

An Overview of Gingival and Periodontal Diseases

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Background

Over the decades, there has been an evolution in the etiological theories of periodontal disease. Prior to 1960, the calculus theory was widely accepted, believing that periodontal disease was caused by the presence of calculus acting as a mechanical irritant. This theory was replaced by the non-specific plaque theory (1965-1975) which thought periodontal disease was caused by bacteria in the plaque and that all bacteria were harmful; emphasizing that the quantity, not the composition, of bacterial plaque determined the development and severity of the disease. Research soon led to the specific plaque theory (1975-1985) which discovered that specific bacteria along with their pathogenic potential were more important than the actual amount of plaque present in the development and severity of the disease. Today, the host-bacterial interaction theory is accepted as the current etiological basis for the disease. According to Jill Nield-Gehrig, "A bacterial infection alone is insufficient to result in periodontal disease. The host response plays a critical role in the tissue destruction seen in periodontitis. Current research findings suggest that everyone is not equally susceptible to periodontal disease. Some individuals are more at risk than others."³ We now know that although specific pathogenic bacteria cause periodontal disease, the extent, severity, and progression of the disease depend on the host's response to the pathogen.

In 1999 the International Workshop for a Classification of Periodontal Diseases and Conditions introduced a new classification system for periodontal diseases. The previous classification system had been in use since 1989 and was thought to be lacking. In Gary Armitage's

position paper on the periodontal disease classifications, four deficiencies in the previous system were found to be "considerable overlap in disease categories, absence of a gingival disease component, inappropriate emphasis on age of onset of disease and rates of progression, and inadequate or unclear classification criteria."¹ The purpose of this continuing education self-study course is to provide an overview of the current classifications of gingival and periodontal diseases. Upon completion of this study, participants will have a clearer understanding of the patient population, clinical manifestations, etiology of disease and associated bacteria where applicable, as well as treatment recommendations. This information is based on the classifications outlined by the American Academy of Periodontology and the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions, Armitage's *Development of a Classification System for Periodontal Diseases and Conditions* published in December of 1999 in the *Annals of Periodontology* (<http://www.perio.org/resources-products/classification.pdf>), the American Academy of Periodontology (www.perio.org), and *Periodontology for the dental hygienist*.^{1,6}

Introduction

The present classification system is no longer based on the age of the patient but includes the etiology of the disease, clinical manifestations, pathogenesis and progression of the disease, as well as how the disease responds to therapy.^{2,6} Major changes to this new classification system included the adoption of a section on gingival diseases, replacement of the classification "Adult Periodontitis" with "Chronic Periodontitis" as well as "Early-onset

Periodontitis" with "Aggressive Periodontitis". In addition, Refractory Periodontitis is no longer a separate category but rather experts believe that this form of the disease has the possibility to recur in all classifications of the diseases. Other revisions include the replacement of Necrotizing Ulcerative Gingivitis with Necrotizing Ulcerative Diseases.¹

Oral Cavity in Health

In health, the gingiva should appear pink or coral pink with no swelling and lie snugly around the tooth. The consistency is firm and resilient and usually has a stippled appearance. The gingival contour should be scalloped and knife edged with the papillae coming to a point and filling the interdental spaces. In health, sulcus depths measure 3 mm or less and there is no presence of bleeding. The bacteria present are relatively simple and sparse and consist mostly of gram-positive, facultative cocci and rods, *Streptococci* and *Actinomyces*.⁶

Gingivitis versus Periodontitis

Gingivitis is the most common disease in humans. This disease is the easiest to treat and control, often painless (although some tenderness or discomfort may be present at times), and often unrecognized, dismissed by patients even with the presence of bleeding, and so common that the dentist and hygienist downplay the severity and habitually do not emphasize the importance.⁶

Gingivitis is defined as inflammation of the gingiva but the infection has not progressed with attachment loss. There is a change in the color from pink to red as well as the presence of bleeding, edema, and occasionally exudate. Gingivitis differentiates into periodontitis when the infection progresses beyond the gingiva into the periodontal ligament, cementum, and alveolar bone causing attachment loss in the periodontium.

