Periodontal Diseases of Children and Adolescents

Originating Group

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Abstract: *This paper was prepared by the Research, Science and Therapy Committee of the American Academy of Periodontology and is intended for the information of the dental profession and the public. It represents a brief summary of the current state of knowledge about periodontal diseases in children and adolescents. J Periodontol 2003;74(11):1696-1704.*

Epidemiologic studies indicate that gingivitis of varying severity is nearly universal in children and adolescents.[1-19] These studies also indicate that the prevalence of destructive forms of periodontal disease is lower in young individuals than in adults. Epidemiologic surveys in young individuals have been performed in many parts of the world and among individuals with a widely varied background. For the most part, these surveys indicated that loss of periodontal attachment and supporting bone is relatively uncommon in the young but that the incidence increases in adolescents aged 12 to 17 when compared to children aged five to 11.[15-22] In general, in the United States, epidemiologic studies indicate that the prevalence of severe attachment loss on multiple teeth among children and young adults is approximately 0.2 to 0.5 percent.[23] Despite this low prevalence, children and adolescents should receive periodic periodontal evaluation as a component of routine dental visits.

On October 30-November 2, 1999, the American Academy of Periodontology assembled an International Workshop for a Classification of Periodontal Diseases and Conditions, which resulted in a new classification.[24] Periodontal diseases discussed here will reflect the new classification system. Clinically distinct periodontal infections that can affect young individuals include: 1) dental plaque-induced gingival diseases; 2) chronic periodontitis; 3) aggressive periodontitis; 4) periodontitis as a manifestation of systemic diseases; and 5) necrotizing periodontal diseases.

Dental plaque-induced gingival diseases

Gingivitis associated with dental plaque only and gingival diseases modified by systemic factors associated with the endocrine system

Gingivitis characterized by the presence of gingival inflammation without detectable loss of bone or clinical attachment is common in children.[1-19,25] Although the microbiology of this disease has not been completely characterized, increased subgingival levels of *Actinomyces* sp., *Capnocytophaga* sp., *Leptotrichia* sp., and *Selenomonas* sp. have been found in experimental gingivitis in children when compared to gingivitis in adults. These species may therefore be important in its etiology and pathogenesis.[26,27]

Normal and abnormal fluctuation in hormone levels, including changes in gonadotrophic hormone levels during the onset of puberty, can modify the gingival inflammatory response to dental plaque.[28,29] Similarly, alterations in insulin levels in patients with diabetes can affect gingival health.[28,29] In both situations, there is an increased inflammatory response to plaque.[28,29] However, the gingival condition usually responds to thorough removal of bacterial deposits and improved daily oral hygiene.[28,29]

Periodontitis

Aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases

Children and adolescents can have any of the several forms of periodontitis as described in the proceedings of the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions (aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases). However, chronic periodontitis is more common in adults, while aggressive periodontitis may be more common in children and adolescents.[24]

The primary features of aggressive periodontitis include a history of rapid attachment and bone loss with familial aggregation. Secondary features include phagocyte abnormalities and a hyperresponsive macrophage phenotype.[24] Aggressive periodontitis can be localized or generalized. Localized aggressive periodontitis (**LAgP**) patients have interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on no more than two teeth other than first molars and incisors. Generalized aggressive periodontitis

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(**GAgP**) patients exhibit generalized interproximal attachment loss including at least three teeth that are not first molars and incisors. In young individuals, the onset of these diseases is often circumpubertal. Some investigators have found that the localized form appears to be self-limiting,[30] while others suggest that it is not.[20] Some patients initially diagnosed as having LAgP were found to have GAgP or to be periodontally healthy at a 6-year follow-up exam.[31,32]

