Disease Staging Index for Aggressive Periodontitis

Srinivas Sulugodu Ramachandra[a] / José Dopico[b] / Nikos Donos[c] / Luigi Nibali[d]

Summary: Major advances in the knowledge about the aetiopathogenesis of aggressive periodontitis (AgP) have been achieved. An ever increasing number of scientific articles related to AgP are published every year contributing significantly to the knowledge of this unique and complex disease. AgP has been classified into localised and generalised forms based on their extent and disease progression with distinct clinical and radiological features. A classification of AgP based on severity (mild, moderate and severe) exists; however, it is not easily applicable. Therefore, studies on AgP do not categorise the disease based on severity. A disease staging index for AgP is proposed based on clinical and radiological features, as well as risk factors. Based on the presence or absence of risk factors confirmed by longitudinal studies, cases of AgP can be divided into low risk, medium risk and high risk profiles for disease progression. Clinicians can devise a broad treatment plan for their AgP cases based on this staging. More frequent recall intervals are proposed for patients at medium and high risk for disease progression. Ten cases of AgP with 10-year follow-up were used to validate the staging index by retrospectively assigning prognosis and associating it with tooth loss. The use of this staging by researchers would increase external validity of research on AgP. Long-term analysis of AgP cases are needed to validate this staging index longitudinally.

Key words: aggressive periodontitis, disease staging, index, prognosis, risk factors, severity, treatment.

Aggressive periodontitis (AgP) is a distinct type of periodontitis that shows familial aggregation of cases and a rapid rate of disease progression in systemically healthy individuals. AgP is classified into localised and generalised forms, based on their extent and disease progression.[5] Great advances in the knowledge of the aetiopathogenesis of AgP have been achieved, and research on AgP continues to be conducted by researchers and clinicians. Armitage et al[6] expressed the need for a periodontal research community to develop and then widely use valid and acceptable case definitions for periodontal diseases. They also suggested that unambiguous case definitions are needed if further progress is to be made in the understanding of the aetiology, pathogenesis and management of AgP.[7] Highfield[25] anticipated that as we learn more about the aetiology and pathogenesis of periodontal diseases, future revisions to the classification will be needed. These revisions or modifications help differentiate the disease better, either for the benefit of the patient or for research purposes. The objectives of this article are to propose a staging index for AgP, stage the cases at baseline and after treatment, retrospectively assign the prognosis, and validate the staging index based on tooth loss. Justification for the features included in the index, its advantages and limitations are also discussed.

EARLIER CLASSIFICATION SYSTEMS AND THEIR DRAWBACKS

In 1927, Gottlieb referred to the destructive periodontal disease seen in children and young adults as 'diffuse atrophy of the alveolar bone' and 'deep cementopathia'.[18,19] Orban and Weinmann[47] coined the term 'periodontosis' to describe severe periodontal disease in young individuals. Baer[9] broadly classified AgP (then still known as periodontosis) into early and advanced stages, and suggested clinical and radiological signs for the two stages. In early stages, the gingiva has a normal colour and physiologic

[a] Associate Professor, Faculty of Dentistry, SEGi University, Malaysia. Idea, hypothesis, wrote and proofread the manuscript, contributed substantially to discussion.

[b] Clinical Lecturer, Periodontology Unit and Division of Clinical Research, UCL Eastman Dental Institute, London, UK; Periodontology Unit, Universidad de Santiago de Compostela, Santiago de Compostela, Spain. Wrote and proofread the manuscript, contributed substantially to discussion.

[c] Professor, Clinical Oral Research Centre, Institute of Dentistry, Queen Mary University London, London, UK. Wrote and proofread the manuscript, contributed substantially to discussion.

[d] Senior Clinical Lecturer, Periodontology Unit and Division of Clinical Research, UCL Eastman Dental Institute, London, UK; Clinical Oral Research Centre, Institute of Dentistry, Queen Mary University London, London, UK. Idea, hypothesis, wrote and proofread the manuscript, contributed substantially to discussion.

Correspondence: *Associate Professor S. Ramachandra, Faculty of Dentistry, SEGi University Sdn.Bhd. No.9, Jalan Teknologi, Taman Sains, Kota Damansara, PJU 5, 47810 Petaling Jaya, Selangor, Darul Ehsan, Malaysia. Tel: +60-0111-541-7228; Email: periosrinivas@gmail.com*

contour. Baer also noted that cases in early stages are diagnosed accidentally during routine dental examinations.[9] Migration and loosening of the teeth were considered as late clinical signs of the advanced stages of the disease.[9] Radiographically, the early stages had angular defects, whereas the late stages had more horizontal bone loss.[9] Manson and Lehner[36] classified AgP (then known as juvenile periodontitis) into two groups. The group of patients between the ages of 14 and 21 years were referred to as having 'juvenile periodontitis'. The older group between 22 to 29 years was called 'post-juvenile periodontitis'. A higher the number of teeth involved, a higher periodontal index and decreased bone loss score were noted in post-juvenile periodontitis patients. This implied a slower rate of bone destruction in older patients.[36] Page and Schroeder[48] identified pre-pubertal, juvenile periodontitis and rapidly progressive periodontitis among five distinct forms of periodontitis. Bial and Mellonig[11] screened 49,380 male naval recruits for juvenile periodontitis. Among them, 182 patients were identified as having juvenile periodontitis, and were categorised as follows: Type I bone loss involving first molars and/or incisors and up to two additional teeth; Type II involving first molars/incisors and several additional teeth; and Type III with generalised involvement (more than 14 teeth) but with bone loss notably more extensive on the first molars and/or incisors.[11]