Gingivitis Associated with Dental Plaque Biofilm Only

Plaque-induced gingivitis is the most common form of gingival diseases encountered in the dental office today. The patient population is non-specific and affects both adolescents and adults. Clinical manifestations of this disease include gingival redness, bleeding, and enlargement of gingiva. There are no signs of clinical attachment loss and bone loss. Gingivitis of this nature is directly related to inadequate oral hygiene and the amount of plaque biofilm present on the teeth. This bacterial plaque is considered a non-specific plaque biofilm because although these bacteria are abundant, the specific bacteria are not the causative agent for the disease. The organisms identified are predominantly Gram-



Figure 1 Plaque-induced gingivitis

negative, anaerobic bacteria and include *Streptococcus sanguis*, *S. mitis*, *Fusobacterium*, *Actinomyces viscosus* and *Veillonella parvula*. The treatment modality of choice is an oral prophylaxis along with personalized oral hygiene instructions. Patients who present with this condition are capable of an excellent prognosis since the disease is completely reversible and curable with the daily removal of dental plaque along with periodic recall visits to remove calculus and other irritants. (See Figure 1)

Gingival Diseases Modified by Systemic Factors:

Association with the Endocrine System

Systemic factors can affect how a person's immune system reacts to the presence of bacterial plaque. Many of these factors in and of themselves are not responsible for causing gingivitis but can intensify it in certain susceptible individuals resulting in the development of the disease. Hormonal imbalances due to puberty, menstruation, pregnancy, oral contraceptive use, menopause, and diabetes are examples of systemic conditions. Other contributing systemic factors include stress and blood dyscrasias such as leukemia.

Puberty, Pregnancy, and Menstrual Gingivitis: The patient population varies for each form. For example, puberty gingivitis

occurs in adolescent boys and girls as they enter puberty whereas menstrual gingivitis is seen in women during the menstrual cycle. Pregnant women as well as menopausal women can also experience forms of gingival diseases intensified by their conditions. These diseases tend to manifest similarly clinically with gingival redness, bleeding, edema and gingival enlargement associated with proliferation of blood vessels and may present as either marginal or diffuse, and localized or generalized. Changes in the levels of circulating estrogen and progesterone can lead to gingival hyperplasia. The systemic factors of puberty, pregnancy, and menstruation along with the subgingival growth of pathogenic bacteria and an exaggerated response to bacterial plaque are thought to be the cause of these diseases. Bacteria organisms associated with these diseases are primarily Gram-negative, anaerobic non-motile species including *Prevotella intermedia*, *Bacterioides*, *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*. Treatment includes scaling and debridement as needed to remove plaque and calculus. In some cases, hormone therapy and medications may need to be modified. Pregnancy gingivitis is most common during the second trimester of pregnancy due to the increase in hormone levels which can result in an increase of gingival inflammation. This condition may be present in individuals with excellent plaque control; however, poor oral hygiene habits can exacerbate the condition. Changes in the gingiva may increase as the pregnancy progresses. Usually, the condition resolves itself with thorough home care and removal of plaque and calculus; however, it is common for it to remain until after the birth of the baby and hormone levels return to normal. *Prevotella intermedia* are the most abundant bacteria present. Severe cases of pregnancy gingivitis may progress to form a pyogenic granuloma or pregnancy tumor. A pregnancy tumor is not a true tumor but instead a localized, mushroom-shaped gingival mass of pyogenic granulation tissue that is the result of an exaggerated inflammatory response to an irritant. These masses can develop at any time during pregnancy and occur more commonly in the maxilla and interproximally. They bleed easily and can cause mobility and migration of the affected teeth. Pregnancy tumors usually regress after childbirth, but on occasion must be surgically removed.

