

# Periodontal Disease: An Overview for Physicians

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## Abstract

Periodontitis is now seen as resulting from a complex interplay of bacterial infection and host response, often modified by behavioral factors. There has been a fundamental change in the prevailing periodontal disease model of the 1960s, which suggested that the susceptibility to periodontitis increases with age, and that all individuals are susceptible to severe periodontal disease. More recent research has changed the belief in universal susceptibility to the current view that only some 5B20% of any population suffer from severe generalized periodontitis, and that only moderate disease affects a majority of adults.

One major risk factor is smoking, as there is now a clear association between smoking and periodontal disease independent of oral hygiene, age, or any other risk factor.

In human periodontitis, there is no simple, direct pathogen-disease link. There are three pathogens that have a strong association with progressive periodontal disease: *Actinobacillus actinomycetemcomitans*, spirochetes of acute necrotizing gingivitis, and *Porphyromonas gingivalis*. These pathogens may be the cause of continued loss of periodontal attachment in all periodontal disease classifications despite diligent periodontal therapy. This loss of attachment, or destruction of the periodontal ligament and loss of adjacent supporting bone, is seen in adult periodontitis, as well as in early-onset periodontitis, which affects young persons who otherwise appear healthy. The three forms of early-onset periodontitis are prepubertal periodontitis, localized and generalized juvenile periodontitis, and rapidly progressive periodontitis. They are distinguished from adult periodontitis by the age of onset of the disease, the rapid rate of disease progression, manifestations of defects in host response, and the composition of the subgingival microflora. Prepubertal periodontitis is associated with attachment loss around teeth of the deciduous and/or permanent dentition, and is often associated with severe congenital defects of hematological origin, and alterations in neutrophil chemotaxis function.

Periodontitis may also be associated with systemic conditions such as metabolic disorders (diabetes mellitus, female hormonal alterations), drug-induced disorders, hematologic disorders/leukemia, and immune system disorders. These systemic disorders have been documented

as capable of affecting the periodontium and/or treatment of periodontal disease.

In order to rationally treat and prevent periodontal disease, we need to know the etiologic agents for specific patients, and the mechanism of bacterial pathogenesis in periodontitis. In systemic diseases in which the periodontal tissues are affected as well, early detection and carefully managed therapeutics with the physician and periodontist working together may prove beneficial to the patient's general health and quality of life.

**Key Words:** Adult periodontitis, early-onset periodontitis, prepubertal periodontitis, juvenile periodontitis, rapidly progressive periodontitis, *P. gingivalis*, *A. actinomycetemcomitans*

Periodontitis is now seen as resulting from a complex interplay of bacterial infection and host response, often modified by behavioral factors (1). Advances in research over recent years have led to a fundamental change in the periodontal disease model. The prevailing model of periodontal disease in the 1960s suggested that all individuals are susceptible to severe periodontitis, that gingivitis progresses to periodontitis with consequent bone and tooth loss, and that the susceptibility to periodontitis increases with age (2B4). All aspects of this model are under strong challenge from the results of more recent research. The belief in universal susceptibility has given way to the current view that only some 5%B20% of any population suffer from severe generalized periodontitis, even though moderate disease affects a majority of adults (5).

Risk factors, such as smoking, have a profound effect on the predisposition to periodontal disease. There is now a clear association between smoking and periodontal diseases, independent of oral hygiene, age, or any other risk factor (6). Studies have shown that there is no difference between smokers and nonsmokers in amounts of plaque accumulation, nor in the prevalence of the principal bacteria which are considered pathogenic for periodontitis (7, 8). What smoking appears to do is suppress the vascular reaction which follows gingivitis (9). In effect this is a masking effect on the signs of inflammation. Smoking may also be a factor in the association between refractory periodontitis and a polymorphonuclear leukocyte defect in the peripheral blood. There is also some evidence that smoking is associated with osteoporosis; the link with dental alveolar bone loss is currently under study (10, 11).