In 1989, the AAP grouped pre-pubertal periodontitis, juvenile periodontitis and rapidly progressive periodontitis affecting young individuals under the term 'early onset periodontitis' (EOP).[3] The 1993 European classification was along similar lines but more simplified.[8] The 1989 AAP[3] and the 1993 European classification[8] were age dependent. These classifications led to confusion for diagnosing young patients presenting with chronic periodontal disease (which could be diagnosed as juvenile periodontitis or rapidly progressive periodontitis). Also, adult patients presenting with classical signs and symptoms of juvenile periodontitis could be diagnosed with juvenile periodontitis or adult periodontitis, causing confusion. Due to these issues, in 1999 the AAP organised an international workshop in order to develop a new classification for periodontal diseases.[5] The workshop proposed a new classification based on the present knowledge of the pathogenesis of periodontal disease and discarded age-dependent criteria.[5] Diseases such as juvenile periodontitis and rapidly progressive periodontitis were grouped in a new category named AgP. Thus, the term EOP was replaced by the term AgP. The obligatory criteria for diagnosis of AgP are non-contributory medical history, rapid loss of attachment and familial aggregation of cases. The facultative criteria are discrepancy between the amount of microbial deposits and severity of periodontal destruction, progression of attachment loss, possibly self-arresting bone loss, elevated proportions of Aggregatibacter actinomycetemcomitans, and phagocytic abnormalities.[4,5]

Researchers at the 1999 AAP workshop agreed that all classification systems have inconsistencies or inaccuracies.[5] The workshop agreed that the classification should not be regarded as a permanent structure and must be adaptable to change and evolve with the development of new knowledge.[5] The 1999 AAP classification subgrouped AgP into localised and generalised types based on extent, disease progression and identification of certain specific features.[4] Localised AgP involves deep periodontal pockets around first molars and incisors, with distolabial migration of the incisors. Radiographically, localised AgP shows arc-shaped bone loss extending from the distal surface of the second premolar to the mesial surface of the second molar. Generalised AgP involves deep periodontal pockets in three or more teeth besides the first molars and incisors. Radiographically, generalised AgP shows generalised interproximal bone loss. However, the 1999 AAP classification did not introduce any subgroups based on disease severity, probably based on the assumption that all AgP cases would be severe by definition.[4,32] It was later suggested that cases of AgP can be classified into slight (mild), moderate and severe based on clinical attachment loss (CAL).[7] However, most cases of AgP are diagnosed with CAL more than 5 mm, so all cases are severe by default. Moreover, mild and moderate subgroups cannot be easily detected in AgP, since these cases are usually not diagnosed at an early stage. This is reflected in the fact that few studies exist in the periodontal literature in which cases of AgP have been classified based on severity.[7] This classification leaves some scope for personal interpretation; hence, later attempts were made to establish more reliable criteria.[6,16] Demmer and Papapanou[16] highlighted the use of the following reliable criteria for diagnosis of AgP: in ages ≤ 25 years, the presence of ≥ 2 interproximal, non-adjacent sites with ≥ 4 mm attachment loss occurring at a minimum of two different teeth, accompanied by bleeding on probing, signifies aggressive periodontitis. In ages 26 to 35 years, a diagnosis of aggressive periodontitis would require the presence of ≥ 2 interproximal, non-adjacent sites with ≥ 6 mm attachment loss occurring at a minimum of two different teeth, accompanied by bleeding on probing.[16] The question remains whether AgP and chronic periodontitis are indeed different pathologic entities.[16,42,50,44] Based on these principles, the need for disease staging criteria for AgP for clinical and especially research purposes appears clear, as recently suggested.[51]

RATIONALE FOR A DISEASE STAGING INDEX

Measuring disease severity helps evaluate the diagnostic efficacy of clinicians, assess quality of care, understand the utilisation of health services, design clinical trials, and reimburse hospitals on the basis of output.[20] Staging is a method for measuring the severity of specific, well-defined diseases. Staging defines discrete points in the course of individual diseases that are clinically detectable, reflect the severity in terms of risk of death or residual impairment (tooth loss in terms of periodontal disease), and possess clinical significance for prognosis and choice of therapeutic modality.[20] The concept behind staging is borrowed from clinical medicine, specifically oncology. During the course of neoplastic diseases, there are discrete stages that can be

defined and detected clinically, which reflect the severity of the disease, and have clinical significance for prognosis and choice of therapeutic modality. This concept has now been applied to other medical and surgical areas to classify many diseases.[20] Staging is based on a conceptual model of the disease process itself rather than on the relative efficacy of medical technology.[20]