Gingival Diseases Modified by Medications

Patients taking anticonvulsants (phenytoin), calcium channel blockers (nifedipine and verapamil), or immunosuppressants (cyclosporine) are susceptible to developing gingival diseases modified by medications. Although all age groups taking these medications are susceptible, there is a higher prevalence in children than adults. Clinical symptoms include gingival hyperplasia, fibrotic gingival tissues, pseudopockets, and often heavy calculus formation and increased levels of inflammation. Anterior teeth are most commonly affected, but symptoms can occur in the posterior sextants, too. Drug-induced gingivitis does not require plaque-induced inflammation in order to develop. Systemic drug administration is the known etiology of this disease; however, like the previously mentioned diseases, plaque can exaggerate the response to the disease. There are no known bacterial organisms associated with these conditions. Treatment consists of removal of bacterial plaque and calculus deposits, gingival stimulation and plaque control by patient. Alteration of drug therapy and gingivectomy may be necessary in severe cases.

Gingival Diseases Modified by Malnutrition

Serious nutritional deficiencies in vitamins A, B₁, B₂, B₆, and C can modify the body's response to bacterial dental plaque. Some examples of possible patient populations affected by this disease classification include infants from low socioeconomic status, institutionalized elderly, homeless, alcoholics, and underdeveloped poor counties. Clinical manifestations of vitamin C deficiency (scurvy) include very hemorrhagic and swollen gums which rapidly progresses to advanced periodontitis resulting in severe bone loss and mobility of teeth. The etiology of the disease is malnutrition and vitamin deficiencies and is not associated with any bacterial organisms. Treatment includes prophylaxis with nutritional counseling and a balanced diet replacing missing vitamins.

Chronic Periodontitis

Chronic periodontitis, previously known as adult periodontitis, is the most common form of periodontal disease encountered in private practice on a day-to-day basis. This disease entity is most commonly seen in adults over 35, but can occur at any age, including in children and adolescents. Chronic periodontitis can present itself as either localized or generalized and can vary from person to person and site to site in the same mouth. Another characteristic of this disease is the slow to moderate progression intermixed with periods of rapid progression. Clinical features include edema, redness, gingival bleeding, periodontal pockets, bone loss, tooth mobility, suppuration, dental calculus and plaque, and attachment loss. The etiology of the disease is the interaction between the host response and the bacterial plaque biofilm and can be modified by other factors including systemic diseases, stress and cigarette smoking. *Porphyromonas*



Figure 2. Chronic Periodontitis

gingivalis is the bacteria predominantly isolated in chronic periodontitis. Other organisms associated include *Tannerella forsythensis*, *Actinobacillus actinomycetemcomitans*, and *Treponema denticola*. Less virulent bacteria include *Campylobacter rectus*, *Eubacterium nodatum*, *Fusobacterium nucleatum*, *Prevotella intermedia*, and *Peptostreptococcus micros*. Treatment includes periodontal debridement, prophylaxis, and/or scaling and root planning, oral hygiene instructions on proper daily plaque biofilm removal by patient to be reinforced at each subsequent appointment during phases I, III, and IV of therapy, antimicrobial agents, control of associated factors such as overhanging margins; periodontal surgery (if indicated), smoking cessation, and routine periodontal maintenance appointments. (See Figure 2)

Periodontitis as a Risk Factor for Systemic Disease

Research continues to demonstrate that periodontal disease acts as a risk factor for certain systemic diseases including cardiovascular diseases, preterm and low birth-weight babies, and bacterial pneumonia, diabetes, and osteoporosis. "According to the third National Health and Nutrition Examination Survey conducted by the National Health Institute ... a 14% increase in the odds of heart attack for every patient with a 10% increase in sites with 3 mm or more attachment loss."⁶

In pregnant women with periodontal disease, inflammatory mediators cross the placenta and directly affect the fetus. Furthermore, endotoxins (LPS) from the bacterial plaque attack the placenta leading to the inflammatory response that elicits uterine contractions and pre-term birth. Evidence supports that active periodontal disease accounts for 18% of all pre-term, low birth-weight babies, which is more significant than either alcohol or cigarette use.^{5, 6}

Research is ongoing to find the correlation between periodontal disease and osteoporosis. So far evidence is limited and the findings are inconsistent. This continues to be an area of ongoing investigation into the possible linkage of the two disease entities.

