

Position Paper

Periodontal Diseases of Children and Adolescents*

THIS POSITION PAPER WAS PREPARED BY THE Committee on Research, Science, and 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* 1996;67:57-62.

INTRODUCTION

Epidemiologic studies indicate that gingivitis of varying severity is nearly a universal finding in children and adolescents.¹⁻¹⁹ These studies also indicate that the prevalence of destructive forms of periodontal disease (i.e., periodontitis) is lower in young individuals than in adults. Epidemiologic surveys indicate that loss of periodontal attachment and supporting bone at one or more sites can be found in 1% to 9% of 5 to 11 year-olds and anywhere from 1% to 46% of 12 to 15 year-olds.¹⁵⁻²⁰ The relatively wide variation in these prevalence figures is probably due to differences in the populations surveyed and the criteria and the methods used for establishing the diagnosis of periodontitis. Nevertheless, available epidemiologic data clearly indicate that destructive forms of periodontal disease can occur in children and adolescents.

Clinical distinct periodontal infections that can affect young individuals include: 1) chronic gingivitis; 2) early-onset periodontitis; 3) necrotizing ulcerative gingivitis/periodontitis; and 4) periodontitis associated with systemic diseases.

CHRONIC GINGIVITIS

Chronic gingivitis is common in children¹⁹ and is characterized by the presence of gingival inflammation without detectable loss of bone or clinical attachment. Little is known about the microbiology of this disease but increased subgingival levels of *Actinomyces* sp. or black-pigmented anaerobic rods may be important.²¹ Gingivitis usually responds to a thorough removal of bacterial deposits and improved daily oral hygiene practices.

EARLY-ONSET PERIODONTITIS (LJP/GJP)

Early-onset periodontitis includes distinct types of periodontitis that affect young individuals who are otherwise healthy. Early-onset periodontitis (or juvenile periodontitis) affects teenagers and young adults. It has been divided into localized and generalized forms.²² The localized form, also called localized juvenile periodontitis (LJP), is found in teenagers and young adults. It appears to be self-limiting. The generalized form is found in older juveniles and young adults, and may involve the entire periodontium. The wide distribution and rapid rate of destruction of generalized juvenile periodontitis (GJP) is recognized by alternate terms for this condition including severe periodontitis and rapidly progressive periodontitis. Localized juvenile periodontitis, affecting mainly the first molars and incisors, and generalized juvenile periodontitis, often affecting most of the dentition, are distinguishable radiographically and clinically. Evidence is accumulating that they also differ microbiologically. For example, more *P. gingivalis* is found in GJP.²³

Localized juvenile periodontitis occurs in children and adolescents without clinical evidence of systemic disease and is characterized by the severe loss of alveolar bone around permanent teeth.²⁴ Frequently the disease is localized to the permanent first molars and incisors. However, some retrospective data obtained from LJP patients suggest that bone loss around the primary teeth can be an early finding in the disease.²⁵ DNA (gene locus) analyses indicate considerable heterogeneity among affected patients which suggests that LJP is probably not a single disease entity.^{26,27} Reported estimates of the prevalence of LJP in geographically diverse adolescent populations range from 0.1% to 15%.²⁵⁻³³ Most reports suggest a low prevalence (0.2%), which is markedly greater in AfricanAmerican populations (2.5%).

Patients with LJP generally form very little supragingival dental plaque or calculus. Bacteria of probable eti-

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ologic importance include highly virulent strains of *Actinobacillus actinomycetemcomitans* (*Aa*) or *Aa* in combination with *Bacteroides*-like species.³⁴⁻³⁶ In some populations *Eubacterium* sp. have been associated with the presence of juvenile periodontitis.^{37,38} A variety of functional defects have been reported in neutrophils from patients with LJP.³⁹ These include anomalies of chemotaxis,⁴⁰⁻⁴² phagocytosis,^{43,44} bactericidal activity,⁴⁵ superoxide production,⁴⁶⁻⁵⁰ leukotriene B₄ generation,^{51,52} and Ca²⁺-channel and second messenger activation.⁵³⁻⁵⁶ The influence of these functional defects on the susceptibility of individuals to LJP is unknown, but it is likely that they play a role in the clinical course of disease. Molecular markers of LJP can include an abnormally low number of chemoattractant receptors and an abnormally low amount of another cell surface glycoprotein designated GP-110.^{57,58} Adherence receptors are normal.^{57,58} If the disease is diagnosed early, LJP may respond well to local treatment supplemented with an appropriate systemic antibiotic such as tetracycline.⁵⁹⁻⁶¹

Generalized juvenile periodontitis, although often considered to be a disease of young adults, can begin at or around puberty.^{62,63} Unlike patients with LJP individuals with GJP exhibit marked periodontal inflammation and have heavy accumulations of plaque and calculus.⁶² In the United States the reported prevalence of GJP in adolescents (14 to 17 years of age) is 0.13%.³² Subgingival sites from affected teeth harbor high percentages of nonmotile, facultatively anaerobic, Gram-negative rods including *Porphyromonas gingivalis*.^{64,65} Neutrophils from patients with GJP frequently exhibit suppressed chemotaxis as observed in LJP,^{65,66} with a concomitant reduction in GP-110 suggesting a relationship between the two conditions.^{57,58}

Treatment

Successful treatment of early-onset periodontitis depends on early diagnosis, directing the therapeutic attack against the infecting microorganisms, and providing an environment for healing free of infection. Reports in the literature are not unanimous. While most authors consider *Aa* the etiologic agent, some recommend a combination of mechanical therapy in conjunction with antimicrobial (antibiotic) therapy for LJP. In a study of 25 deep periodontal lesions, it has been demonstrated that scaling and root planing alone was ineffective for the elimination of *Aa*.⁶⁷ However, surgical resection of periodontal tissues or systemic antibiotic therapy was effective for the long-term elimination of *Aa*. These findings are supported by other work in which meticulous, repeated mechanical therapy proved to be sufficient to arrest most cases of LJP.⁶⁸ It is not known, however, if *Aa* is the only organism involved in the pathogenesis of disease.