The long-term prognosis of AgP cases is probably influenced by the timing of the diagnosis. In other words, early detection and treatment of AgP could result in better treatment outcomes than for cases detected after further disease progression has occurred. A systematic review[45] on tooth loss in AgP identified only one published paper[21] which reported associations of diagnosis staging, expressed as severity of initial bone loss, with tooth loss. That study showed an association between initial 'hopeless' prognosis (based on bone loss > 70%) and subsequent tooth loss.[21] Most of the studies in the review reported average clinical and radiographic parameters and did not use categorical variables indicating severity.[45] Since establishing the age of onset is difficult in the absence of previous records, a disease staging index could be the most practical means of obtaining a homogeneous, measurable outcome to compare across different studies.[46] In certain situations, cases of AgP have been referred to specialists or referral centers by general dental practitioners after non-surgical therapy has been completed. A disease-staging index becomes invaluable in the absence of patient records that could give details on the duration or onset of the disease.

The present authors propose a subject-based system for staging AgP cases, which should be validated by independent research studies having disease progression as an outcome. These features do not include the number of affected sites or teeth, and thus may apply to both localised AgP and generalised AgP. Although localised AgP and generalised AgP may have different features, it has been shown that untreated localised AgP cases may develop into generalised AgP, and disease staging criteria may be used for both conditions.[12] This staging could be used once the AgP diagnostic criteria described by Lang et al[32] have been confirmed. The items to classify subjects have been chosen based on the papers included in a recent systematic review on long-term follow-up of AgP cases.[45] CAL, initial bone loss and smoking seem to be the factors invariably associated with response to therapy, progression and risk of tooth loss in AgP. Initial tooth prognosis based on CAL and bone loss was associated with tooth loss.[21] Schenkein et al[52] reported smokers with EOP had greater CAL compared to non-smokers. Al-Habashneh et al[2] assessed the relationship between smoking and AgP, finding a higher mean CAL in smokers with AgP compared to non-smokers with AgP. Furthermore, smoking has been consistently associated with poorer treatment response and progression of AgP cases.[10,26,27,29] Another patient-based factor associated with AgP progression is positive family history.[1] Invariably, compliance to treatment and maintenance therapy remain the most important predictors of long-term stability.[45]

PROPOSED AgP STAGING SYSTEM

AgP progresses from the initial to the advanced stages as mentioned below. Table 1 summarises the proposed staging of AgP based on clinical and radiological features, as well as the presence or absence of known risk factors.

Stage I (Fig 1): Stage I AgP has at least two sites with CAL > 5 mm and bone loss of < 50% as discriminant clinical and radiologic features, respectively. Further features often seen in Stage I cases are the presence of vertical bony defects,[9] although no teeth would have been lost due to AgP yet. Patients without risk factors are categorised as stage IA with lower risk of disease progression. Patients with risk factors such as smoking and a positive family history are categorised as Stage IB with a moderate risk of disease progression. The treatment plan for Stage IA cases in the presence of active periodontal pockets would normally involve oral hygiene instructions (OHI), scaling and polishing, root surface debridement with adjunctive systemic antimicrobial therapy followed by recall intervals of 6 months. The treatment plan for Stage IB cases in the presence of active periodontal pockets involves smoking cessation advice (when applicable) along with the previously mentioned treatment approaches and shorter recall intervals of three to six months. Stage I cases are diagnosed only by comprehensive periodontal examination, which stresses the importance of periodontal probing during examination and diagnosis.

Stage II (Figs 2 and 3): Stage II AgP has at least two sites with CAL > 6 mm and radiographic bone loss between 50% and 70%. Facultative features are pathological tooth migration, especially of anterior teeth with concomitant diastema formation. Up to three teeth may already have been lost due to periodontal disease, or up to three teeth may have a hopeless prognosis. Radiographs show a combination of horizontal and vertical defects. The absence of risk factors would place the patient in Stage IIA with a moderate risk of disease progression. The presence of risk factors such as smoking and positive family history would place the patient in Stage IIB with a high risk of disease progression. The treatment plan for Stage IIA patients with active periodontal pockets involves OHI, scaling and polishing, root surface debridement with adjunctive systemic antimicrobial therapy, possible periodontal surgeries (regenerative therapy based on bony defects) and orthodontic treatment if spontaneous correction of migrated teeth does not occur after periodontal therapy, replacement of lost teeth (if any), and recall intervals of 3 to 6 months. The treatment plan for Stage IIB patients with active periodontal pockets involves shorter recall intervals of three months and smoking cessation counseling, along with the previously mentioned treatment approach.

