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Periodontal diseases in the child and adolescent

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Abstract
Background: Periodontal diseases are among the most frequent diseases affecting children and adolescents. These include gingivitis, localized or generalized aggressive periodontitis (a.k.a., early onset periodontitis which includes generalized or localized prepubertal periodontitis and juvenile periodontitis) and periodontal diseases associated with systemic disorders. The best approach to managing periodontal diseases is prevention, followed by early detection and treatment.
Methods: This paper reviews the current literature concerning the most common periodontal diseases affecting children: chronic gingivitis (or dental plaque-induced gingival diseases) and early onset periodontitis (or aggressive periodontitis), including prepubertal and juvenile periodontitis. In addition, systemic diseases that affect the periodontium and oral lesions commonly found in young children are addressed. The prevalence, diagnostic characteristics, microbiology, host-related factors, and therapeutic management of each of these disease entities are thoroughly discussed.

Key words: periodontal disease; children; pedodontics; early onset periodontitis; aggressive periodontitis; localized juvenile periodontitis

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Dental clinicians must be adept at diagnosis and management of periodontal disease in children and adolescents. Periodontal diseases are not limited to adults. On the contrary, periodontal diseases are prevalent among children and adolescents. For example, gingivitis affects more than 70% of children older than seven years of age (Page & Schroeder 1982, Stamm 1986). Bimstein (1991) stressed the importance of prevention, early diagnosis and early treatment of periodontal diseases in children and adolescents because (1) the prevalence of and severity of periodontal diseases are high; (2) incipient periodontal diseases in children may develop into advanced periodontal diseases in adults; (3) there is association between periodontal and systemic diseases; (4) patients, families, or populations at risk may be identified and included in special prevention or treatment programs; and (5) prevention and treatment of most periodontal diseases are relatively simple and very effective, providing lifetime benefits.

The nomenclature and classification systems we use to describe periodontal disease have changed periodically over the past few decades. This leads to some confusion when one reviews past literature about disease prevalence, treatment, etc. The *Consensus Report* of the *1989 World Workshop in Clinical Periodontics* used the following criteria to distinguish various forms of periodontitis: (1) age of onset; (2) rate of disease progression; (3) distribution of affected sites; (4) presence or absence of systemic medical conditions; (5) presence or absence of specific microbial or host factors; and (6) response of the disease to therapy (American Academy of Periodontology: AAP 1989). The classification (AAP 1989) was simplified by the Consensus Report of the First European Workshop on Periodontology (Attström & van der Velden 1994), and more recently, a new classification of periodontal diseases and conditions was presented at the 1999 International Workshop on Periodontics (Armitage 1999). The 1999 International Workshop classification system includes a greater variety of disease categories that base the diagnosis on clinical history and findings, relying less on age of onset as a major criterion (Table 1).

This paper primarily follows the classification of periodontal disease from the 1989 World Workshop in Clinical Periodontics (AAP 1989) when describing conditions because most clinicians are familiar with it. For each condition, we also include the International Workshop in Periodontics classification (Armitage 1999) parenthetically to introduce readers to the new system and to highlight the changes between the two.

The aim of this paper is to review the current literature concerning the most common periodontal diseases affecting children: chronic gingivitis and early onset periodontitis, including prepubertal and juvenile periodontitis. In addition, systemic diseases that affect the periodontium and oral lesions commonly found in young children will be addressed.

Table 1. Classifications of periodontitis

The 1989 World Workshop in Clinical Periodontics	
I.	Adult periodontitis
II.	Early-onset periodontitis
A.	Prepubertal periodontitis (generalized, localized)
B.	Juvenile periodontitis (generalized, localized)
C.	Rapidly progressive periodontitis
III.	Periodontitis associated with systemic disease
IV.	Necrotizing ulcerative periodontitis
V.	Refractory periodontitis
The 1994 First European Workshop on Periodontology	
I.	Early onset periodontitis
II.	Adult periodontitis
III.	Necrotizing periodontitis
The 1999 International Workshop on Periodontology	
I.	Gingival diseases
II.	Chronic periodontitis
III.	Aggressive periodontitis
IV.	Periodontitis as a manifestation of systemic diseases
V.	Necrotizing periodontal diseases
VI.	Abscesses of the periodontium
VII.	Periodontitis associated with endodontic lesions
VIII.	Developmental or acquired deformities and conditions

(I). Chronic Gingivitis (Dental Plaque-induced Gingival Diseases)

Chronic gingivitis is the most common periodontal infection among children, and adolescents. These may include plaque-induced chronic gingivitis (the most prevalent form), steroid hormone related gingivitis, drug-influenced gingival overgrowth, and others. It is characterized by inflammation of the marginal gingiva without detectable loss of bone or connective tissue attachment. The initial clinical findings in gingivitis include redness and swelling of marginal gingiva, and bleeding upon probing. As the condition persists, tissues that were initially edematous may become more fibrotic. Gingival margins, normally knife-edged in contour, may become rolled, and interdental papillae may become bulbous and enlarged. Probing depths may increase if significant hypertrophy or hyperplasia of the gingiva occurs.

Histologically, ulceration of the sulcular epithelium and inflammatory cell infiltration of the underlying connective tissue characterize gingivitis. At the microscopic level, T-lymphocytes predominate in children (Seymour et al. 1981) whereas B-cells predominate in adults (Page & Schroeder 1976).