In human periodontitis, there is no simple, direct pathogen-disease link. More than 500 bacteria have been identified within periodontal pockets (12). Moreover, it is impossible to accurately associate the complex microbiota in a periodontal lesion, in a given patient, with the bacterial species that may have contributed to development of that lesion. Much evidence suggests, however, that a finite set of pathogenic microbiota, sometimes working alone, or in combinations, cause periodontal diseases in humans. Most pathogens associated with periodontal lesions are Gram-negative anaerobic rods, while some pathogens are also Gram-positive facultative and anaerobic cocci and rods, and Gram-negative facultative rods. Due to this mixed variety of pathogens, and their variability in sensitivity to antibiotics, simplistic antimicrobial chemotherapy

is problematic. Three pathogens have an especially strong association with the presence of progressive periodontal disease: *Actinobacillus actinomycetemcomitans*, spirochetes of acute necrotizing gingivitis, and *Porphyromonas gingivalis* (13). One potential virulence factor recently ascribed to *P. gingivalis* and *A. actinomycetemcomitans*, which is shared by a number of respiratory and enteric pathogens, is the ability to enter mammalian cells (14). These pathogens are very often the cause of continued loss of periodontal attachment despite diligent conventional mechanical periodontal therapy, as well as causing refractory periodontitis (15), localized juvenile periodontitis, and other types of early-onset periodontitis (16). Patients who suffer from these conditions may benefit from antibiotic therapy.

Evaluation of the patient's periodontal status requires obtaining a relevant medical and dental history and conducting a thorough clinical and radiographic examination, with evaluation of extraoral and intraoral structures. A medical history should be taken and evaluated to identify predisposing conditions that may affect treatment, patient management and outcomes. Such conditions include, but are not limited to, diabetes, hypertension, and pregnancy. Factors which may also play a role in treatment outcome are smoking, substance abuse and medications.

Adult periodontitis is defined as inflammation of the gingiva and the adjacent dental attachment apparatus (17). The disease is characterized by loss of clinical attachment due to destruction of the periodontal ligament and loss of the adjacent supporting bone (18). As mentioned previously, dental plaque bacteria play a key role in what is now understood to be the complex process by which the common types of oral diseases occur C dental caries and periodontal diseases (18). As with other infectious diseases, there is a balance between the host immune responses on one hand, and the microbial pathogenesis on the other hand. In health, host immune responses are sufficient to hold in check the pathogenic potential of both the normal resident microbial flora and exogenous microbial pathogens. Infectious diseases such as periodontal disease occur when this equilibrium is disturbed.

Clinical features may include combinations of the following signs and symptoms: edema, erythema, gingival bleeding upon probing, and/or suppuration (19) (Fig. 1). Adult periodontitis with slight to moderate destruction is characterized by a loss of up to 1/3 of the supporting periodontal tissues; a loss of over 1/3 of the periodontal supporting tissues is seen in advanced adult periodontitis. Radiographic evidence of bone loss is apparent in advanced adult periodontitis, and may be evident in adult periodontitis with slight to moderate destruction. Adult periodontitis with slight to moderate or advanced loss of periodontal supporting tissues may be localized, involving one area of a tooth's attachment, or more generalized, involving several teeth or the entire dentition (19). A patient may simultaneously have areas of health and adult periodontitis with slight, moderate, and advanced destruction.



**Fig. 1** Adult periodontitis C heavy deposits of plaque and calculus resulting in inflammation and bone loss.

The therapeutic goals of periodontal therapy are (1) to alter or eliminate the microbial etiology and contributing risk factors for periodontitis, thereby arresting the progression of disease and preserving the dentition in a state of health, comfort, and function with appropriate esthetics; and (2) to prevent the recurrence of periodontitis (19). In addition, regeneration of the periodontal attachment apparatus, where indicated, may be attempted.

Clinical judgment is an integral part of the decision-making process. Many factors affect the decisions for appropriate therapy(ies) and the expected therapeutic results. Patient-related factors include systemic health, age, compliance, therapeutic preferences, and patient=s ability to control plaque. Other factors include the clinician's ability to remove subgingival deposits, prosthetic demands, and the presence and treatment of teeth with more advanced adult periodontitis. In general the treatment should include oral hygiene instruction, and reinforcement and evaluation of the patient=s plaque control; mechanical therapy, i.e., supra- and sub-gingival scaling and root planing to remove microbial plaque and calculus; and control of other local factors. If this initial therapy resolves the periodontal condition, supportive periodontal therapy should be scheduled at appropriate intervals; but if the periodontal condition is not resolved, periodontal surgery should be considered to correct anatomic defects, and/or to regenerate hard and soft tissues.

