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Aggressive Periodontitis – As A Clinical Entity

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Abstract
Aggressive periodontitis encompasses distinct types of periodontitis that affect people who, in most cases, otherwise appear healthy. It tends to have a familial aggregation and there is a rapid rate of disease progression. Aggressive periodontitis occurs in localized and generalized forms. Aggressive periodontitis is much less common than chronic periodontitis.

Key Words
Aggressive periodontitis, Chronic periodontitis, disease progression, clinical features.

Introduction:
Periodontitis can have many different presentations. Aggressive periodontitis comprises a group of rare, often severe, rapidly progressive form of periodontitis often characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families.

Historical Perspective:
What is now known as generalized aggressive periodontitis was not clearly described until the latter part of the 20th century. G.V. Black used the terms *phagedenic pericementitis* and *chronic suppurative pericementitis* to describe patients who suffered from a rapid destruction of alveolar bone. 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 rapidly progressive or simply severe periodontitis*[1]. In most respects, the disease clinically resembles chronic periodontitis except the affected individuals are much younger and the rate of progression is rapid. 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 from1920 to 1928, Bernard Gottlieb described an unusual form of periodontal disease that primarily affected some or all of the permanent incisors and first molars of young individuals. 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*. Since his adolescent patients did not exhibit the intense gingival inflammation ordinarily seen in adult patients 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 and therefore the disease was subsequently referred to as *diffuse atrophy of the alveolar bone or periodontosis*[2]. As a secondary phenomenon, once a pocket formed, it became susceptible to colonization by oral bacteria and periodontal inflammation.

Scientific proof for this hypothetical series of events could not be provided, and, at the 1966 World Workshop in *Periodontics*, it was concluded that the term periodontosis is ambiguous and that the term should be eliminated from periodontal nomenclature. 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.[3]

In1971, Paul Baer wrote a paper in which he suggested that the term *periodontosis* be retained. 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.[4]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 and13 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 [4]. 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[5]. As a reflection of this changing opinion regarding the etiology of the disease, *juvenile periodontitis* replaced *periodontosis* as the preferred term for the condition[5]. However, all of the other characteristics of the disease listed by Baer

in 1971 [4] have been retained. In the 1999 classification system, the name of the disease was changed to localized aggressive periodontitis.[6]

Clinical Features Of Aggressive Periodontitis

Aggressive periodontitis have a number of significant clinical features which can be studied under following headings:

(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).

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. There were very heavy deposits of plaque and calculus and extremely intense gingival inflammation. 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 and have separate genetic risk factors.[7]

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.[6] 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 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. Aggressive forms of periodontitis progress at a rapid rate where as chronic periodontitis has traditionally been viewed as a slowly progressing disease. [4].Baer estimated that the loss of attachment in aggressive periodontitis patients progressed three or four times faster than in cases of chronic periodontitis. The average annual rate of progression was approximately 0.25 mm at affected sites.[8]

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, 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 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 etal.[1] 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.[6]

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.[4] 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. Burmeister et al. [1] 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. In contrast, patients with generalized aggressive or chronic forms of periodontitis usually present with relatively intense gingival inflammation. 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. [9]

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[4] 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.[10]In contrast, teeth with chronic periodontitis usually have very complex and thick deposits of polymicrobial communities on affected root surfaces.[9]

Similar Clinical Presentations For Chronic And Aggressive Periodontitis:

Chronic and Aggressive periodontitis share many clinical features though not identical in both forms of the disease.

1. In both cases, the disease-producing biofilms comprise microorganisms that are components of the indigenous (normal) oral microbiota[11,12]
2. Both the infections occur in susceptible hosts.[13,14]
3. Loss of periodontal attachment and alveolar bone supporting the teeth.[15,16]
4. 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 [7]. According to the 1999 classification,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.
5. Anti-infective treatment is usually effective in the management of both chronic and aggressive forms of periodontitis.[17]

Summary And Conclusions

In summary, aggressive periodontitis has been historically conceptualized as encompassing

a group of different diseases. 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 with respect to 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 comprehensive family history are important additional criteria for either diagnosis or classification.

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