Matsson & Goldberg (1985) suggested that there is no clear-cut age at which the gingival reaction to bacterial insult in children becomes similar to that in adults. However, there is gradual increase in gingival activity from early childhood to adult age. Previously, Matsson (1978) performed a 21-day experimental gingivitis study comparing 6 children, aged 4 to 5 years, with 6 dental students, aged 23 to 29 years. They found that the children developed gingivitis less readily than the adults. After 21 days without plaque removal, the children had less gingival exudate and a lower percentage of bleeding sites than the adults. Also, the children had a smaller increase in gingival crevicular leukocytes than the adults in response to plaque accumulation. The study hypothesized that children may have a different vascular response than adults.

Gingivitis does not always progress to periodontitis, but periodontitis is preceded by gingivitis. According to Marshall-Day et al. (1955), gingivitis without evidence of bone loss was confined to a younger age group, and bone loss without gingival alterations was rarely found.

Plaque-induced chronic gingivitis is commonly found in young children and can be managed by mechanical removal of plaque and high levels of oral hygiene. The etiologic nature of plaque in gingivitis was demonstrated by experimental gingivitis studies in humans (Löe et al. 1965, Theilade et al. 1986). Therefore, dentists and dental hygienists must educate parents and their children about the importance of oral hygiene in the prevention of caries and periodontal diseases. Electric toothbrushes and antibacterial mouthrinses may be useful adjuncts to standard toothbrushing and flossing for children who are physically challenged, such as children with Downs syndrome, and for children undergoing orthodontic care (Trombelli et al. 1995, Grossman & Proskin 1997, Boyd 1989).

Steroid hormone-related gingivitis is associated with elevated sex hormone levels that amplify clinical inflammatory changes of gingivitis (Suzuki 1988). Increased levels of estrogen and progesterone during pregnancy (Löe 1965), during puberty, or in patients medicated with oral contraceptives (Kalkwarf 1978) have been reported to result in increased gingival vascularity and inflammation. Removal of local factors is the key to the management of steroid hormone-related gingivitis; however, it is often necessary to surgically excise unresolved gingival overgrowth.

Drug-influenced gingival enlargement has been observed in patients taking cyclosporin, phenytoin and calcium channel blockers, with higher prevalence in children (Mariotti 1999, Seymour et al. 2000). Gingival enlargement was reported in 30% of patients taking cyclosporin (Seymour & Heasman 1992), 50% of patients taking phenytoin (Hassell 1981), and 15% of patients taking calcium channel blockers such as nifedipine (Lederman et al. 1984), verapamil (Pernu et al. 1989), and amlodipine (Seymour et al. 1994). Gingival enlargement starts in the interdental papilla region and spreads to involve the marginal gingiva. In severe cases the enlarged gingiva may cover the incisal and occlusal surfaces of the teeth (Chapple 1996). Histologically, the overgrowth is fibroepithelial in nature, with epithelial acanthosis, increased numbers of fibroblasts and increased collagen production. The severity of gingival enlargement is closely related to the level of plaque control, and therefore reductions in dental plaque can limit the severity of the lesion (Mariotti 1999).

Treatment of drug-influenced gingival enlargement should start with oral hygiene instructions, and scaling and polishing of the teeth to remove bacterial deposits. This may require several visits. 2× daily chlorhexidine rinses (Lang et al. 1982) should be prescribed when normal brushing and flossing do not achieve the desired level of plaque control. Gingivectomy or gingivoplasty may be needed to correct gingival con-

tours for hygienic and esthetic reasons. The patient and parent should be told that gingival enlargement may recur after surgery, and that immaculate home plaque control and regular periodontal maintenance are the key to decreasing the likelihood or severity of recurrence. In addition, the patient's physician should be consulted to determine if the patient could be switched to a medication that does not cause gingival enlargement. The dentist should never recommend that a patient discontinue or change medications without medical consultation.

(II). Early Onset Periodontitis (New Classification: Aggressive Periodontitis)

Characteristics of early onset periodontitis include onset before age 35 years, rapid disease progression, somewhat different distribution of lesions than in adult periodontitis, and distinct etiologic characteristics.

1. Prepubertal periodontitis (PP) (new classification: aggressive periodontitis)

The prevalence of PP ranges from 0.84% to 26.9%, based on a limited number of case reports (Jamison 1963, Sweeney et al. 1987, Bimstein et al. 1988). Variation in the reported prevalences of PP may depend on genetic factors, methodological factors, and the selection of non-random sample populations (Watanabe 1990). Prepubertal periodontitis may be generalized or localized and can affect both primary and mixed dentition. Onset occurs between the eruption of the primary dentition and puberty. PP is characterized by severe gingival inflammation, rapid bone loss, tooth mobility, and tooth loss. Suzuki (1988) reported that PP patients are usually between ages 5 and 8, they have low caries rates, and there is no sex predilection. Page et al. (1983) first described "prepubertal periodontitis" as a disease entity and further subdivided it into a localized form of PP (LPP) and a generalized form of PP (GPP). This was based on the clinical description of only three and two cases, respectively. Diagnostic characteristics for prepubertal periodontitis are listed in Table 2.