Early-onset periodontitis encompasses distinct types of periodontitis that affect young persons who, in most cases, otherwise appear healthy. Early-onset periodontitis may be distinguished from adult periodontitis by the age of the disease onset, the rapid rate of the disease progression, manifestations of defects in host response, and composition of the associated subgingival microbial flora (19). Early-onset periodontitis includes three forms: prepubertal periodontitis, localized and generalized juvenile periodontitis, and rapidly progressive periodontitis (20).

There is evidence that genetic factors influence susceptibility to the different forms of early-onset periodontitis. However, it is unlikely that a specific gene will be identified as causing enhanced disease susceptibility. It is more likely that the genetic influences are as multifactorial as the diseases themselves, and a complex interplay between genetically determined host responses and environmental challenges may determine whether disease is present (21).

Prepubertal periodontitis is associated with attachment loss (gingival pocket formation and radiographic evidence of bone loss) around teeth of the deciduous and/or permanent dentition (22). It can occur between the time of tooth eruption and the beginning of puberty. As a consequence of this destruction, exfoliation of the deciduous teeth starts prior to the eruption of the permanent dentition. Prepubertal periodontitis may occur in either generalized or localized forms. The generalized form is most frequently associated with severe congenital defects of hematological origin and is usually accompanied by alterations in neutrophil chemotaxis function. Some forms of prepubertal periodontitis can be considered to be complications of a severe systemic disorder, such as acrodynia, cementopathia, Chédiak-Higashi syndrome, chronic neutropenia, histiocytosis X, HIV infection, hypophosphatasia, leukemia, Papillon-Lefèvre syndrome, and fibrous dysplasia (23) (Fig. 2). The localized form is usually not associated with a systemic disorder, and presently there is no



**Fig. 2.** Prepubertal periodontitis C severe bone loss of the primary dentition in a child diagnosed with AIDS.

evidence that localized prepubertal periodontitis will carry over to the permanent dentition.

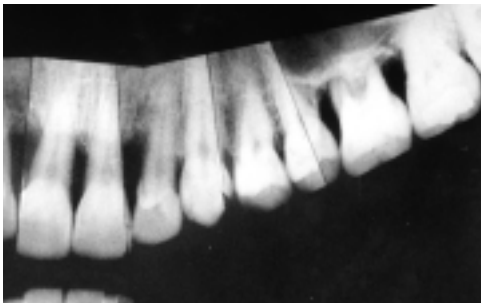
In most reported cases of generalized prepubertal periodontitis with congenital defects of

hematologic origin, the disease could not be arrested unless all the teeth were extracted (23). There are very few reports concerning the treatment of localized prepubertal periodontitis in which aggressive treatment consisted of extraction of hopeless teeth, scaling and root planing, daily subgingival irrigation, and antibiotic coverage. In general, the data support conservative supportive treatment of localized prepubertal periodontitis, which includes mechanical therapy, antibiotic coverage, and maintenance.

Juvenile periodontitis also presents in either localized or generalized forms (17). Generalized juvenile periodontitis (GJP) usually occurs in the late teenage years and affects most teeth. The disease has been associated with a variable microbial etiology that may include *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* (24B26). Contributing risk factors such as smoking should be considered. Localized juvenile periodontitis (LJP) has an age of onset at or around puberty and is associated with molar and incisor bone and attachment loss (Figs. 3 and 4). However, atypical patterns of disease have been observed. The localized form is frequently characterized by association with the periodontal pathogen *Actinobacillus actinomycetemcomitans* and neutrophil function abnormalities (27). Both GJP and LJP may exhibit abnormalities in host immune cell functions that appear to follow a familial pattern.



**Fig. 3.** Juvenile periodontitis C typical bone loss associated with the central incisors and first molars.



**Fig. 4.** Juvenile periodontitis C surgical exposure exemplifying the severe bone loss associated with the first molar.