LAgP occurs in children and adolescents without clinical evidence of systemic disease and is characterized by the severe loss of alveolar bone around permanent teeth.[31] Frequently, the disease is localized to the permanent first molars and incisors. However, some retrospective data obtained from LAgP patients suggest that bone loss around the primary teeth can be an early finding in the disease.[33] Linkage studies of the Brandywine population (a segregated group of people in Maryland that represents a relatively closed gene pool) have found a gene conferring increased risk for LAgP on chromosome 4.[34] Subsequent linkage studies of African American and Caucasian families did not confirm linkage to this locus, suggesting that there may be genetic and/or etiologic heterogeneity for aggressive periodontitis.[35-37] Reported estimates of the prevalence of LAgP in geographically diverse adolescent populations range from 0.1 to 15 percent.[23,33-35,37-42] Most reports suggest a low prevalence (0.2 percent), which is markedly greater in African American populations (2.5 percent).

Many reports suggest that patients with LAgP generally form very little supragingival dental plaque or calculus.[31,43] In contrast, other investigators find plaque and calculus at levels similar to other periodontal diseases.[44,45] Bacteria of probable etiologic importance include highly virulent strains of *Actinobacillus actinomycetemcomitans* in combination with Bacteroides-like species.[46-49] In some populations, *Eubacterium sp*. have been associated with the presence of LAgP.[50,51] To date, however, no single species is found in all cases of LAgP.[52]

A variety of functional defects have been reported in neutrophils from patients with LAgP.[53-55] These include anomalies of chemotaxis,[56-58] phagocytosis,[59,60] bactericidal activity,[61] superoxide production,[62-66] FcgIIIB (CD16) expression,[67] leukotriene B^4 generation,[68,69] and Ca2+–channel and second messenger activation.[70-75] The defect in chemotaxis is thought to be an intrinsic defect by some investigators[56-58] and an induced defect by others.[76] The influence of these functional defects on the susceptibility of individuals to LAgP is unknown, but it is possible that they play a role in the clinical course of disease in some patients. Indeed, in some cases exhibiting phagocyte abnormalities, neutrophil defects may still be present after treatment.[77] Molecular markers of LAgP can include an abnormally low number of chemoattractant receptors[78-81] and an abnormally low amount of another cell surface glycoprotein designated GP-110.[82,83] Adherence receptors on neutrophils and monocytes, such as LFA-1 and Mac-1, are normal in LAgP patients.[82,83]

GAgP, often considered to be a disease of adolescents and young adults, can begin at any age and often affects the entire dentition.[84,85] Individuals with GAgP exhibit marked periodontal inflammation and have heavy accumulations of plaque and calculus.[84] In the United States, the reported prevalence of GAgP in adolescents (14 to 17 years of age) is 0.13 percent.[23] Subgingival sites from affected teeth harbor high percentages of non-motile, facultatively anaerobic, Gram-negative rods including *Porphyromonas gingivalis.*[86,87] In one report, the levels of *P. gingivalis* and *Treponema denticola* were significantly higher in GAgP and LAgP patients compared to matched controls, with GAgP patients having the highest levels.[88] Neutrophils from patients with GAgP frequently exhibit suppressed chemotaxis as observed in LAgP[77,87] with a concomitant reduction in GP-110. This suggests a relationship between the two variants of aggressive periodontitis.[82,83]

Alterations in immunologic factors such as immunoglobulins are known to be present in aggressive periodontitis. Immunoglobulins appear to be influenced by both genetic and environmental factors and have important protective disease-limiting effects in aggressive periodontitis patients.[89-93] Human IgG antibody molecules (immunoglobulin G) are categorized into four subclasses designated as IgG1-4. Most of the antibody reactive with *A. actinomycetemcomitans* is specific for high molecular weight lipopolysaccharide and is of the IgG2 subclass. This antibody response appears to be protective, as early-onset periodontitis patients having high concentrations of antibody reactive with *A. actinomycetemcomitans* lipopolysaccharide have significantly less attachment loss (a measure of disease severity) than patients who lack this antibody.[89,90]