Pathogenic bacteria that have been associated with PP include *Actinobacillus actinomycetemcomitans* (*A.a.*), *Prevotella intermedia (P.i.)*, *Capnocytophaga* species (Delaney & Kornman 1987, Sweeney et al. 1987), *Porphyromonas gingivalis (P.g.)* (Mishkin et al. 1986), and *Eikenella corrodens (E. corrodens)* (Delaney & Kornman 1987). Delaney & Kornman (1987) found an increased proportion of black-pigmented anaerobic rods, *E. corrodens*, *Capnocytophaga* species, *A.a.*, and *Fusobacterium nucleatum (F. nucleatum)* in the cultivable flora in LPP patients compared to controls. Studies have tried to determine whether PP ultimately progresses to juvenile periodontitis (JP). According to Watanabe (1990), if pathogenic bacteria around the deciduous dentition in PP patients remain to infect the area or neighboring area during eruption of the permanent teeth, subsequent infection of newly erupted permanent teeth may occur. The coexistence of *A.a.* in lesions of both PP and localized juvenile periodontitis (LJP) (Zambon et al. 1983) may explain the possibility. However, the possible mechanism of disease progression needs to be re-evaluated since not all PP develops into JP.

Although the primary etiology remains bacterial plaque, other factors may predispose otherwise healthy patients to PP. These may include cementum defects (Page & Baab 1985), functional defects in leukocyte chemotaxis (Page et al. 1983), and/or presence of bacteriophage (Watanabe 1990). It has been generally recognized that GPP and leukocyte adhesion deficiency (LAD) are related. Waldrop et al. (1987) demonstrated that the molecular basis of GPP was leukocyte adhesion deficiency by using monoclonal antibodies against Mac-1, LFA-1, and p150,95 surface adhesion molecules. On the other hand, immunological data about LPP have shown that a neutrophil or monocyte chemotaxis defect, but not both, exists in LPP patients.

Systemic diseases associated with prepubertal periodontitis (new classification: periodontitis as a manifestation of systemic diseases) may include insulin dependent diabetes mellitus (IDDM), Papillon-Lefèvre syndrome, hypophosphatasia, neutropenia, Chediak-Higashi syndrome, leukemias, histiocytosis X, acrodynia, acquired immunodeficiency syndrome (AIDS), Down syndrome, and leukocyte adhesion deficiency.

Insulin dependent diabetes mellitus (IDDM, Juvenile DM) is a relative or absolute decrease in insulin secretion or availability, caused by a genetic defect in pancreatic beta cell productivity, defective insulin release mechanisms (Johnes & Mason 1980) or by destruction of beta cells (Jackson & Guthrie 1986). Bernick et al. (1975), Gislen et al. (1980), and Cianciola et al. (1982) studied the association between juvenile diabetes and periodontal diseases in young children. Bernick et al. (1975) examined 50 diabetic children and 36 healthy control children who were matched for age and oral hygiene status. They found that gingival inflammation was more prevalent in the diabetic group. Furthermore, Gislen et al. (1980) showed that diabetic children

Table 2. Diagnostic characteristics of prepubertal periodontitis

Localized prepubertal periodontitis (LPP)
- Onset: around 4 years of age or older
- Affected teeth: either few or many teeth
- Gingival manifestation: minor inflammation, if any
- Microbial plaque: minimal
- Alveolar bone destruction: faster than adult periodontitis but much slower than generalized form of PP
- Functional defects: either neutrophils or monocytes but not both
- Otitis media and upper respiratory infections in some cases
- Amenable to mechanical and antibiotic therapy

Generalized prepubertal periodontitis (GPP)
- Onset: at the time of tooth eruption
- Affected teeth: all primary teeth; the permanent dentition may or may not be affected
- Gingival manifestation: fiery red gingiva with acute inflammation around all teeth; gingival proliferation, cleft formation, and recession
- Alveolar bone destruction: rapid
- Functional defects: both neutrophils and monocytes
- Absence of PMNs in the gingival tissue and marked increase in peripheral blood white cell count
- Otitis media and upper respiratory infections in most cases
- Refractory to mechanical and antibiotic therapy

with poor metabolic control appeared to have greater gingival index scores than children without diabetes. Cianciola et al. (1982) investigated prevalence of periodontal disease in IDDM in 263 IDDM patients and 208 control subjects. For children between 11 and 18 years old, 9.8 % of the IDDM patients showed signs of periodontitis versus only 1.7% of the controls. They found no periodontitis in children 10 years old or younger in either group. The article argues that probing alone is not sufficient to assess periodontitis, particularly when applied to the mixed dentition. Adjunctive use of radiographs was recommended for the assessment of periodontitis in children. IDDM patients have also been shown to have a reduced PMN response to chemotactic stimuli (Miller 1972).

Papillon-Lefèvre syndrome (PLS) is a genetic condition inherited as an autosomal recessive trait, characterized by hyperkeratosis of the palms of the hands and soles of the feet and early onset periodontitis (Johnes & Mason 1980). Severe periodontitis can affect both the primary and permanent dentitions (Smith & Rosenzweig 1967, Gorlin et al. 1964). The prevalence of PMN chemotactic defects in the PLS patients remains controversial.

