Comparison of the clinical features of chronic and aggressive periodontitis

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Historical perspective

In the late 1800s, what is now known as chronic periodontitis was clinically characterized as a slowly progressive destruction of the periodontium due to the accumulation of ‘lime deposits’ on the teeth (21), or ‘calcic inflammation of the peridental membrane’ secondary to deposits of ‘salivary’ and/or ‘serosal’ calculus (13). The calcified deposits were considered to be mechanical irritants that led to gingival recession and a generalized or even pattern of bone loss (13, 21). Throughout most of the 20th century, this form of periodontitis has been considered an inflammatory disease associated with local irritants and the formation of dental plaque (biofilms) on tooth surfaces (4). This concept prevails today.

What is now known as ‘generalized aggressive periodontitis’ was not clearly described until the latter part of the 20th century. However, G.V. Black used the terms ‘phagedenic pericementitis’ and ‘chronic suppurative pericementitis’ to describe patients who suffered from a rapid destruction of alveolar bone (14). In the past three decades, authors have used a variety of terms for cases in which there is generalized severe periodontal destruction in young patients, including ‘generalized juvenile’ (16, 94), ‘rapidly progressive’ (46, 64), or simply ‘severe’ periodontitis (18). In most respects, the disease clinically resembles chronic periodontitis except the affected individuals are much younger and the rate of progression is assumed to be rapid since there is extensive periodontal damage in a young person.

Current views regarding the major characteristics of localized aggressive periodontitis have been considerably influenced by historical perspectives of the disease. In a series of papers from 1920 to 1928, Bernard Gottlieb of the University of Vienna School of Medicine described an unusual form of periodontal disease that primarily affected some or all of the permanent incisors and first molars of young individuals (29–33). Based on certain histological observations such as thin cementum on extracted teeth from affected sites, he believed that the disease was due to defective deposition of cementum or ‘cementopathia’ (32–34). Gottlieb applied the principles of classical pathology as they were practiced in the 1920s, which stated that all human non-neoplastic diseases could be classified as either inflammatory or non-inflammatory (4). Since his adolescent patients did not exhibit the intense gingival inflammation ordinarily seen in other patients (i.e. adults) with periodontitis, he believed that the disease was a non-inflammatory or degenerative condition. It was claimed that initial stages of the disease were not associated with local irritants such as dental plaque or calculus, and therefore the disease was subsequently referred to as ‘diffuse atrophy of the alveolar bone’ or ‘periodontosis’ (34, 63). According to this hypothesis, the alveolar bone degenerated and the teeth drifted apart or migrated without the formation of periodontal pockets (90). Since it was thought that continuous deposition of cementum was required in order to prevent the apical migration of epithelium along the root surface, pockets formed at sites that were supposedly afflicted with cementopathia. As a secondary phenomenon, once a pocket formed, it became susceptible to colonization by oral bacteria and was ‘...a potential trap for accumulating deposits from saliva’ (63). In the final stage of the disease, bacterial toxins and other irritants eventually caused some inflammation, which contributed to additional loss of bone and connective tissue attachment to the
tooth. Therefore, it was believed that periodontal inflammation was often observed in cases of periodontosis, but only after non-inflammatory degeneration of the alveolar bone and impairment of cementum deposition established conditions that promoted pocket formation.

Scientific proof for this hypothetical series of events could not be provided, and, at the 1966 World Workshop in Periodontics, it was concluded that there is little to no evidence for the existence of non-inflammatory degenerative periodontal disease. It was the consensus of the group of experts at the workshop ‘...that the term periodontosis is ambiguous and that the term should be eliminated from periodontal nomenclature’ (86). Nevertheless, the group also acknowledged the possible existence of a form of periodontitis in adolescents and young adults that was clinically different from the common ‘adult periodontitis’ found in older individuals (86).

In 1971, Paul Baer wrote a paper in which he suggested that the term ‘periodontosis’ be retained (10). He also supplied a definition based on some of the clinical features of the disease, which has not been significantly modified in the past four decades (10):

‘Periodontosis is a disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition. There are two basic forms in which it occurs. In one form of the disease, the only teeth affected are the first molars and incisors. In the other, more generalized form, it may affect most of the dentition. The amount of destruction manifested is not commensurate with the amount of local irritants present.’