Stage III (Fig 4): Stage III AgP has at least two sites with CAL > 8 mm and radiographic bone loss over 70%. Facultative features seen are pathological tooth migration leading to crowding and extrusion of the involved teeth. The pattern of bony destruction is predominantly horizontal. There could be loss of tooth vitality due to primary periodontal involvement (secondary endodontic involvement). More than three teeth may have already been lost due to periodontal dis-

Table 1 The proposed disease staging index for aggressive periodontitis

Stages	Discriminant clinical and radiological features	Facultative features	Sub-group	Risk factors	Risk of disease progression	Broad treatment plan (only when clinical indications such as periodontal pocket depth are present)
Stage I	At least 2 sites with CAL >5 mm, radiographic bone loss < 50%	No teeth lost due to periodontal disease. Predominantly shallow vertical bony defects compared to horizontal bone defects	Stage I A (Fig 1)	Non-smoker, negative family history	Low	OH instructions, education and motivation, scaling and polishing, root surface debridement, adjunctive antimicrobial therapy. Recall intervals of six months.
			Stage I B	Smoker or positive family history	Moderate	As above plus smoking cessation advice, shorter recall intervals of 3-6 months.
Stage II	At least 2 sites with CAL > 6 mm; maximum radiographic bone loss: 50%–70%	Pathological tooth migration, especially of anterior teeth with concomitant diastema formation. Up to three teeth lost due to periodontal disease or up to three teeth with hopeless prognosis. Combination of angular defects and horizontal defects (cases of localised AgP may have classical mirror image appearance)	Stage II A (Fig 2, Fig 3)	Non-smoker, negative family history	Moderate	OH instructions, education and motivation. Extraction of teeth with hopeless prognosis, scaling and polishing, root surface debridement, adjunctive antimicrobial therapy, possible periodontal surgery, orthodontic treatment if spontaneous correction of migrated teeth does not occur after periodontal therapy, replacement of lost teeth. Recall intervals of 3–6 months.
			Stage II B	Smoker or positive family history	High	As above plus smoking cessation advice. Shorter recall intervals of 3 months.
Stage III	At least 2 sites with CAL > 8 mm and bone loss ≥ 70%. Radiographic bone loss more than 70%.	Pathological tooth migration leading to crowding and extrusion of the involved teeth. Loss of vitality of some teeth (secondary endodontic involvement). Pattern of bone destruction prevalently horizontal. More than three teeth lost due to periodontal disease or more than three teeth having hopeless prognosis.	Stage III A (Fig 4)	Non-smoker, negative family history	Moderate	OH instructions, education and motivation, extraction of teeth with hopeless prognosis, scaling and polishing, root surface debridement, adjunctive systemic antimicrobial therapy. Endodontic treatment for non-vital teeth before periodontal surgery, replacement of lost teeth. Patient to be placed under maintenance with short recall intervals of 3 months.
			Stage III B	Smoker or positive family history	High	As above, plus smoking cessation advice. Patient to be placed under maintenance with short recall intervals of 3 months.

CAL: clinical attachment level; OH: oral hygiene. Smokers are considered such if they smoke at least 10 cigarettes/day on average.
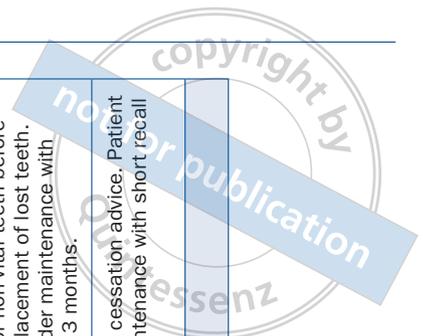
ease or more than three teeth have a hopeless prognosis. Owing to the presence of more than 70% bone loss, Stage III cases (both A and B) should be considered as cases with a high risk of disease progression and recalled at short intervals of 3 months. The treatment plan for Stage IIIA cases with active periodontal disease involves OHI, scaling and polishing, root surface debridement with adjunctive systemic antimicrobial therapy, extraction of teeth with a hopeless prognosis, endodontic treatment for nonvital teeth before periodontal surgery and replacement of lost teeth. The treatment plan for Stage IIIB patients with active periodontal pockets involves smoking cessation counseling along with the previously mentioned treatment approach.

INITIAL VALIDATION OF THE PROGNOSTIC SYSTEM

In order to assess the ease of diagnosis staging and the extent to which the prognosis system is associated with stability, 10 cases of AgP with a 10-year clinical and radiographic follow-up were included and staged using the proposed staging. The clinical study had received ethical approval by the National Research Ethics Service Committee, London (ref 13/LO/0874), and each subject had signed informed consent. All cases had received comprehensive periodontal treatment at the Eastman Dental Hospital in London and had then been referred back to their general dentist for periodontal maintenance. Staging was done retrospectively using the baseline clinical and demographic data by two authors (SR and LN) unaware of the 10-year follow-up status. When clinical and radiographic data at the 10-year follow-up were assessed, the risk of progression of the cases was categorised into low, moderate and high. Teeth lost during supportive periodontal therapy were noted. The agreement rate observed for the staging between the two authors was 100%, which showed the calibration of the researchers for baseline definitions. Among the 10 AgP cases, 6 cases had a high risk of disease progression, 3 cases moderate, and one had a low risk of disease progression. During supportive periodontal therapy period (10-year follow-up), tooth loss was noticed in two cases out of six with high risk of disease progression. Tooth loss was noticed in one case out of three with moderate risk of disease progression. No tooth loss was observed in the only case with low risk of disease progression. Table 2 shows the segregation of 10 AgP cases based on the proposed disease-staging index for AgP followed up for 10 years. The justification for the proposed staging follows below.

