathic gingival fibromatosis. The differential diagnosis is based on the history of the patient, clinical features and histopathological examination of the enlarged gingival tissue. In the present case the patient was a teenager who had a history of recurrent episodes of gingival enlargement in the previous year. Based on the clinical presentation of enlarged tissue along with the age, negative medical, familial and drug history of the patient, the case was diagnosed as gingival enlargement associated with puberty. Although gingival enlargement during puberty has all the clinical features generally associated with chronic inflammatory gingival enlargement, it is the degree of enlargement and the tendency to develop massive recurrence in the presence of relatively scant plaque deposits that distinguish pubertal gingival enlargement from uncomplicated chronic inflammatory enlargement. Clinically, gAP is characterised by ‘generalised interproximal attachment-loss affecting at least three permanent teeth other than first molars and incisors’1. This destruction is followed by stages of quiescence of variable length (weeks to months to years). Radiography often shows that bone loss has progressed since the previous evaluation4. Although all these mentioned features were identifiable in the present patient7, some of the primary and secondary features were absent in this patient. However, it has been agreed that not all the listed primary and secondary features are necessary in order to assign an AP diagnosis, and that the diagnosis may be based on clinical, radiographic, or historical data alone. Although helpful, laboratory testing may not be essential for diagnosis1. In the present case, clinical findings were supported by radiographic and microbiological data, which strongly suggested the diagnosis of gAP.

The treatment plan included gingivectomy for management of enlarged gingiva; the treatment for gAP was directed towards elimination and suppression of infecting microorganisms and providing an environment conducive to long-term maintenance. Use of antibiotics has been suggested as a rational compliment to mechanical debridement and the decision to use a combination of metronidazole and amoxycillin was taken because metronidazole in combination with amoxycillin may suppress Aa more effectively than a single antibiotic regime8,9. Extraction of incisors was unavoidable due to their poor prognosis. A removable partial denture was chosen to replace the extracted teeth as a fixed prosthesis was not possible due to poor bone support in the abutment teeth. Successfully treated cases of gAP may subsequently show signs of relapse10. With this in mind the patient was instructed to report every 3 months for oral hygiene reinforcement and scaling.

Conclusions

Early diagnosis is the key to prevent rapid destruction of periodontal tissues in gAP. A clinician should be
aware of the fact that associated conditions like gingival enlargement may complicate the diagnosis of gAP. Therefore, diagnosis should always be based on detailed study of the patient’s history, and clinical, radiographic and microbiological findings.

References