The goal of treatment in early-onset periodontitis is to alter or eliminate the microbial etiology and the contributing risk factors, and faster regeneration of the periodontal apparatus. Due to the complexity of this type of periodontal disease with respect to systemic factors, immune defects, and the microbial flora, control of the disease may not be possible in all instances. In such

cases, a reasonable treatment objective is to slow the progression of the disease by administering the appropriate antibiotic regimen, and providing repeated microbiological testing and an intensified, supportive periodontal therapy program.

Rapidly progressive periodontitis (RPP) is typically found in patients 20-35 years old. With the exception of the age of onset, the clinical, microbiological, and immunologic diagnostic findings in RPP are similar to those in GJP (17).

The goals of periodontal therapy are similar to those in the treatment of adult periodontitis, including attempting the regeneration of the periodontal attachment apparatus where indicated. Due to the complexity of the early-onset periodontal diseases with regard to systemic factors, immune defects, and the microbial flora, control of the disease may not be possible in all instances. In such cases, a reasonable treatment objective is to slow the progression of the disease.

In general, treatment methods for early-onset periodontal diseases may be similar to those used for adult periodontitis. These methods should include oral hygiene instruction, and reinforcement and evaluation of the patient's plaque control; supra- and sub-gingival scaling and root planing to remove microbial plaque and calculus; control of other local factors; occlusal therapy as necessary; periodontal surgery as necessary; and supportive periodontal therapy. In addition, a general medical evaluation may determine if systemic disease is present in children and young adults who exhibit severe periodontitis, particularly if early-onset periodontitis appears to be resistant to therapy (28). Consultation with the patient's physician may be indicated to coordinate medical care in conjunction with periodontal therapy. In the early stages of disease, lesions may be treated with adjunctive antimicrobial therapy combined with scaling and root planing with or without surgical therapy. Microbiological identification and antibiotic sensitivity testing may be considered. The long-term outcome may depend upon the patient's compliance, and delivery of supportive periodontal therapy at appropriate intervals, as determined by the clinician. If primary teeth are affected, eruption of permanent teeth should be monitored to detect possible attachment loss.

### **Periodontitis Associated with Systemic Conditions**

A number of systemic factors have been documented as capable of affecting the periodontium and/or treatment of periodontal disease. Systemic etiologic components may be suspected in patients who exhibit periodontal inflammation or destruction which appears disproportionate to the local irritants. Periodontal therapy may be modified based on the current medical status of the patients. Periodontal organisms may be the source of infections elsewhere in the body. Therefore, those infections may also affect systemic health.

The therapeutic goal is to achieve a degree of periodontal health consistent with the patient's overall health status. Achieving this goal, however, may be directly affected by the degree of control of the systemic condition. The systemic and psychological status of the patient should be identified, therefore, to reduce medical risks that may compromise or alter the periodontal treatment.

Patients with systemic conditions that contribute to progression of periodontal diseases may be successfully treated using established periodontal treatment techniques. However, the systemic/psychologic status of the periodontal patient may alter the nature of therapy rendered and may adversely affect treatment outcomes.

## **Metabolic Disorders**

### **Diabetes Mellitus**

Patients with undiagnosed or poorly controlled insulin dependent diabetes mellitus (IDDM) or non-insulin dependent diabetes mellitus (NIDDM) are at risk for periodontal disease. Periodontitis also progresses more rapidly in poorly controlled diabetics (29), and early age of onset of the disease is seen as a risk factor for more severe disease (30). Conversely, most well-controlled diabetic patients can maintain periodontal health and will respond favorably to periodontal therapy. Metabolic control is a prime factor in maintaining periodontal health among NIDDM patients (31B33), and there is some evidence for the converse, namely that periodontal health may affect diabetic control (34).

The suggested mechanisms by which diabetes may contribute to periodontitis include vascular changes, polymorphonuclear cell dysfunction, abnormal collagen synthesis, and genetic predisposition (35B37). While the mechanism by which diabetes exacerbates periodontal destruction is still not fully understood, periodontitis can be considered a complication of both types of diabetes.