He also indicated that the disease appeared to have its onset in the circumpubertal period (i.e. between the ages of 11 and 13 years), was more common in females than males, and had a familial background. Furthermore, he stated that, in a few patients, the disease may be self-limiting as ‘...the loss of alveolar bone may progress only to a certain point, and then may remain stationary for many years’ (10).

In the late 1970s and early 1980s, the idea that the disease may be due to degeneration of cementum, or any other components of the periodontium, was laid to rest when it was shown that the condition was an infection (49, 61, 62, 81, 94, 96) that could be effectively treated by therapy based on excellent plaque control (95). These findings were of major clinical importance since infections were considered treatable, whereas degenerative diseases were deemed beyond the scope of conventional therapeutic approaches. As a reflection of this changing opinion regarding the etiology of the disease, ‘juvenile periodontosis’ replaced ‘periodontosis’ as the preferred term for the condition (48, 49, 57, 70, 81, 94). However, all of the other characteristics of the disease listed by Baer in 1971 (10) have been retained. In the 1999 classification system, the name of the disease was changed to localized aggressive periodontitis (3, 45).

### Shared clinical features

In a general way, chronic and aggressive periodontitis share many clinical features, but the specific details of the shared features are not necessarily identical in both forms of the disease. Nevertheless, it is well established that they are both complex infections that occur in susceptible hosts and are caused by biofilms that form on tooth surfaces (37, 47, 66, 83, 96, 99). In both cases, the disease-producing biofilms comprise microorganisms that are components of the indigenous (normal) oral microbiota (7). In addition, host inflammatory/immune reactions to the presence of the biofilms are primarily responsible for the loss of periodontal attachment and alveolar bone supporting the teeth (26, 27, 78). Anti-infective treatment is usually effective in the management of both chronic and aggressive forms of periodontitis (23). The eventual outcome of untreated disease is tooth loss.

One of the shared clinical characteristics of chronic and aggressive periodontitis is that affected individuals have no known medical or general health conditions that might contribute to development of their periodontitis. According to the 1999 classification (3), if an individual has a systemic disease that can profoundly modify the initiation and clinical course of periodontal infections, the resulting periodontitis should be classified as ‘periodontitis as a manifestation of systemic disease’. Examples include severe cases of plaque-induced periodontitis associated with immunosuppression acquired via chemotherapy (41), viral infection (44, 67) or any other means, and inherited defects in neutrophil function (19, 20, 68, 87, 98), as well as other syndromic perturbations in host–parasite interactions (1, 11, 17, 22, 24, 38, 39, 71, 73, 97). Therefore, individuals with significant and powerful modifiers of the innate and adaptive immune responses should not be classified as having either chronic or aggressive periodontitis. The term ‘periodontitis as a manifestation of systemic disease’ should only be used when the systemic disease profoundly impairs the ability of the host to cope with the bacterial challenge associated with periodontitis.
Exacerbation of the periodontal infection becomes so marked that any existing periodontitis is transformed into a clinically different entity. For example, an uncomplicated case of chronic periodontitis superimposed on a patient with severe neutropenia becomes a periodontal infection with a significantly modified clinical course. Effective treatment of such a systemically modified infection requires a different strategy than would be routinely used for a medically healthy individual with chronic periodontitis.