Hypophosphatasia is a genetic disorder. Diagnostic findings include low levels of serum alkaline phosphatase and excretion of phosphoethanolamine, the substrate for alkaline phosphatase, in the urine. The disorder can be classified into 3 types: (1) infantile, an autosomal recessive condition occurring before 6 months of age (the most severe form); (2) childhood, an autosomal recessive or autosomal dominant condition occurring after 6 months of age; and (3) adult hypophosphatasia, an autosomal dominant condition that is the least severe form (Watanabe 1990). The most consistent clinical sign is premature loss of deciduous teeth (Fallon et al. 1984). The teeth may have large pulp chambers, and skeletal deformities may occur. Baer & Benjamin (1974) claimed that only deciduous teeth are generally affected. Baab et al. (1986) reported normal polymorphonuclear leukocyte (PMN) chemotaxis and abnormal monocyte chemotaxis in 3 children with hypophosphatasia; however, age range was not designated in the study.

Neutropenia is a disorder in which the number of PMNs in the peripheral blood is below 1000/mm^3 in infants, below 1000/mm^3 or 1500/mm^3 in children, and below 1800/mm^3 in adults. Neutropenias associated with oral manifestations in children include agranulocytosis (malignant neutropenia, rare in children), cyclic neutropenia, chronic benign neutropenia of childhood, chronic idiopathic neutropenia, and familial benign neutropenia. Prichard et al. (1984) and Cohen & Morris (1961) reported that cyclic neutropenia was associated with severe gingivitis with ulceration and frequent history of recurrent infections such as otitis media and upper respiratory infection.

Chediak-Higashi syndrome is a rare autosomal recessive disease associated with impaired function of cytoplasmic microtubules or microtubule assembly in PMNs. Diagnostic features include characteristic, abnormally large granules in granulocytes. According to Clawson et al. (1978), PMN chemotactic defects in Chediak-Higashi syndrome may be due to mechanical interference in deformability by the giant cytoplasmic granules. Moreover, impaired microtubule assembling function (Oliver 1976) and hyperadhesiveness (Keller et al. 1984) were suggested as the mechanism for PMN chemotactic defects.

Leukemias are characterized by progressive uncontrolled proliferation of white blood cells and classified into acute and chronic lymphocytic leukemia, acute and chronic granulocytic leukemia, acute and chronic monocytic leukemia, and other rare forms such as plasma cell leukemia. Oral manifestations of leukemias in children are gingival hemorrhage, petechiae, lymphadenopathy, gingival hyperplasia or hypertrophy, and gingival pallor. Bender (1944) and Deasy et al. (1976) reported association between periodontal disease and leukemia in children.

Histiocytosis X represents a disturbance of the reticulo-endothelial system. It results from the proliferation and dissemination of pathological Langerhans cells and includes three disorders: Letterer-Siwe disease (the most severe form and usually occurs in infants or before age 3); Hand-Schuller-Christian disease (usually occurs before age 5, but can occur in adolescents and even in young adults); and eosinophilic granuloma (occurs primarily in older children and young adults) (Hartman & Colonel 1980). Increased susceptibility to bacterial infections in histiocytosis X patients may result from PMN defects (Tomooka et al. 1986) or decreased monocyte function (Kragballe et al. 1981).

Acrodynia is a rare disease characterized by a wide variety of clinical signs including gingival and mucosal hyperplasia, alveolar bone loss with early loss of primary teeth, loss of hair, abnormal cramps, profuse sweating and salivation, and gastrointestinal upsets. Etiology of acrodynia is known to be a mercurial toxicity reaction, either actual mercury poisoning or, more likely, an idiosyncracy to mercury (Warkany & Hubbard 1948).

Acquired immunodeficiency syndrome (AIDS) develops as a result of infection with the human immunodeficiency virus (HIV). Children with AIDS may develop an atypical form of acute necrotizing ulcerative gingivitis (ANUG) (Leggott 1987); however, no reports of prepubertal pediatric AIDS patients presenting with alveolar bone loss exist.

Down's syndrome (Trisomy 21 syndrome; Mongolism) is a common and easily recognizable chromosomal aberration. The dentition exhibits a number of characteristic anomalies including malformation of the teeth, enamel hypoplasia, and microdontia. Other common oral manifestations include macroglossia, fissured or pebbly tongue, and a high arched palate. Due to a defective immune system, rampant and precocious periodontal disease is prevalent. Barr-Agholme et al. (1998) compared 20 Down's syndrome patients, aged 9 to 21 years, with 19 healthy controls and reported that gingival inflammation, probing depth, calculus, and marginal bone loss were significantly greater in the Down's syndrome group. No significant differences were found between the two groups, regarding levels of serum IgA, IgG_2, IgG_3, IgG_4, IgM, or albumin. However, the proportion of IgG_1 in total IgG was significantly higher in the Down's syndrome group compared to the control group.