Nearly all diabetics seem to respond to conventional periodontal therapy. Since diabetics are more prone to infections, adjunct prophylaxis may be necessary if surgical therapy is considered. To date, there are no reports of the specific use of antibiotics in diabetics and their effects on the outcome of therapy. More important, despite the modality of treatment chosen, the age of the patient, amount of plaque and calculus, and the level of diabetic control need to be taken into consideration.

### **Female Hormonal Alterations**

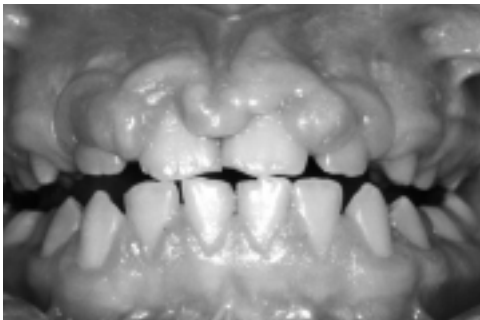
Hormonal fluctuations in the female patient may alter the status of periodontal health (38, 39). Such changes may occur during puberty, the menstrual cycle, pregnancy, or menopause. Changes may also be associated with the use of oral contraceptives. The most pronounced periodontal changes occur during pregnancy, as a significant proportion of pregnant women suffer from pregnancy gingivitis (40). This transient and self-limiting condition includes an increase in bleeding, gingivitis, and a subgingival microbial shift. Gingival tissues return to their original healthy state postpartum when estrogen and progesterone levels reach baseline values. This reversal trend seems to parallel that observed in pubescent females. Aside from these transient changes, pregnant women in good health are unlikely to experience any significant gingival response that would have serious clinical implications. Women who are susceptible or have a preexisting gingival condition should seek treatment to prevent extension of the inflammatory process, especially to avoid periodontal abscess formation that could cause a bacteremia. In general, pregnant women should note that preventive measures consisting of dental prophylaxis and meticulous plaque control help to prevent any periodontal condition from developing.

Women on hormonal replacement therapy and oral contraceptives experience a statistically significant increase in gingival inflammation (41, 42). With oral contraceptives, this increase in gingival inflammation is related to the duration of use, and results of recent studies suggest that prolonged use of oral contraceptives may detrimentally affect the periodontium (43).

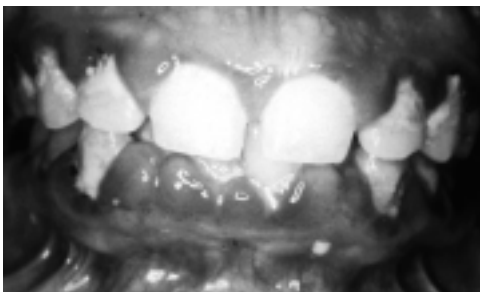
In menopausal and/or postmenopausal women, osteoporosis is not considered an etiologic factor in periodontitis, though it may affect the severity of the disease in preexisting periodontitis (44). In general, women with preexisting gingival conditions or susceptibility to periodontal disease may experience an exacerbated response to bacterial plaque if they are pregnant, use oral contraceptives, have hormonal replacement therapy or undergo menopause. Given the effects of hormonal changes and their corresponding impact on the periodontium, proper periodontal evaluation and treatment for these women is recommended. For most healthy women without the predisposition to periodontal disease, the negative influence of the changes in estrogen and progesterone levels can be controlled by additional plaque control, and these women should not be considered to be at increased risk of periodontal disease.

### **Drug-Induced Disorders**

Drugs can be a contributing etiologic factor in periodontal diseases. Drugs such as anticonvulsants, calcium channel blocking agents, and cyclosporine may induce gingival overgrowth (45) (Figs. 5 and 6). In addition, drugs can cause xerostomia, osteoporosis, lichenoid, and other hypersensitivity reactions. It is important, when possible, to have a baseline periodontal evaluation prior to initiation or modification of drug therapy. If there is gingival overgrowth, or other adverse reactions, there will be a need for modification of the drug regimen prescribed. Often surgery is necessary to eliminate gingival overgrowth, and this overgrowth may easily recur if drug therapy