This raises the interesting question ‘At what point does a modifier of host immune responses become so important that the periodontitis becomes a manifestation of that modifier?’ There is no simple answer to this question. In a patient with severe chronic periodontitis who also has poorly controlled type 2 diabetes mellitus, there are excellent data to show that the systemic disease (i.e. diabetes) significantly modifies the course of the periodontal infection (35, 69, 84, 89). It can be argued that the periodontal disease in such a patient could properly be categorized as any of the following: (i) diabetes-associated chronic periodontitis, (ii) periodontitis as a manifestation of systemic disease, or (iii) diabetes-associated periodontitis. In the last two categories, omission of the term ‘chronic’ implies that the periodontitis has become a different entity than a routine and relatively uncomplicated form of slowly progressing periodontal disease. In this case, for all practical purposes, it does not matter what the condition is called so long as it is recognized that medical management of the systemic disease is an important component of successful treatment of the periodontal infection. A similar situation exists in heavy cigarette smokers whose longstanding habit is clearly a powerful modifier of the severity of periodontal infections (43, 72). Should a longtime heavy smoker with severe chronic periodontitis be categorized as having: (i) chronic periodontitis in a smoker, (ii) smoking-associated chronic periodontitis, or simply (iii) smoking-associated periodontitis? Again, in clinical practice, it does not matter what the condition is called so long as it is recognized that the treatment of the periodontal infection should include recommendation or inclusion of a program of smoking cessation.

**Classification systems versus diagnoses**

Classification systems and diagnoses often serve different functions when multifactorial diseases or conditions are under consideration. Such diseases are usually associated with complex combinations of incompletely understood risk factors and etiological agents. Simple cause-and-effect dual-purpose classification and diagnostic statements (e.g. streptococcal sore throat) are not possible because of an insufficient understanding of the etiopathogenesis of the disease. Classification systems for these types of diseases are useful in studying disease patterns and types in large populations of patients. They can provide a framework for studying the epidemiology, etiology and treatment outcomes for a given group of similar diseases (6). Such systems can serve as a starting point for considering a diagnosis for a given patient, but are ill-suited for direct and rigid application to individuals. Indeed, one of the main objections to the 1999 classification has been from those who have found it difficult to apply the system to individual patients and have therefore advocated a nominalistic or descriptive approach to the classification of periodontitis (9, 59, 91, 92). Van der Velden (93) has stated that it would be worthless to group periodontal diseases on the basis of their probable causes since ‘...the causal web for periodontitis is so complex and involves so many different constellations that a classification based on etiology is effectively precluded’. Part of the problem for those who have resisted acceptance of the 1999 system is that they have attempted to use a classification scheme to generate clinical diagnoses for individual patients. As mentioned above, any existing classification system for periodontal infections cannot be simply applied to generate diagnoses for patients with these types of multifactorial diseases. Such systems are not intended to be rigidly and inflexibly used to generate a diagnosis for an individual patient. The best these systems can do is to serve as a first step in generating a clinically meaningful diagnosis for an individual.

A diagnosis is a summary statement of the clinician’s best estimate regarding the disease or condition detected in a given patient. It is derived from a thorough analysis of all information collected during a review of relevant data from medical/dental histories, the results of diagnostic tests, and findings from a careful clinical examination (5, 6). Most importantly, a diagnosis should be a short and concise statement that conveys an idea or mental picture of what disease or condition is present in a specific patient. It is extremely valuable in communication with colleagues and provides a basis for thinking about appropriate treatment approaches. For a variety of practical reasons, such as categorizing a disease for third-party payment purposes, it should be
possible to fit the diagnosis assigned to a specific patient somewhere into a currently recognized or widely used classification system. The fit of the diagnosis into the classification system does not have to be precise, somewhere in the general vicinity is usually sufficient. Clinicians should avoid rigid application of disease-category definitions of classification systems in arriving at a diagnosis. For example, arguments among clinicians about whether the patient has chronic or aggressive periodontitis are pointless, especially if the proposed treatment is going to be the same.