DISCUSSION

Flexibility in diagnosis is acceptable for clinicians, especially if there is not much difference in the treatment to be rendered (i.e. nonsurgical therapy with adjunctive antibiotics and/or surgical therapy for cases of chronic periodontitis and AgP).[7] This flexibility is not useful for researchers who intend to carefully study the epidemiology, aetiology or treatment for a well-defined group of periodontal infections. Prior to performing a study, investigators should adopt a classification system that can be reproducibly applied to a study population.[7]

Our proposed staging follows the natural course of AgP, justifying the need to categorise certain groups of clinical and radiological features to be placed under a particular stage. Clinical attachment loss (CAL), radiographic bone loss and smoking have been considered key factors for assigning a stage definition, based on their proven role in the risk of progression and bone loss in periodontitis.[21,22,38,39] All cases have a CAL > 5 mm, in line with previously suggested criteria for definition of severe periodontitis.[49] For a Stage I classification, no more than one site should have > 50% radiographic bone loss (below the previously-reported threshold for a 'questionable' prognosis).[21,24,38,39] Baer highlighted the presence of vertical or angular bone defects in earlier stages of juvenile periodontitis.[9] This makes recognition of this stage practically entirely dependent on periodontal probing, emphasising the importance of probing in diagnosis of periodontal diseases. Stage I cases of AgP can be distinguished from those of chronic periodontitis based on systemic health, familial aggregation of cases, and the inconsistency between the amount plaque and the extent of periodontal destruction.[6,7] As the disease progresses into Stage II, CAL increases (at least 2 sites > 6 mm) as does bone loss (50% to 70% maximum bone loss as seen in radiographs),[21,38,39] resulting in distolabial migration of maxillary and mandibular incisors. Baer reported that distolabial migration was not a feature of the early stage of the disease, instead a sign of the advanced stage.[9]

As the disease progresses to Stage III, CAL increases (at least 2 sites > 8 mm CAL) and bone loss progresses to the apical third of teeth (threshold based on previous evidence on progression in AgP),[21] leading to higher chances of nonvitality for periodontal reasons (periodontal-endodontic pathology).[40,54] Distolabial migration is more pronounced with added extrusion of teeth, especially concerning maxillary incisors.[54] Baer reported that the advanced stages of the disease (formerly known as juvenile periodontitis) had more horizontal bone loss than vertical bony defects.[9] An increase in pocket depth is also noted, which may result in the nonvitality of teeth for periodontal reasons (periodontal-endodontic pathology).[54]

Each stage (Stage I, Stage II and Stage III) can be further divided into A and B subcategories based on the presence or absence of other known risk factors (smoking, and positive family history). Each of the subcategories can be linked to either low, moderate or high risk of disease progression based on the presence or absence of risk factors.[34,41]

Smoking as an environmental factor needs to be included in AgP staging, owing to its profound effect on disease progression[2,52] and prognosis of AgP.[26–28] Patients who are current smokers (at least 10 cigarettes/day) have been grouped into subcategory B in each of the stages in our proposed staging, based on evidence that smoking in AgP leads to a less favourable response to treatment.[26,27]

Fig 1 An illustration of Stage I: orthopantomograph(OPG) of a 20-year-old female patient with bone loss in the coronal third (< 50%) around first molars and incisors. The pattern of bone destruction is prevalently angular or vertical. All the teeth can be saved by comprehensive periodontal therapy. There is no distolabial migration, instead it is spacing due to arch length-tooth size discrepancy.

Fig 2 An illustration of Stage II: deep periodontal pockets (CAL > 5mm) with pathologic tooth migration (distolabial migration) of the maxillary incisors.
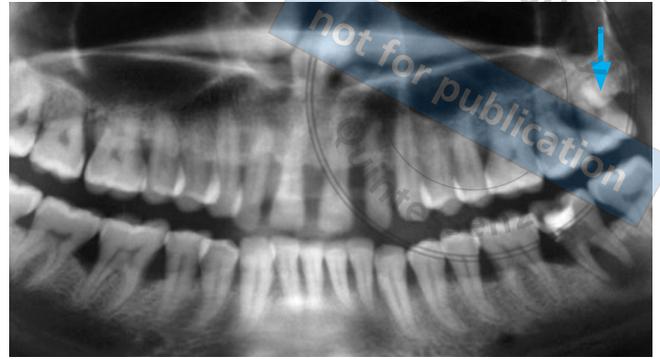
Fig 3 An illustration for stage II: OPG of the patient shown in Fig 2. Bone loss up to the mid third of the roots can be appreciated (maximum bone loss between 50% and 70%). The pattern of bone destruction is a combination of horizontal and vertical bone loss. All the teeth can be saved with an integrated treatment of periodontics and orthodontics. The left mandibular molar needs endodontic therapy due to caries reasons.
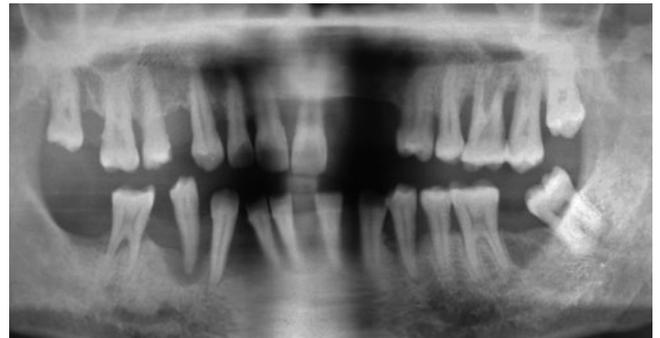
Fig 4 An illustration for stage III: OPG showing extensive bone loss up to the apical third (> 70%). Many teeth have widened periodontal ligament space with loss of lamina dura (periapical changes) suggesting non-vitality due to periodontal reasons. More than three teeth have already been lost due to periodontal reasons and many have hopeless prognosis. Pattern of bone destruction is prevalently horizontal in nature. The case has a poor prognosis and needs an integrated treatment of extraction of hopeless teeth, endodontic treatment of non-vital teeth before periodontal surgeries and prosthetic replacement of missing teeth. Radiograph by courtesy of Dr Sushmini Hegde, Reader, Department of Oral Medicine and Radiology, Oxford Dental College and Hospital, Bangalore, India.