Overall levels of IgG2 in serum are under genetic control.[91] These levels have also been shown to be affected by periodontal diagnosis (LAgP patients have very high levels), race (African Americans have higher levels than Caucasians), and smoking (smokers have lower levels of IgG2, with notable exceptions in some patient groups).[91,92,94,95] These factors also influence specific antibody responses to *A. actinomycetemcomitans.*[91-93,95] Thus, the protective antibody response afforded by IgG2, as well as the clinical manifestations of aggressive periodontitis, is modified by patients' genetic background as well as environmental factors such as smoking and bacterial infection.[89,91-93,95,96]

Successful treatment of aggressive periodontitis depends on early diagnosis, directing therapy against the infecting microorganisms and providing an environment for healing that is free of infection.[97] While there is some disagreement among individual studies regarding treatment of LAgP, most authors recommend a combination of surgical or non-surgical root debridement in conjunction with antimicrobial (antibiotic) therapy.[47,98] These findings are supported by other work in which meticulous and repeated mechanical therapy with antibiotics proved to be sufficient to arrest most cases of LAgP.[99]

However, surgical treatment may be effective in eliminating *A. actinomycetemcomitans* without the use of antibiotics.[100] In a study of 25 deep periodontal lesions (probing depths five to 11 millimeters) in young LAgP patients, scaling and root planing alone were ineffective for the elimination of *A. actinomycetemcomitans*, while surgical therapy was effective.[100] It is not known, however, if *A. actinomycetemcomitans* is the only organism involved in disease pathogenesis.

The majority of reports suggest that the use of antibiotics is usually beneficial in the treatment of LAgP. Two reports described using antibiotics exclusively.[97,101] In both reports, LAgP patients attained significant clinical attachment gain when assessed after 12 months with tetracycline therapy alone. Most reports in the past 10 years, however, have recommended combination therapy using antibiotics and surgical or non-surgical root debridement as the optimal treatment for LAgP.[98,102-116] The most successful antibiotics reported are the tetracyclines, sometimes prescribed sequentially with metronidazole.[103,117,118] Metronidazole in combination with amoxicillin has also been utilized, especially where tetracyline-resistant *A. actinomycetemcomitans* are present.[111] A single randomized control study in which oral penicillin was used reported that therapy was successful with or without the antibiotic.[119]

While the use of antibiotics in conjunction with surgical or non-surgical root debridement appears to be quite effective for the treatment of LAgP, GAgP does not always respond well to conventional mechanical therapy or to antibiotics commonly used to treat periodontitis.[30,118,120] Alternative antibiotics may be required, based upon the character of the pathogenic flora. In GAgP patients who have failed to respond to standard periodontal therapy, laboratory tests of plaque samples may identify periodontal pathogens that are resistant to antibiotics typically used to treat periodontitis.[103] It has been suggested that follow-up tests after additional antibiotic or other therapy is provided may be helpful in confirming elimination of targeted pathogenic organisms.[103]

Chronic periodontitis is most prevalent in adults, but can occur in children and adolescents. It can be localized (less than 30 percent of the dentition affected) or generalized (greater than 30 percent of the dentition affected) and is characterized by a slow to moderate rate of progression that may include periods of rapid destruction. Furthermore, the severity of disease can be mild (one to two millimeters clinical attachment loss), moderate (three to four millimeters clinical attachment loss), or severe (≥ five millimeters clinical attachment loss). Children and young adults with this form of disease were previously studied along with patients having LAgP and GAgP. Therefore, published data are lacking for this group. In patients with one of several systemic diseases that predispose to highly destructive disease of the primary teeth, the diagnosis is periodontitis as a manifestation of systemic disease. As with adults, periodontitis associated with systemic diseases occurs in children and adolescents. Such diseases include Papillon-Lefévre syndrome,[121-125] cyclic neutropenia,[126-130] agranulocytosis,[131,132] Down syndrome,[133-135] hypophosphatasia,[136] and leukocyte adherence deficiency.[137,138] It is probable that defects in neutrophil and immune cell function associated with these diseases play an important role in increased susceptibility to periodontitis and other infections. In Down syndrome, for example, the amount of periodontal destruction has been shown to be positively correlated with the severity of the neutrophil chemotaxis defect.[135] In some cases, specific genes have been associated with these diseases. Examples include the cathepsin C gene and Papillon-Lefévre syndrome[139-141] and the tissue non-specific alkaline phosphatase gene and hypophosphatasia.[136]