This flexibility is not possible for clinician scientists who intend to carefully study the epidemiology, etiology or treatment for a well-defined group of periodontal infections. Prior to performing their study, the investigators should use or adopt a classification system that can be reproducibly applied to a study population. For example, if epidemiologists intend to study the prevalence of severe generalized aggressive periodontitis in a given population, they must first agree upon an acceptable ‘case definition’ for the disease. Such a case definition must be able to clearly distinguish individuals without overlap with other disease categories. If a new anti-infective treatment for generalized severe chronic periodontitis is going to be tested, investigators must agree on the disease criteria (i.e. inclusion/exclusion criteria) for potential study volunteers prior to starting the study. Disease category definitions found in classification systems can be extremely important in planning most types of clinical studies. In these situations, arguments among scientists about whether a potential study volunteer has chronic or aggressive periodontitis are very important. Their decision will influence how the results of the investigation will be interpreted. For example, the presence of severe disease does not necessarily mean that the condition should be classified as aggressive periodontitis. The case definition of aggressive periodontitis needs to include as many of the primary features as possible to discriminate sufficiently between a severe case of chronic periodontitis and aggressive periodontitis. Unfortunately, at the current time, there is no universal agreement among researchers as to the optimal or best case definitions for studies of periodontal diseases (25, 56, 74). In addition, no existing or proposed classification system is able to provide precise guidelines for selecting an optimal case definition. This problem has been around for a long time and will not be resolved anytime soon. Fortunately, the case definition problem is not a major issue in the management of specific patients in clinical practice, as the diagnosis is tailor-made for the individual.

Significant clinical differences between chronic and aggressive periodontitis

Although similar in many general or overall respects, it has been suggested that chronic and aggressive forms of periodontitis have a number of significant clinical differences including: (i) age of onset (i.e. detection), (ii) rates of progression, (iii) patterns of destruction, (iv) clinical signs of inflammation and (v) relative abundance of plaque and calculus. Indeed, combinations of these clinical differences are the primary basis for placing affected individuals into one of the three major categories of periodontitis (i.e. chronic periodontitis, localized aggressive periodontitis and generalized aggressive periodontitis).

Localized and generalized chronic periodontitis are usually considered to be two clinical expressions of the same disease. In both cases, there are similar signs of inflammation (e.g. redness, swelling, bleeding on probing) associated with moderate to heavy deposits of plaque and calculus. They also share slow rates of progression, affect similar populations (e.g. age range, gender), and are associated with similar genetic and environmental risk factors. Except for a general tendency to exhibit bilateral symmetry (60), no consistent pattern of destruction is usually observed. In addition, there do not appear to be any noticeable differences in the subgingival microbiota (7) or histopathological features (82) between localized and generalized forms of the disease.

In contrast, localized and generalized aggressive periodontitis can be considered to be different diseases. In localized aggressive periodontitis, especially in its early stages, there are often only minimal signs of clinical inflammation associated with a thin and unimpressive biofilm on the affected tooth surfaces (10). Fig. 1 shows a 17-year-old Hispanic female with localized aggressive periodontitis on the mesial surface of a mandibular first molar. There was only mild inflammation, no detectable supragingival or subgingival dental calculus, and the lesion was associated with a relatively thin biofilm. Fig. 2 shows the appearance of a 15-year-old white male with generalized aggressive periodontitis affecting most of the permanent dentition. There were very heavy deposits of plaque and calculus and extremely intense gingival inflammation. Although there are extensive
differences between the clinical appearance of the patients shown in Figs 1 and 2, this striking contrast is not always found when comparing the two diseases. In some instances, the levels of inflammation and amounts of plaque are approximately the same in both conditions, with the only difference being the number of affected teeth or pattern of damage (i.e. localized to incisors and molars versus generalized involvement). However, the two forms of aggressive periodontitis appear to be associated with somewhat different bacterial profiles in the subgingival microbiota (7) and have separate genetic risk factors (79).

Age of onset

The age of onset, or age at the time of detection, is an important feature that has traditionally been used to help place patients in either the aggressive or chronic periodontitis category. The 1999 classification recommended deletion of age-dependent terms such as ‘adult’ and ‘juvenile’ periodontitis since age is not an appropriate descriptor for use in diagnostic categories (3). There was considerable uncertainty about setting arbitrary upper age limits for certain forms of periodontitis. For example, it was not clear what criteria should be used to distinguish between an adult and a juvenile. Should one use legal definitions for the age ranges of an adult versus juvenile, or should an attempt be made to develop a more biologically based definition? Therefore, the age-dependent categories were eliminated from the classification.