Leukocyte adhesion deficiency (LAD) is a genetic disease inherited as an autosomal recessive condition in which the expression of Mac-1, LFA-1, and p150,95 glycoprotein adhesion molecules on leukocytes is severely reduced. Springer et al. (1984) suggested that LAD occurs as a result of a defect in the gene that codes for the common beta subunit of cell surface integrins which are required for leukocyte diap-

Table 3. Diagnostic characteristics of juvenile periodontitis

Localized juvenile periodontitis (LJP)
• Onset: around puberty
• Affected teeth: permanent incisors and/or first molars
• Associated microorganism: *Actinobacillus actinomycetemcomitans*
• Greater prevalence in blacks
• Neutrophil dysfunction and high IgG2 response
• Familial distribution
Generalized juvenile periodontitis (GJP)
• Onset: during late teen years
• Affected teeth: generalized involvement of permanent teeth
• Associated microorganism: not clear (*P. gingivalis* may be involved)
• Greater prevalence in blacks
• Neutrophil dysfunction
• Familial distribution

edesis. Because emigration of PMNs from blood vessels is dependent upon adhesion of PMNs to endothelial cell surfaces, patients with LAD have a poor response to bacterial infections. Therefore, these children are plagued with recurrent bacterial infections and impaired wound healing. Patients with this rare disease present with extremely acute inflammation, proliferation of the gingival tissues, and rapid bone loss. An association between generalized prepubertal periodontitis and LAD was demonstrated by Waldrop et al. (1987) and Page et al. (1987).

Treatment for PP has ranged from scaling and root planing or curettage, with or without systemic antibiotics, to the extraction of involved teeth (Watanabe 1990). The generalized form, unfortunately, does not respond as favorably to treatment as the localized form. The majority of cases have been treated by extraction of involved deciduous teeth and permanent teeth. Since these patients may have associated LAD and may be undergoing antibiotic therapy for recurrent serious infections, it is necessary to have a consultation with the treating physician.

2. Juvenile periodontitis (JP) (new classification: aggressive periodontitis)

Juvenile periodontitis, formerly called periodontosis, occurs in children and teenagers who are otherwise healthy and is characterized by a rapid alveolar bone loss in one or more teeth of the permanent dentition (Baer 1971). Terminology for this disease entity has repeatedly changed. Gottlieb et al. (1923) first described this condition ''diffuse atrophy of the alveolar bone.'' His findings in this case included widened periodontal ligament (PDL), cementopathia and alveolar bone resorption, but no pathology in gingival tissues. Later, the degenerative process was termed periodontosis (Orban & Weinmann 1942), who described three stages of the disease: degeneration of PDL fibers; proliferation of the junctional epithelium; and the development of deep infrabony pockets. The term ''periodontosis'' was favored by Baer (1971). In his article, the disease entity was divided into two categories, a localized form affecting the first molars and incisors, and more generalized form affecting most of dentition. The term ''juvenile periodontitis'', introduced by Butler (1969), accurately describes the disease as an inflammatory process. Waerhaug (1977) described that the primary etiology of the disease is subgingival bacterial plaque. The plaque front is consistently located within 0.2 mm to 1.5 mm from the border of loss of attachment. The plaque grows with a faster speed of 3 to 5 microns a day in this disease compared to 0.2 to 1.0 μm a day in ordinary cases. Diagnostic characteristics of juvenile periodontitis are illustrated in Table 3.

Estimates of the prevalence of JP vary from 0.1% to 15% (Sjödin et al. 1993, Hart et al. 1993, Saxén 1980, Löe & Brown 1991, Neely 1992), with differences attributed to varying criteria, geographics, and a diverse data base. Reports show greater prevalence in African-Americans than in Caucasians (Burmeister et al. 1984, Löe & Brown 1991, Melvin et al. 1991). Albandar et al. (1997a) estimated prevalence of EOP (JP) in a group of schoolchildren, aged 13 to 17 years, based on the data of a national survey conducted during the 1986–1987 school year. They showed that approximately 10.0% of African-American, 5.5% of Hispanic, and 1.3% of white adolescents had EOP. Hørmand & Frandsen (1979) explained that the higher incidence of LJP in females could result from an earlier occurrence of the disease in females. Stabholz et al. (1998) reported a high prevalence of LJP in an Israel population consisting of 86 individuals from 30 families, aged 12 to 20 years. Out of 86 subjects, 33 individuals from 15 families showed LJP (38.4%), but none of the subjects showed signs of generalized juvenile periodontitis. Except for two pairs of families with genetic ties, no familial connections could be traced between the different nuclear families affected by LJP, and there were no differences in oral hygiene status between LJP and non-LJP groups. The study suggests that environmental factors may influence the occurrence of LJP.

The microflora associated with JP is mainly composed of Gram-negative, capnophilic, and anaerobic rods (Slots 1979, Newman & Socransky 1977). *Actinobacillus actinomycetemcomitans* (*A.a.*), a Gram-negative, non-motile, coccobacillus has been well documented in relation to LJP (Newman & Socransky 1977, Slots et al. 1980, 1982, Kornman & Robertson 1985). Asikainen et al. (1991) examined *A.a.* isolates from subgingival sites in 136 subjects in Finland and found that among five serotypes of *A.a.*, serotype b was predominant in LJP and adult periodontitis, whereas serotype c predominated in healthy individuals. Wilson & Hamilton (1992) also demonstrated that black subjects with LJP responded to *A.a.* serotype b serospecific antigens by the production of IgG antibodies of the IgG2 subclass. Lu et al. (1993, 1994) confirmed this by demonstrating that IgG2 was markedly elevated over all other Ig classes reactive with *A.a.* serotype b. Albandar et al. (1997b) found that EOP patients had higher prevalence and levels of *P.g.*, *P.i.*, *F. nucleatum.*, *C. rectus*, and *T. denticola* compared to healthy controls. *P.g.*, *T. denticola*, and *P.i.*, in descending order of importance, were significantly associated with the generalized and/or rapidly progressing disease.