The consensus report of the 1999 Workshop specifically excluded diabetes-associated periodontitis as a specific form of periodontitis associated with systemic disease. Participants concluded that diabetes is a significant modifier of all forms of periodontitis. In a survey of 263 type 1 diabetics, 11 to 18 years of age, 10 percent were found to have overt periodontitis often localized to first molars and incisors, although periodontitis was also found in a generalized pattern.[142] Affected subgingival sites harbored *A. actinomycetemcomitans* and *Capnocytophaga* sp.[143]

Periodontitis as a manifestation of systemic disease in children is a rare disease that often begins between the time of eruption of the primary teeth up to the age of four or five.[144,145] The disease occurs in localized and generalized forms. In the localized form, affected sites exhibit rapid bone loss and minimal gingival inflammation.[144] In the generalized form, there is rapid bone loss around nearly all teeth and marked gingival inflam-mation. Neutrophils from some children with a clinical diagnosis of periodontitis as a manifestation of systemic disease have abnormalities in a cell surface glycoprotein (LFA-1, leukocyte functional antigen–1, also known as CD11, and Mac-1). The neutrophils in these patients having LAD (leukocyte adhesion deficiency) are likely to have a decreased ability to move from the circulation to sites of inflammation and infection.[137] Affected sites harbor elevated percentages of putative periodontal pathogens such as *A. actinomycetemcomitans, Prevotella intermedia, Eikenella corrodens,* and *Capnocytophaga sputigena.*[146,147]

Treatment of periodontitis as a manifestation of systemic disease in children is similar to the treatment of localized and generalized aggressive periodontitis in the permanent dentition and has been reported to include surgical or non-surgical mechanical debridement and antimicrobial therapy.[124,127-130,132,134,142,144] Localized lesions have been treated successfully with this approach,[144,145] but the degree of predictable success in managing generalized periodontitis is low when systemic diseases are contributing factors.[144,145] In many cases, the affected teeth had to be extracted.[138,144,145]

Necrotizing periodontal diseases

Necrotizing periodontal diseases (**NPD**) occur with varying but low frequency (less than one percent) in North American and European children. It is seen with greater frequency (two percent to five percent) in certain populations of children and adolescents from developing areas of Africa, Asia, and South America.[148-150] The two most significant findings used in the diagnosis of NPD are the presence of interproximal necrosis and ulceration and the rapid onset of gingival pain. Patients with NPD can often be febrile. Necrotizing ulcerative gingivitis/periodontitis sites harbor high levels of spirochetes and *P. intermedia*,[151] and invasion of the tissues by spirochetes has been shown to occur.[152] Factors that predispose children to NPD include viral infections (including HIV), malnutrition, emotional stress, lack of sleep, and a variety of systemic diseases.[148-150,153] Treatment involves mechanical debridement, oral hygiene instruction, and careful follow-up.[154-156] Debridement with ultrasonics has been shown to be particularly effective and results in a rapid decrease in symptoms.[157] If the patient is febrile, antibiotics may be an important adjunct to therapy. Metronidazole and penicillin have been suggested as drugs of choice.[151,158]

Summary

Children and adolescents are subject to several periodontal diseases. Although there is a much lower prevalence of destructive periodontal diseases in children than in adults, children can develop severe forms of periodontitis.[23] In some cases, this destructive disease is a manifestation of a known underlying systemic disease. In other young patients, the underlying cause for increased susceptibility and early onset of disease is unknown. These diseases are often familial, suggesting a genetic predisposition for aggressive disease. Current modalities for managing periodontal diseases of children and adolescents may include antibiotic therapy in combination with non-surgical and/or surgical therapy. Since early diagnosis ensures the greatest chance for successful treatment,[97] it is important that children receive a periodontal examination as part of their routine dental visits.

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