Nevertheless, age is still an important characteristic that can be useful in differentiating between chronic and aggressive forms of periodontitis. Given similar amounts of periodontal damage (i.e. probing depths, attachment loss, alveolar bone resorption), people with aggressive periodontitis are significantly younger than individuals with chronic periodontitis. The age difference is a general feature that can be useful in preliminarily deciding whether a patient has chronic or aggressive periodontitis. However, there is

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Fig. 1. (A) Clinical appearance of the mandibular right gingiva in a 17-year-old Hispanic female with localized aggressive periodontitis. The mesial surface of the first molar had a probing depth of 8 mm, with 7 mm of clinical attachment loss. There was some bleeding on probing immediately after the site was examined. Note that there are no heavy deposits of supragingival dental plaque. (B) Radiograph of the site shown in (A).

Fig. 2. (A) Clinical appearance of the gingival tissues in a 15-year-old white male with generalized aggressive periodontitis. Note the intense gingival inflammation and heavy deposits of plaque and calculus. Compare with the patient shown in Fig. 1. (B) Representative radiographs showing examples of the massive bone loss that was present on virtually all teeth.
no fixed or arbitrary upper age limit that can be used for this purpose. It is a mistake to rigidly adhere to an arbitrary age when deciding whether a patient has chronic or aggressive periodontitis. It is pointless for clinicians to argue about what is the best cut-off age to distinguish between aggressive and chronic periodontitis (e.g. should it be 30 or 35 years?). In fact, the diagnostic label is unimportant as the treatment of a 30-year-old patient with severe periodontitis will probably be the same regardless of what the disease is called. However, for research purposes and depending on the research question, it may well be reasonable to include age limits in case definitions to reduce heterogeneity within study groups and to ensure that there is no overlap in disease categories.

**Rates of progression**

The rate at which loss of supporting periodontal tissues occurs has long been considered an important characteristic by which chronic and aggressive forms of periodontitis can be clinically distinguished. Chronic periodontitis has traditionally been viewed as a slowly progressing disease, whereas aggressive forms of periodontitis progress at a rapid rate (10). Baer estimated that the loss of attachment in aggressive periodontitis patients progressed three or four times faster than in cases of chronic periodontitis (10).

Figures 3 and 4 show the progression of chronic periodontitis in posterior regions of the mandible over a 9-year period in a white male who was 51 years of age at baseline. Periodontal treatment during this observation period was provided by the general dentist, and included scaling and root planing at baseline followed by coronal polishing (‘oral prophylaxis’) once a year. Appropriate periodontal maintenance therapy was not provided. The average annual rate of progression was approximately 0.25 mm at affected sites.

Rates of progression of various forms of periodontitis are difficult to study in a formal or organized way since there are many factors that influence how rapidly periodontal tissues are destroyed. Progression of periodontal diseases is affected by the effectiveness of oral hygiene habits, access to dental care, genetically controlled susceptibility to periodontal infections, certain systemic diseases (e.g. diabetes mellitus) and other powerful host-response modifiers (e.g. smoking). Several longitudinal studies have demonstrated that there is a tendency for increased progression of periodontitis with age (2, 8, 52, 65, 77). In the few studies that have been performed on the natural history of progression of chronic periodontitis, the disease was found to progress at a full-mouth average rate of approximately 0.2 mm/year (15, 50, 52, 53, 65, 88). On a population basis, untreated chronic periodontitis progresses slowly over time, and the 0.2 mm/year rate appears to be applicable to sites without prior attachment loss as well as to those with advanced disease at the initial examination (50).

In contrast, there are forms of periodontitis with faster rates of progression. In a longitudinal study of Sri Lankan workers on a tea plantation, one group of individuals lost, on a full-mouth basis, an average...
of 0.46 mm/year (53). This group was referred to as ‘rapidly progressive’, and would probably be classified as generalized aggressive periodontitis using the current system of nomenclature. However, in a longitudinal assessment of young individuals with localized aggressive periodontitis \((n = 40)\) or generalized aggressive periodontitis \((n = 48)\), it was found that the average full-mouth rate of attachment loss was only 0.06 mm/year (36). The reasons for this difference are unclear, but many of the patients in the latter study received some form of periodontal therapy. Further, use of full-mouth averages probably had a diluting effect, especially in the case of localized aggressive periodontitis where the majority of sites are unaffected and show no attachment loss.