**Fig. 5.** Gingival overgrowth associated with the use of dilantin.

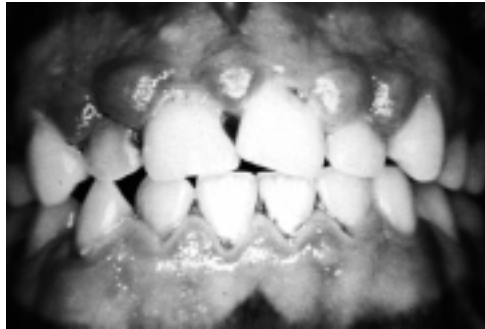


**Fig. 6.** Gingival overgrowth caused by cyclosporine in a patient with a kidney transplant.

cannot be modified, and/or if there is not adequate plaque control on the part of the patient.

### **Hematologic Disorders / Leukemia**

Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia (46) (Fig. 7). Patients with chronic leukemia may experience



**Fig. 7.** Patient diagnosed with leukemia and associated hemorrhagic gingival overgrowth.

similar but less severe periodontal changes. Chemotherapy or therapy associated with bone marrow transplantation may also adversely affect the gingiva (46, 47). Considerations for patients with hematologic disorders and periodontal disease should include coordination of treatment with the patient's physician. Prior to the treatment of leukemia and/or transplantation, appropriate periodontal therapy is rendered for minimization of sites of periodontal infection. If there is bone marrow transplantation, the patient should be monitored for evidence of host-versus-graft disease and drug-induced gingival overgrowth.

### **Immune System Disorders**

Some forms of periodontal disease may be more severe in individuals with immune systems disorders. Patients infected with Human Immunodeficiency Virus (HIV), may have especially severe forms of periodontal disease, and this periodontal manifestation may often be the first clinical expression of the virus itself (48). The likelihood of acute necrotizing gingivitis and necrotizing periodontitis may increase in the patient with acquired immunodeficiency syndrome (AIDS) (49).

Patients who have received organ transplants or are undergoing cancer treatment may be taking immunosuppressive medications. There are special considerations for immune system disorder patients with periodontal disease. Aside from being aware of the patient's immune status, the patient must be closely monitored for controlling associated mucosal diseases and acute periodontal infections.

The usual treatment involves gross scaling to remove visible plaque and calculus deposits, and the debridement of necrotic tissue when present. Following initial debridement, follow-up visits are necessary for close monitoring and evaluation of the oral tissues and the patient's plaque control, as well as removal of additional deposits. Home use of an antimicrobial mouth rinse such as chlorhexidine has been shown to be effective in reducing the acute symptoms and recurrence of lesions. Antibiotics should be used with caution, due to the increased risk of overgrowth of *Candida albicans* and other microflora associated with the HIV infection. The use of a concurrent antifungal

agent has been recommended, as well as narrow spectrum antibiotics such as metronidazole, which leave the aerobic Gram positive flora undisturbed (50). The response to therapeutic intervention may, however, depend upon the patient's current immune status and/or HIV stage, intake of systemic medications, intake of antibiotics, and oral habits (e.g., tobacco smoking).

### **Conclusion**

While many patients respond well to conventional periodontal therapy, others do not. Strategies that have improved our understanding of how bacteria cause medically important infections are starting to be successfully applied to examine mechanisms of bacterial pathogenesis in periodontitis. In order to rationally treat and prevent recurrence of periodontal diseases, we need to know the identity of etiologic agent(s) for specific patients, and the virulence properties of the pathogenic species involved. Future studies are necessary to determine these answers, and improve our understanding of the mechanism of bacterial pathogenesis in periodontitis. This knowledge, in turn, should lead to the development of highly specific antimicrobials to prevent and treat periodontal diseases.

What becomes evident from this review is that the periodontal tissues are often associated with manifestations of a wide range of systemic diseases. Early detection and carefully managed therapeutics with the physician and periodontist working hand-in-hand may prove beneficial to the patient's general health and quality of life. It is important that physicians recognize and familiarize themselves with these possible oral manifestations, and refer for the treatment of the periodontal disease. When there are signs of destructive oral disease of the hard and soft tissues, it is important that the dentist or periodontist determine if there may be an underlying systemic factor requiring referral to the physician.

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