The literature contains many references to severe generalised AgP, without explaining what exactly qualifies as severe.[13] Ababneh et al[1] evaluated the prevalence and risk indicators of gingivitis, AgP and chronic periodontitis. The authors concluded that family history was significantly associated with increased risk of periodontal disease.[1] Usually, Stage III cases need integrated treatment which involves the disciplines of periodontics, prosthodontics, endodontics and orthodontics. Clear guidelines exist on how and when to orthodontically treat a periodontally involved case.[13,23,33,35]

IL-1 composite genotype[31] positivity has been suggested to influence the pathogenesis and progression of AgP.[10] However, the present authors feel that there is little evidence to support its significance in all populations. Bäumer et al[10] studied 86 patients with AgP to evaluate the predictive value of modified periodontal risk assessment (PRA) in patients with AgP. They suggested excluding IL-1 composite genotyping in the modified PRA for accurate classification of AgP patients into high-, moderate- or low risk-profiles.[10] Exclusion of the factor IL-1 composite genotype from the modified PRA led to a model with a significant impact on tooth loss.[10] Genetic variants may in the future turn out to be associated with AgP progression. Since the evidence for specific genetic variations affecting progression of AgP across all populations is currently weak, this study excluded IL-1 genotyping and/or genetic variations from the proposed staging.

Table 2   Ten cases of AgP divided into various stages according to the proposed disease staging index for AgP, number of cases with tooth loss during active periodontal therapy, and number of cases with tooth loss noticed in the 10-year maintenance period

Stage	Number of cases	Risk of disease progression	No. of cases with tooth loss during active periodontal therapy	No. of cases with tooth loss seen in maintenance phase
Stage I A	1	Low	0	0
Stage I B	1	Moderate	1	0
Stage II A	2	Moderate	1	1
Stage II B	2	High	1	1
Stage III A	1	High	1	0
Stage III B	3	High	3	1
Total	10			

This staging should not be used to diagnose cases of AgP or to differentiate between cases of AgP and chronic periodontitis, but only after a diagnosis of AgP has been made based on the already existing criteria of Lang et al.[32] This would allow a more homogeneous definition of cases for research purposes, hence improving the possibility of associating disease progression and tooth loss with initial disease severity. Page and Eke[49] introduced a new case definition for use in population-based surveillance of periodontitis.[17,49] Surprisingly, they also suggested that for the purposes of surveillance, there seems to be no reason for separating chronic and AgP.[17] This is in strong contrast to the prevalent opinion that to study the disease, stringent disease criteria need to exist to define the disease.[30]

While discussing the differences in response to treatment in cases of AgP and chronic periodontitis, Deas and Mealey[15] suggested that future research involving modulation of host inflammatory responses may clarify the reasons for differences in clinical outcomes between patients of these two disease types. They were hopeful that with future research there could be alterations in the present classification of periodontal disease. They predicted that with the influx of knowledge, it was possible to have categories or variations in the presently accepted diagnostic groups of AgP.[15] Once periodontal health has been obtained in cases of AgP through non-surgical and/or surgical therapy, patients must be enrolled in a strict maintenance programme that is directed toward controlling risk factors for disease recurrence and tooth loss.[43,53] In patients with AgP, shorter recall intervals than for CP are advised.[14,28] In patients with low risk, moderate risk and high risk, intervals of 6 months, 3 to 6 months, and 3 months, respectively, are advised. These recall intervals suggested for different risk profiles are an oversimplification of the periodontal risk assessment. The recall intervals for individual patients of AgP should be based on periodontal risk assessment after evaluation of all the modifiable (e.g. smoking), nonmodifiable (e.g. family history) and local risk factors. The possible effect of AgP staging on disease progression, treatment response and hence recall intervals suggested by the present review needs to be investigated and confirmed by further studies with a larger study population.