The most compelling argument indicating that aggressive periodontitis progresses at a rapid rate comes from case series and epidemiological reports showing extensive periodontal damage at some sites in adolescents and young adults (10, 18, 49, 57, 76). Fig. 5 shows the relatively rapid progression of untreated localized aggressive periodontitis over an 8-year period in a white female who was 12 years of age at baseline. At affected sites, the annual rate of progression was approximately 1–2 mm. The radiographs were taken in the early 1960s and were retrieved from old dental records. The patient was regularly seen by her general dentist, who assigned her a diagnosis of ‘periodontosis’. She was provided with oral prophylaxis at 6-month intervals. Her dentist believed that she had a degenerative periodontal disease that would not respond to anti-infective therapy. This is an excellent example of how a diagnostic label (i.e. periodontosis) led her dentist to believe that her disease was untreatable. Most dentists trained prior to 1960 were taught that the disease would not respond to conventional therapy.

There is a long-standing clinical impression that the rate of disease progression slows down or stops entirely in a small percentage of patients with localized aggressive periodontitis (10, 45). Baer (10) referred to this phenomenon as ‘burn out’ of the disease. A possible example of this apparent self-limiting feature of the disease is shown in Fig. 6, in which there was no radiographic evidence of progression of untreated localized aggressive periodontitis in a white female from the age of 12 to 16 years. The patient was temporarily lost to follow-up and received no periodontal therapy during the 4-year period. Fig. 7 shows a 24-year-old African-American female who had periodontal destruction localized to all first molars and two mandibular incisors in a pattern consistent with that observed in cases of localized aggressive periodontitis. She had no history of any periodontal therapy and her periodontal tissues had minimal to no signs of clinical inflammation. In these two cases, it is possible that the periodontal disease had merely gone into remission and could become active sometime in the future. There are data suggesting that the progression of periodontal diseases is an episodic phenomenon, with alternating periods of exacerbation and remission (28). In a longitudinal study of patients with either localized or generalized forms of aggressive periodontitis, Gunsolley et al. (36) noted that the localized form appeared to be a ‘stable disease in
most individuals, whereas individuals with the generalized form continued to lose periodontal attachment and teeth over time. Despite this observation, it should not be assumed that all, or even most, cases of localized aggressive periodontitis will be self-limiting. Indeed, in the study by Gunsolley et al. (36), it was found that two of the 42 patients initially diagnosed with localized aggressive periodontitis were reclassified as having the generalized form of the disease at the end of a 3-year observation period. This is consistent with the conclusions of Hørmand & Frandsen (40) and Saxén (75) who suggested, based on cross-sectional data, that localized forms of the disease become generalized with increasing age.

Fig. 8 shows radiographs of a 20-year-old white female who had localized aggressive periodontitis that was not treated and appears to have spread to the mandibular bicuspids and the cuspid on the mandibular left side. It is of interest that the infection on the mandibular right side does not appear to have involved the bicuspids and cuspid. This is the same patient who is shown in Fig. 5 whose periodontal infection was mistakenly thought to be an untreatable degenerative disease (i.e. ‘periodontosis’). (A) Age 12 years + 2 months. (B) Age 13 years + 4 months. (C) Age 14 years + 1 month. (D) Age 16 years + 1 month. (E) Age 18 years + 7 months. (F) Age 19 years + 5 months. (G) Age 20 years + 4 months.
It is possible that instances where localized aggressive periodontitis appears to spread to adjacent teeth and acquire a generalized pattern of destruction are due to the development of a new periodontal infection rather than the spread of an existing one. This possibility is supported by the observation of multiple types of periodontitis in the same family (54, 58, 85) or in a single individual (80). This issue is primarily of academic interest as it is likely that treatment would be the same for a spreading infection or superimposition of a new infection on an old one.