Reports indicate that JP patients have defects in neutrophil chemotaxis or phagocytosis (Cianciola et al. 1977, Clark et al. 1977, Van Dyke et al. 1987, Suzuki et al. 1984, Kimura et al.1992, etc.). Neutrophils in LJP patients have fewer receptors on their plasma membrane for certain chemotaxis-inducing

factors, such as FMLP, C5a, and GP110 (Van Dyke et al. 1981, 1983, 1987). Agarwal et al. (1994) further suggested that cytokines, such as tumor necrosis factor (TNF) and interleukin-1 (IL-1), present in the serum of LJP patients are responsible for priming and inducing altered response in neutrophils. In the study, it was found that treatment of neutrophils in healthy subjects with LJP-sera resulted in a decreased neutrophil chemotactic response and down regulation of FMLP receptors on the cell surface. On the other hand, pretreatment of LJP-sera with anti-TNF and anti-IL-1 antibodies effectively neutralized the ability of LJP-sera to modulate chemotaxis and FMLP receptor levels in healthy subjects. Sigusch et al. (1998) found that EOP patients showed reduced expression of monocyte HLA-DR, IFN-γ, and IL-2 mRNA, and reduced proliferation of peripheral blood monocytes, which are known to be positively expressed by activated T_H1 cells. This indicates an impaired T_H1 cell response in EOP patients. Ozmeric et al. (1998) further suggested that the IL-8, a chemoattractant, produced by LJP patients may be less active than in healthy individuals.

The etiology of JP can be broadly divided into two categories, bacterial plaque and impaired host defense mechanism; therefore, the treatment should aim to control pathogenic microflora and compensate for impaired immune function. Systemic approaches such as antibiotic therapy are often indicated in the management of JP. In fact, several studies (Slots & Rosling 1983, Christersson et al. 1985, Kornman & Robertson 1985) showed that scaling and root planing alone failed to suppress or eliminate *A.a.* from the diseased site and failed to improve clinical conditions. Treatment modalities in the management of JP include mechanical therapy, such as scaling and root planing and periodontal surgery, local delivery of antimicrobials, and systemic antibiotics such as tetracycline, doxycycline, metronidazone, combination of metronidazole and amoxicillin, or combination of metronidazole and Augmentin (amoxicillin + clavulanic acid).

Gunsolley et al. (1994) compared nonsurgical and surgical periodontal treatment in 23 young patients with severe generalized periodontitis. They found that *P.g.* was virtually eliminated by scaling and root planing while levels of *A.a.* were not significantly reduced by scaling and root planing alone. However, after flap surgery, the levels of *A.a.* were significantly reduced, suggesting that surgery and/or antibiotics are necessary to reduce levels of *A.a.* in young patients with severe generalized periodontitis. This result was in agreement with other studies (Slots & Rosling 1983, Lindhe & Liljenberg 1984, Kornman & Robertson 1985).

Young individuals generally have excellent healing potential. Therefore, in JP patients the combination of systemic antibiotics and regenerative surgery is often successful in treating intrabony defects and early furcation involvement. Mattout and Roche (1984) found complete fill of furcations and significant supracrestal repair in an 18-year-old JP patient after treatment with iliac crest autografts. Mabry et al. (1985) treated intrabony defects in 16 LJP patients with a combination of freeze-dried bone allograft (FDBA) and tetracycline. The group that received systemic tetracycline therapy and local treatment with a mixture of tetracycline powder and FDBA had significantly better resolution of intrabony defects than the groups that received FDBA alone or debridement alone with/without systemic tetracycline. Contrary to these findings, DiBattista et al. (1995) found no statistically significant differences between flap debridement alone and various regenerative procedures in 7 LJP patients. All patients in this study received systemic doxycycline. The regenerative procedures utilized were ePTFE membranes, ePTFE plus root conditioning with doxycycline solution (pH: 2.5), and ePTFE + root conditioning + composite graft.

Based upon the contradictory results, it is apparent that the results achieved with regenerative procedures will vary depending on case selection, the procedure utilized and operator experience. Therefore, regenerative procedures should be selected based on the appropriate selection criteria.

(III). Common Intraoral Lesions

Dental care providers must be familiar with oral lesions that are commonly found in pediatric patients. The 6 most

Table 4. Therapeutic management of common pediatric oral lesions

Primary herpetic gingivostomatitis
- Topical anesthetics/coating agents:
  - 1 to 1 mixture of diphenhydramine (Benadryl) elixir & Maalox [OTC]
  - diphenhydramine (Benadryl)/lidocaine/Maalox mouth rinse
  - Acyclovir elixir
- Systemic acyclovir antiviral therapy

Recurrent herpes simplex
- Avoidance of perpetuating factors
- Acyclovir (Zovirax) 5% ointment or elixir
- Systemic acyclovir as active treatment or prophylaxis for immunosuppressed patients

Recurrent aphthous stomatitis
- Topical anesthetics/coating agents: Zilactin-B [OTC], Orabase-B [OTC], or Oraloe [OTC]
- Antimicrobial mouthrinses: 0.12% chlorhexidine gluconate
- Topical steroids: triamcinolone (Kenalog) in orabase 0.1%, betamethasone valerate 0.1% ointment, dexamethasone (Decadron) elixir 0.5 mg/5ml, or fluocinonide (Lidex) gel 0.05%