ADVANTAGES AND LIMITATIONS OF STAGING

Most researchers and clinicians regard AgP as a severe disease by definition. In the proposed disease staging index, based on the limited available previous literature, AgP has been subclassified based on severity. The advantages of this index are that it follows the natural course of the disease, and the three stages can be identified by discriminant clinical and radiological features. An added advantage is that, based on the risk factors, the chances of progression of the disease can be predicted and a broad treatment plan can be formulated. Categorising a patient by severity may be beneficial in the management of the periodontal patient.[37] The disease stage can be used to establish a criterion and target for care. For example, treatment can result in nearly no lost teeth when the severity is low, and this advantage is lost when the severity is high. The disease stage provides an objective means to quickly determine severity. An increase in the disease stage provides evidence that a new treatment plan is needed. Therefore, the effect of the routine use of the disease stage could result in fewer patients with severe disease and reduce the number of teeth lost.[37]

A preliminary analysis of disease progression based on the proposed staging was presented here. Long-term analyses of AgP cases are needed to longitudinally validate this staging index and increase the extent to which the results of this study can be generalised to other situations and used by other clinicians.

REFERENCES

1. Ababneh KT, Abu Hwaij ZM, Khader YS. Prevalence and risk indicators of gingivitis and periodontitis in a multi-centre study in North Jordan: a cross sectional study. BMC Oral Health 2012;12:1.

2. Al-Habashneh R, Al-Omari MA, Taani DQ. Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic. Int J Dent Hyg 2009;7:55–61.

3. American Academy of Periodontology. Consensus report. Discussion section I. In: Nevins M, Becker W, Kornman K (eds). Proceedings of the World Workshop in Clinical Periodontics. Chicago: American Academy of Periodontology, 1989:23–32.

4. American Academy of Periodontology. Parameter on aggressive periodontitis. J Periodontol 2000(supplement); 867–869.

5. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4:1–6.

6. Armitage GC, Cullinan MP, Seymour GJ. Comparative biology of chronic and aggressive periodontitis: introduction. Periodontol 2000 2010a;53:7–11.

7. Armitage GC, Cullinan MP. Comparison of the clinical features of chronic and aggressive periodontitis. Periodontol 2000 2010b; 53:12–27.

8. Attström R, van der Velden U. Consensus report (epidemiology). In: Lang NP, Karring T (eds). Proceedings of the 1st European Workshop on Periodontics, 1993. London: Quintessence, 1994:120–126.

9. Baer PN. The case for periodontosis as a clinical entity. J Periodontol 1971;42:516–520.

10. Bäumer A, Pretzl B, Cosgarea R, Kim TS, Reitmeir P, Eickholz P, Dannewitz B. Tooth loss in aggressive periodontitis after active periodontal therapy: patient-related and tooth-related prognostic factors. J Clin Periodontol 2011;38:644–651.

11. Bial JJ, Mellonig JT. Radiographic evaluation of juvenile periodontitis (periodontosis). J Periodontol 1987;58:321–326.

12. Brown LJ, Albandar JM, Brunelle JA, Löe H. Early-onset periodontitis: progression of attachment loss during 6 years. J Periodontol 1996;67: 968–975.

13. Castellanos-Cosano L, Machuca-Portillo G, Mendoza-Mendoza A, Iglesias-Linares A, Soto-Pineda L, Solano-Reina E. Integrated periodontal, orthodontic, and prosthodontic treatment in a case of severe generalized aggressive periodontitis. Quintessence Int 2013;44:481–485.

14. Darcey J, Ashley M. See you in three months! The rationale for the three monthly periodontal recall interval: a risk based approach. Br Dent J 2011;211:379–385.

15. Deas DE, Mealey BL. Response of chronic and aggressive periodontitis to treatment. Periodontol 2000. 2010;53:154–166.

16. Demmer RT, Papapanou PN. Epidemiologic patterns of chronic and aggressive periodontitis. Periodontol 2000 2010;53:28–44.

17. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. J Periodontol 2012;83:1449–1454·

18. Gottlieb B. Tissue changes in pyorrhea. J Am Dent Assoc 1927;14: 2178–2207.

19. Gottlieb B. The formation of the pocket: diffuse atrophy of alveolar bone. J Am Dent Assoc 1928;15: 462–476.

20. Gonnella JS, Hornbrook MC, Louis DZ. Staging of disease. A case-mix measurement. JAMA 1984;251:637–644.

21. Graetz C, Dörfer CE, Kahl M, Kocher T, Fawzy El-Sayed K, Wiebe JF, Gomer K, Rühling A. Retention of questionable and hopeless teeth in compliant patients treated for aggressive periodontitis. J Clin Periodontol 2011;38:707–714.

22. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993;64:16–23.

23. Hazan-Molina H, Levin L, Einy S, Aizenbud D. Aggressive periodontitis diagnosed during or before orthodontic treatment. Acta Odontol Scand 2013;71:1023–1031.

24. Hermes CR, Baumhardt SG, Rösing CK. Occurrence of aggressive periodontitis in patients at a dental school in southern Brazil. Acta Odontol Latinoam 2013;26:84–88.

25. Highfield J. Diagnosis and classification of periodontal disease. Aust Dent J 2009;54(suppl 1):S11–26.

26. Hughes FJ, Syed M, Koshy B, Marinho V, Bostanci N, McKay IJ, Curtis MA, Croucher RE, Marcenes W. Prognostic factors in the treatment of generalized aggressive periodontitis: I. Clinical features and initial outcome. J Clin Periodontol 2006a;33:663–670.