Patterns of destruction

In cases of chronic periodontitis, there is no consistent pattern to the number and types of teeth involved. The disease can be localized to a few teeth or can affect the entire dentition. There is a slight tendency for the destruction to exhibit bilateral symmetry (60), but there is no well-defined pattern in most cases. In cases of generalized aggressive periodontitis, most permanent teeth are usually affected. There are no evidence-based criteria to determine when a localized periodontal infection becomes generalized. The 1999 classification (45) suggested that the pattern of damage in generalized aggressive periodontitis includes situations where there is ‘...generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors’. This is similar to the criteria used by Burmeister et al. (18), who suggested that a generalized pattern of destruction is present if there is ‘...attachment loss on 8 or more teeth, at least 3 of which were not first molars or incisors’. These case definitions may be useful for the purposes of epidemiological investigations but they lose most of their clinical utility in the diagnosis and management of an individual patient. For example, if only eight teeth are affected, most clinicians would characterize the disease as a localized rather than a generalized condition.

It was the consensus of the group at the 1999 Classification Workshop that the extent of the disease be considered localized if ≤ 30% of the sites (or teeth) are affected, and generalized if > 30% of the sites (or teeth) are involved (3). This suggestion was not based on any data and was completely arbitrary. The only reason for including a description of the extent of the disease is to facilitate communication among colleagues as to the general location of the problem. For example, in written communications, most clinicians simply state the diagnosis (e.g. chronic periodontitis) followed by a list of the affected teeth. When the list of the affected teeth becomes quite long, it is often easier to merely indicate that the disease is generalized. The fallacy of rigidly using the 30% cut-off point between localized and generalized patterns of disease is nicely demonstrated in a classic case of localized aggressive periodontitis in which 12 teeth are affected (i.e. all incisors and first molars). If such a patient has only 28 teeth, then 12/28 or 42.9% teeth have the disease. Therefore, if the 30% figure is rigidly applied, some individuals with localized aggressive periodontitis paradoxically have generalized disease!
Fig. 9 shows a classic case of localized aggressive periodontitis in a 19-year-old white male in whom 12 permanent teeth were affected (i.e. all maxillary and mandibular incisors and first molars). This pattern is probably the exception rather than the rule (18). In many cases, a subset of these teeth are affected, often only including the first molars and a few incisors. In the case series reported by Liljenberg & Lindhe (49), of the eight patients with localized aggressive periodontitis, the number of affected teeth per patient ranged from 3 to 6, whereas in the seven patients with chronic periodontitis, the number ranged from 18 to 28. This is consistent with the observations of other investigators who reported similar variations in the patterns or numbers of affected teeth (12, 18, 40, 42, 76).

In some instances, the isolated periodontal damage associated with localized aggressive periodontitis can be mimicked by an infection around a retained root fragment of a primary tooth. Fig. 10A shows a radiographically visible bone loss localized to all first molars. (F) Clinical and radiographic appearance of the mandibular right first molar. Note the absence of clinically visible inflammation and supragingival plaque. There was slight bleeding on probing of the mesial and distal interproximal surfaces. Based on these findings, it is impossible to determine whether the disease is in remission or progressing.

Fig. 7. Possible example of self-limiting of untreated localized aggressive periodontitis in a 24-year-old African American female. (A) Gingival tissues show no obvious clinical signs of inflammation. (B) Healthy gingival tissues on anterior palate. (C) Slight gingival inflammation with visible supragingival calculus on the distal surface of the mandibular left central incisor. (D) Radiographs of the anterior teeth showing bone loss isolated to a few sites. (E)
radiograph from a 10-year-old female with a retained root fragment of a carious primary tooth adjacent to the mesial surface of a mandibular left permanent first molar (i.e. tooth 19). Similar cases have been reported in the literature (55). Bitewing radiographs (Fig. 10B,C) show that the bone loss was only on the left side, and that this patient had a severe problem with multiple carious teeth. The root fragment was most certainly heavily colonized by bacteria, which presumably resulted in a biofilm-induced destruction of the periodontal tissues on the permanent molar. An appropriate working diagnosis for such a situation might be: ‘severe periodontitis on the mesial of tooth 19 associated with a retained root fragment of a primary molar’. In this case, it is unnecessary to include any decision on the type of periodontitis (i.e. chronic versus aggressive). It serves no purpose in the management of the problem. Appropriate treatment included removal of the root fragment and debridement of the site.