Candidiasis
- Topical antifungal agents:
  - Nysatin (Mycostatin) oral suspension 100,000 units/ml or popsicles
  - Clotrimazole (Mycelex) troches or swabs
- Antimicrobial mouthrinses: 0.12% chlorhexidine gluconate

Angular cheilitis
- Topical antifungal agents: nystatin or clotrimazole ointment
- Topical antifungal/steroid agent: nystatin/triamcinolone acetonide (Mucolog II) ointment
- Topical antifungal/antibacterial/steroid agent: hydrocortisone/iodoquinol (Vytone) cream 1%

Geographic tongue
- Topical anesthetic/analgesic mouth rinse: Ulcer-Ease [OTC]
- Topical antifungal/steroid agent: triamcinolone acetonide 0.1% in nystatin suspension
- Alkaline saline mouth rinse
- Topical anesthetics/coating agents: 1 to 1 mixture of diphenhydramine (Benadryl) elixir & Maalox [OTC]

commonly found pediatric oral lesions are: primary herpetic gingivostomatitis, recurrent herpes simplex infection, recurrent aphthous stomatitis, diffuse intraoral candidiasis, angular cheilitis, and geographic tongue. Table 4 summarizes the therapeutic management of each of these conditions.

Primary herpetic gingivostomatitis (PHG)

The primary etiology of this disease is herpes simplex virus (HSV) type 1. Viral transmission may occur via oral-genital or oral-oral direct mucocutaneous contact of infected secretions. The initial infection of PHG primarily affects children under 10 years of age with a peak incidence at 2–4 years of age, and secondarily young adults, aged 15 to 25 years (Main 1989). The incubation period of HSV infection ranges approximately 3 to 10 days. Clinical manifestations include fever, malaise, irritability, lymphadenopathy, widespread inflammation in the marginal and attached gingiva, and small clusters of vesicles throughout the mouth. The vesicles often coalescence and burst, forming large ulcers. It often causes severe pain and debilitation. Consequently, mastication and swallowing may be too painful, resulting in dehydration. This can be managed by supportive treatment with high oral fluid intake or intravenous fluid infusion (Dohvoma 1994). PHG is a contagious disease that usually regresses spontaneously within 12 to 20 days. Amir et al. (1997) reported that acyclovir oral suspension treatment (15 mg/kg 5 times a day) for herpetic gingivostomatitis, started within 3 days of onset, significantly reduced the duration of clinical manifestations and infectivity of affected children.

Recurrent herpes simplex (RHS)

Following primary infection by herpes simplex virus, the virus ascends through sensory or autonomic nerves and persists in neuronal ganglia. It becomes dominant within the nucleus and is present as a latent HSV (Corey & Spear 1986). In about 30 to 40% of population, secondary manifestations may occur as a result of precipitating factors such as sunlight, trauma, fever, immunosuppression, or stress. These secondary manifestations are recurrent herpes labialis, herpes genitalis, ocular herpes, and herpes encephalitis (Park 1988). Vesicles often develop at the same site and are usually present in small clusters following the distribution of the infected nerve. In healthy individuals, the disease is limited to periosteal-bound, keratinized mucosa whereas recurrences in the buccal mucosa and tongue may develop in immunocomprimised patients. The lesions are most frequently seen as herpes labialis, where lesions occur at the vermillion border of the lip.

Recurrent aphthous stomatitis (RAS)

RAS is the most common oral mucosal disease in North America (Murray & Amedee 2000). Approximately 20% of population experiences minor apthae, which is the most common form of childhood RAS (Field et al. 1992). These ulcerative lesions are often referred as ''canker sores'' by the patient. RAS lesions range from occasional small (0.5 to 1.0 cm in diameter), well-defined round or ovoid shallow ulcers with a gray-yellowish central area surrounded by an erythematous halo to larger (1 to 3 cm in diameter) oval or irregular ulcers. Small lesions heal in 7 to 10 days without scarring whereas larger lesions persist for weeks and heal with scarring (Eversole 1989). The etiology is unknown, yet suggested etiologies include the L-form of streptococcus and/or an immunopathic process involving cell-mediated cytolytic activity in response to HLA or foreign antigens. Precipitating factors may include trauma, stress, menstruation, nutritional deficiencies, food allergies, and endocrinopathies. In children with Behcet's disease and HIV infection, RAS lesions are frequently found (Krause et al. 1999, Ramos-Gomez et al. 1999). Clinical management of RAS may include mouthrinses such as chlorhexidine gluconate, topical corticosteroids, topical tetracyclines, immunomodulators, and others (Porter et al. 1998).

Candidiasis

Candidiasis is the most prevalent mycotic infection in the oral mucosa. It is caused by an overgrowth of a superficial fungus, *Candida albicans*, opportunistic fungus found in the oral cavity, gastrointestinal tract, and vagina. Clinically, it appears as diffuse, curdy, or velvety white mucosal plaques that can be wiped off. Infants whose mothers display vaginal thrush at the time of birth and adults on long-term antibiotics or steroid therapy are frequently affected. In addition, individuals with diabetes, hypoparathyroidism, immunodeficiency, or those undergoing chemotherapy are also often affected. Particularly, children and adolescents with HIV-infection are predisposed to the development of oral candidiasis (Flaitz & Hicks 1999). Like other forms of Candida infection, such as angular cheilitis and geographic tongue, the lesions can be managed by both topical and systemic treatment with antifungal medications, such as Nystatin ointment.