27. Hughes FJ, Syed M, Koshy B, Bostanci N, McKay IJ, Curtis MA, Marcenes W, Croucher RE. Prognostic factors in the treatment of generalized aggressive periodontitis: II. Effects of smoking on initial outcome. J Clin Periodontol 2006b;33:671–676.

28. Kamma JJ, Baehni PC. Five-year maintenance follow-up of early-onset periodontitis patients. J Clin Periodontol 2003;30:562–572.

29. Kamma JJ, Giannopoulou C, Vasdekis VG, Mombelli A. Cytokine profile in gingival crevicular fluid of aggressive periodontitis: influence of smoking and stress. J Clin Periodontol 2004;31:894–902.

30. Kebschull M, Guarnieri P, Demmer RT, Boulesteix AL, Pavlidis P, Papapanou PN. Molecular differences between chronic and aggressive periodontitis. J Dent Res. 2013;92:1081–1088.

31. Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TG Jr, Higginbottom FL, Duff GW. The interleukin-1 genotype as a severity factor in adult periodontal disease. J Clin Periodontol 1997;24: 72–77.

32. Lang NP, Bartold M, Cullinan M. Consensus report: aggressive periodontitis. Ann Periodontol 1999;4:53.

33. Levin L, Einy S, Zigdon H, Aizenbud D, Machtei EE. Guidelines for periodontal care and follow-up during orthodontic treatment in adolescents and young adults. J Appl Oral Sci 2012;20:399–403.

34. Lü D, Meng H, Xu L, Lu R, Zhang L, Chen Z, et al. New attempts to modify periodontal risk assessment for generalized aggressive periodontitis: a retrospective study. J Periodontol 2013;84:1536–1545.

35. Maeda S, Maeda Y, Ono Y, Nakamura K, Sasaki T. Interdisciplinary treatment of a patient with severe pathologic tooth migration caused by localized aggressive periodontitis. Am J Orthod Dentofacial Orthop 2005;127: 374–384.

36. Manson JD, Lehner T. Clinical features of juvenile periodontitis (periodontosis). J Periodontol 1974;45:636–640.

37. Martin JA, Page RC, Loeb CF, Levi PA Jr. Tooth loss in 776 treated periodontal patients. J Periodontol 2010;81:244–250.

38. McGuire MK, Nunn ME. Prognosis versus actual outcome. II. The effectiveness of clinical parameters in developing an accurate prognosis. J Periodontol 1996a;67:658–665.

39. McGuire MK, Nunn ME. Prognosis versus actual outcome. III. The effectiveness of clinical parameters in accurately predicting tooth survival. J Periodontol 1996b;67:666–674.

40. Meng HX. Periodontic-endodontic lesions. Ann Periodontol 1999;4:84–90.

41. Meyer-Bäumer A, Pritsch M, Cosgarea R, El Sayed N, Kim TS, Eickholz P, Pretzl B. Prognostic value of the periodontal risk assessment in patients with aggressive periodontitis. J Clin Periodontol 2012;39:651–658.

42. 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;discussion 37–38.

43. Nanaiah KP, Nagarathna DV, Manjunath N. Prevalence of periodontitis among the adolescents aged 15–18 years in Mangalore City: an epidemiological and microbiological study. J Indian Soc Periodontol 2013;17: 784–789.

44. Nath SG, Raveendran R. „What is there in a name?“: A literature review on chronic and aggressive periodontitis. J Indian Soc Periodontol 2011;15:318–322.

45. Nibali L, Farias BC, Vajgel A, Tu YK, Donos N. Tooth loss in aggressive periodontitis: a systematic review. J Dent Res 2013a;92:868–875.

46. Nibali L. Response to letter to the editor, „Tooth loss in aggressive periodontitis: a systematic review“. J Dent Res 2014;93:213.

47. Orban B, Weinmann JP. Diffuse atrophy of the alveolar bone (periodontosis). J Periodontol 1942;13: 31–45.

48. Page RC, Schroeder HE. Periodontitis in man and other animals. A comparative review. Basel: Karger, 1982.

49. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. J Periodontol 2007;78(7 suppl):1387–1399.

50. Picolos DK, Lerche-Sehm J, Abron A, Fine JB, Papapanou PN. Infection patterns in chronic and aggressive periodontitis. J Clin Periodontol 2005;32:1055–1061.

51. Ramachandra SS. Letter to the editor, "Tooth loss in aggressive periodontitis: a systematic review". J Dent Res 2014;93:212.

52. Schenkein HA, Gunsolley JC, Koertge TE, Schenkein JG, Tew JG. Smoking and its effects on early-onset periodontitis. J Am Dent Assoc 1995;126: 1107–1113.

53. Teughels W, Dhondt R, Dekeyser C, Quirynen M. Treatment of aggressive periodontitis. Periodontol 2000 2014;65:107–133.

54. Zehnder M. Endodontic infection caused by localized aggressive periodontitis: a case report and bacteriologic evaluation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92:440–445.