The above case is a good example of the limitations of any classification system when a diagnostic statement for a specific patient is being generated. According to the 1999 classification system (3), the patient’s problem could be classified as a periodontal abscess (associated with a root fragment) or a gingival lesion (not otherwise specified). No clinically useful purpose is served by forcing the patient’s problem into the existing matrix of a classification system. Although not likely, the working diagnosis might be wrong, and the patient may actually have localized aggressive periodontitis isolated to the mesial surface of a single molar.

Clinical signs of inflammation

One of the features of localized aggressive periodontitis described originally was the relatively low level of gingival inflammation (e.g. redness, swelling) compared with other forms of periodontitis (10, 29–33, 63). It was this observation that was partly responsible for early authors concluding that the condition was a degenerative non-inflammatory disease. In fact, most patients with the disease often exhibit some clinical inflammation at affected sites, such as bleeding upon gentle probing along with...
slight redness and swelling of the gingival margin (Fig. 9B,C). Burmeister et al. (18) examined a population of these patients and found that the gingival index, gingival bleeding and suppuration scores at sites with attachment loss were equally high in patients with localized or generalized forms of aggressive periodontitis. Late in the disease, when there are very deep probing depths together with massive loss of periodontal support, the clinical inflammation can be quite marked (Fig. 11). In contrast, patients with generalized aggressive or chronic forms of periodontitis usually present with relatively intense gingival inflammation (Figs. 2 and 12). The reasons for these differences are not understood, but they are...
probably related to the time of initial presentation and hence amounts of microbial biomass that form on tooth surfaces over time. In localized aggressive periodontitis, the biofilms that form on tooth surfaces are often quite thin, but these deposits are usually quite thick and abundant in the other forms of periodontitis (51).

Plaque and calculus formation

In many patients with localized aggressive periodontitis, there are only thin deposits of dental plaque (i.e. biofilm), with little or no calculus (10, 49, 51). However, sites affected by the disease are not biofilm-free. Electron microscopic observations of teeth extracted because of localized aggressive periodontitis revealed that root surfaces were covered with thin deposits of gram-negative coccoid and filamentous bacteria together with other microorganisms.

The microbiota on the root surfaces was described as ‘...relatively sparse and simple’ (51). In contrast, teeth with chronic periodontitis usually have very complex and thick deposits of polymicrobial communities on affected root surfaces (51). In addition, population surveys of patients with localized aggressive periodontitis have shown that there are clinically detectable biofilms at affected sites (18).

Summary and Conclusions

Overall, while most clinicians would agree that aggressive forms of periodontitis exist as clinical entities, the clinical distinction between chronic and aggressive periodontitis (especially generalized) is not clear cut. This may not be all that significant from a treatment perspective, in so far as individualized anti-infective therapies are effective for both forms of the disease. However, from a research perspective, it is essential that these diseases be clearly distinguished in order to gain a complete understanding of their etiology and pathogenesis. The relative lack of clinical inflammation often associated with the localized molar-and-incisor form of aggressive periodontitis has been commented on for almost 100 years, and it is generally accepted that this form of the disease is associated with a thin biofilm, at least in its early stages. In contrast, the presence of clinical inflammation in generalized aggressive periodontitis appears to be similar to that observed in chronic periodontitis, and in this situation age of onset and family history are important additional criteria for either diagnosis or classification. It is also generally recognized that chronic periodontitis may subsequently be superimposed on both localized and generalized forms of aggressive periodontitis. While
this may have little bearing on the treatment of such cases, it could have an enormous impact on both the design and interpretation of research studies, whether basic science or clinical. This highlights the essential difference between a diagnosis and a classification, whereby a diagnosis is the clinician’s best guess, leading on to a treatment plan, whereas a classification does not allow such flexibility, requiring non-overlapping case definitions for research purposes if the underlying etiology of these diseases is ever to be fully elucidated.

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