Angular cheilitis

Angular cheilitis, perleche, is a painful condition beginning as an inflammation of the commissure of the lips, followed by erosion, ulceration, and fissuring. It is known to be associated with *Candida albicans* and *Staphylococcus aureus.* The possible predisposing factors may include immunodeficiency, riboflavin (vitamin B_2) deficiency, trauma, and loss of vertical dimension. According to Flaitz & Hicks (1999), oral candidiasis, including angular cheilitis, is the most common oral manifestation in HIV-infected children. Treatment may include removal, if possible, of predisposing factors and application of antifungal medications.

Geographic tongue (benign migratory glossitis)

Geographic tongue is a benign inflammatory condition that is characterized by desquamation of superficial keratin and the filiform papillae. It affects approximately 1 to 2% of population. Kleinman et al. (1994) reported 0.6% prevalence of geographic tongue in 39,206 U.S. schoolchildren, aged 5–17 years. The etiology is unknown, but correlation with nutritional deficiencies and/or emotional stress has been suggested. The condition is often restricted to the dorsum and lateral borders of the tongue. Sometimes these usually benign lesions can become symptomatic exhibiting a burning sensation. Palliative treatment, including avoidance of acidic and spicy foods and drinks is advisable for those cases exhibiting symptoms. Topical and systemic antihistamine may be used to manage the lesions of geographic tongue (Sigal & Mock 1992).

Summary

Periodontal diseases are among the most frequent diseases affecting children and adolescents. Dental clinicians must be aware of the prevalence, diagnostic characteristics, microbiology, host-related factors, and therapeutic management of each of these disease entities. It is well known that the primary etiology of periodontal diseases is bacterial plaque. However, patients affected by early onset periodontitis (or aggressive periodontitis) often present with impaired immune function, mainly neutrophil dysfunction. Therefore, it is important when managing periodontal diseases in young individuals, the dentist should rule out systemic diseases that can affect host defense mechanisms. In addition, commonly found oral lesions such as primary herpetic gingivostomatitis, recurrent herpes simplex, recurrent aphthous stomatitis, diffuse intraoral candidiasis, angular cheilitis, and geographic tongue should be promptly identified and treated if necessary. It is believed that the best approach to manage periodontal diseases is prevention, followed by early detection and treatment. To achieve this, profound knowledge about periodontics and pedodontics as well as intimate periodontal-pedodontics interactions are essential.

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Zusammenfassung

Parodontale Erkrankungen bei Kindern und Heranwachsenden

Hintergrund: Parodontale Erkrankungen gehören zu den häufigsten Erkrankungen, die Kinder und Heranwachsende betreffen. Dies bezieht die Gingivitis, die lokalisierte oder generalisierte aggressive Parodontitis (früh einsetzende Parodontitis, die die generalisierte oder lokalisierte präpubertale Parodontitis und die juvenile Parodontitis einbezieht) und parodontale Erkrankungen verbunden mit systemischen Störungen ein. Der beste Weg für die Beschäftigung mit parodontalen Erkrankungen ist die Prävention, gefolgt von der frühen Entdeckung und der Behandlung. **Methoden:** Diese Arbeit bewertet die gegenwärtige Literatur die sich mit den häufigsten parodontalen Erkrankungen bei Kindern beschäftigt: chronische Gingivitis (oder Plaque induzierte gingivale Erkrankungen) und früh einsetzende Parodontitis (oder aggressive Parodontitis), einschließlich präpubertale und juvenile Parodontitis. Zusätzlich werden systemische Erkrankungen, die das Parodontium und die orale Schleimhaut betreffen und bei jungen Kindern gewöhnlich gefunden werden, behandelt. Die Prävalenz, die diagnostischen Merkmale, die Mikrobiologie, die Wirtsfaktoren und das therapeutische Management von jeder dieser Erkrankungen werden gründlich diskutiert.

Résumé

Maladies parodontales chez l'enfant et l'adolescent

Origine: Les maladies parodontales sont parmi les maladies les plus fréquentes affectant les enfants et les adolescents. Elles comprennent la gingivite, la parodontite agressive localisée ou généralisée (parodontite précoce qui inclut les formes généralisées ou localisées de parodontite prépubertaire et la parodontite juvénile) et les maladies parodontales associées aux problèmes systémiques. La meilleure approche pour traiter les maladies parodontales est la prévention suivie par une détection précoce et un traitement.

Méthodes: Ce manuscrit revoit la littérature actuelle concernant les maladies parodontales les plus communes affectant les enfants: gingivite chronique (ou maladie gingivale induite par la plaque dentair) et parodontite précoce (ou parodontite agressive) incluant les parodontites prépubertaire et juvénile. De plus, les maladies systémiques qui affectent le parodonte et les lésions orales communément trouvées chez les jeunes enfants sont reprises. La fréquence globale, les caractéristiques du diagnostic, la microbiologie, les facteurs de l'hôte et l'approche thérapeutique de chacune de ces entités de maladie sont discutés en profondeur.

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