The vast majority of the reports suggest the use of antibiotics in the treatment of LJP. Two reports used only antibiotics in the treatment of LJP which suggests that

elimination of the *Aa* infection is the critical outcome of successful therapy.^{60,69} In both reports, LJP patients attained significant clinical attachment gain when assessed after 12 months with tetracycline therapy alone. Many reports in the past 10 years have recommended combination therapy of antibiotics and mechanical therapy (open or closed) as the optimal treatment for LJP.^{61,70-81} The most successful antibiotics reported are the tetracyclines, sometimes in combination with metronidazole.⁸² A single report using oral penicillin did not report any advantage in this antibiotic group, although therapy was successful.⁸³ In those cases that exhibit phagocyte abnormalities, neutrophil defects are reported to be still present after treatment of LJP.⁶⁶

While the use of antibiotics in conjunction with mechanical therapy appears to be quite predictable for the treatment of LJP, treatment of other forms of early-onset periodontitis is often less predictable. Generalized juvenile periodontitis does not respond well to these same therapies^{82,84} and may require other antibiotics, based upon the character of the pathogenic flora.

NECROTIZING ULCERATIVE GINGIVITIS/ PERIODONTITIS

Necrotizing ulcerative gingivitis/periodontitis (NUG/P) occurs with varying but low frequency in North American and European children; however, it is seen with greater frequency in certain populations of children and adolescents from underdeveloped areas of Africa, Asia, and South America.⁸⁵⁻⁸⁷ The two most significant findings used in the diagnosis of NUG/P are the presence of interproximal necrosis and ulceration and the rapid onset of gingival pain. An elevated systemic temperature can often be detected. Affected sites harbor high levels of spirochetes and *Prevotella intermedia*⁸⁸ and invasion of the tissues by spirochetes has been shown to occur.⁸⁹ Factors that predispose children to NUG/P include viral infections, malnutrition, emotional stress, lack of sleep, and a variety of systemic illnesses. Treatment involves local therapy and careful follow-up.⁸⁵⁻⁸⁷

PERIODONTITIS ASSOCIATED WITH SYSTEMIC DISEASE

As with adults, periodontitis associated with systemic disease occurs in children and adolescents. Such diseases include Papillon-Lefevre syndrome,⁹⁰⁻⁹³ cyclic neutropenia,⁹⁴⁻⁹⁸ agranulocytosis,^{99,100} Down syndrome,¹⁰¹⁻¹⁰³ Type I diabetes mellitus,¹⁰⁴ and leukocyte adherence deficiency.^{105,106} 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.

It has been reported that diabetic children have more gingival inflammation than children without diabetes, despite similar plaque scores.¹⁰⁷ In a survey of 263 Type I

diabetics, 11 to 18 years of age, 10% were found to have overt periodontitis often localized to first molars and incisors, although it was also found in a generalized pattern.¹⁰⁴ Affected subgingival sites harbor *Aa*, *Capnocytophaga*, and anaerobic vibrios¹⁰⁸ suggesting that local debridement and systemic antimicrobial therapy may be useful in management of periodontitis in diabetics.

Periodontitis associated with leukocyte adherence deficiency (LAD) is a rare disease that begins between the time of eruption of the primary teeth up to the age of 4 or 5, and has been termed "prepubertal periodontitis."^{109,110} The disease occurs in localized and generalized forms. In the localized form, affected sites exhibit rapid bone loss and minimal gingival inflammation.¹⁰⁹ In the generalized form, there is rapid bone loss around nearly all teeth and marked gingival inflammation. Neutrophils from some patients with a clinical diagnosis of prepubertal periodontitis have abnormalities in a cell surface glycoprotein that mediates adherence to surfaces.¹⁰⁶ It has been suggested that the diagnosis of prepubertal periodontitis should not be made solely on the basis of the patients' dental histories or clinical appearances since some of these individuals have underlying systemic diseases such as hypophosphatasia or agranulocytosis with normal neutrophil adhesions and chemotaxis.¹¹⁰ It is perhaps useful to term these forms of periodontal disease periodontitis associated with LAD or hypophosphatasia. Affected sites harbor elevated percentages of putative periodontal pathogens such as *Actinobacillus actinomyces*, *Prevotella intermedia*, *Eikenella corrodens*, and *Capnocytophaga sputigena*.^{111,112} It should be noted that some prepubertal children may have severe periodontitis, although the presence of an associated systemic disease cannot be confirmed.

SUMMARY

Children and adolescents are subject to a wide variety of periodontal infections. Although there is a much lower prevalence of destructive periodontal diseases in children than in adults, children can develop severe forms of periodontitis. Often the diagnosis is more difficult due to the uncommon occurrence of periodontitis in children. Since early diagnosis is important for successful treatment, it is imperative that children receive a periodontal examination as part of their routine dental visits. Furthermore, destructive periodontal disease often occurs in adolescents with certain systemic diseases. Indeed, the presence of severe periodontitis in children and adolescents may be an early sign of systemic disease. A general medical evaluation to determine if systemic diseases are present should be considered in children who exhibit severe periodontitis, especially if the disease appears resistant to therapy.

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