Periodontitis as a Manifestation of Systemic Disease

Research continues to demonstrate that some systemic diseases alter both presentation and progression of periodontitis by increasing the severity and character of periodontal disease as well as weakening the person's resistance to all other disease processes.⁶ A red flag to clinicians should be a severity of periodontal disease that exceeds beyond the normal expectations based on the amount of plaque and calculus present. When this occurs, the recommendation of a thorough medical examination should be suggested. A good example of this is leukemia. Dental hygienists and dentists are often the first to notice the signs of this disease because oral manifestations are often the initial signs of leukemia.

Systemic diseases that manifest periodontal disease include Down syndrome, diabetes, AIDS, Papillon-Lefevre syndrome, and leukemia. Diabetes increases the patient's susceptibility to many infections. With periodontal infections, diabetics tend to have an increase in

CAL, especially if the individual is also a smoker. The attachment loss is typically localized, sporadic, and rapid and is usually the first sign of diabetes. Patients who are able to control their diabetes have a better prognosis through treatment than those whose diabetes remains uncontrolled.

Papillon-Lefevre syndrome is a rare and devastating condition that produces areas of thick, cracked skin on the soles, knees, palms, and elbows. This condition typically affects children and young adults and manifests orally with rapid tooth loss and severe periodontitis.

Clinical features of this classification of periodontal disease mimic chronic or aggressive periodontitis. The systemic diseases in and of themselves act as the contributing factors to the disease and interfere with the body's resistance to infection, thus a reduced host response. No known organisms are associated with this disease entity. Treatment includes an increased level of personal plaque control and more frequent periodontal recall visits.

Prepubertal periodontitis is a rare condition seen in children that can occur as either localized or generalized. This condition is unique in that both deciduous and permanent teeth may be affected. Clinical features include severe gingival inflammation, rapid bone loss, tooth mobility, and early tooth loss. This periodontal infection causes primary teeth to be lost prematurely and the secondary dentition is infected during eruption, usually resulting in the loss of all their teeth during childhood.⁶ The etiology of the disease is unclear at this time but is thought to be contributed to defective polymorphonuclear leukocytes (PMNs or neutrophils), mononuclear leukocytes, or systemic disease. Previously, this disease was classified with the juvenile aggressive periodontal disease, however, with the present classification system, it is considered as a manifestation of systemic diseases. Bacterial organisms are primarily Gram-negative and include *Prevotella intermedia*, *Capnocytophaga sputigena*, and *Eikenella corrodens*. Treatment is experimental at this time and referral to a specialist is indicated due to the unresponsive nature of this disease to conventional treatments such as SRP, surgery, and antibiotics.

Aggressive Periodontitis

Aggressive periodontitis, previously known as juvenile periodontitis, is characterized by a rapid progression with massive bone loss possibly due to defects in the immune system. Many of the diseases under this classification respond poorly to conventional treatment, such as antibiotics which tend to slow, rather than arrest disease progression. Another characteristic of aggressive forms of periodontal disease is that the level of severity of the disease is not consistent with the amount of plaque and calculus present. Aggressive periodontitis includes prepubertal periodontitis, localized aggressive periodontitis, and rapidly progressive periodontitis.

Localized aggressive periodontitis occurs in otherwise healthy children and teenagers. LAP manifests clinically with a bilaterally symmetric, localized rapid destruction of the periodontal attachment over a short period of time around the permanent incisors and first molars.

Gingiva may be normal in appearance but deep pockets are found on affected teeth and severity of destruction is not consistent to the relatively sparse dental plaque and lack of clinical inflammation. These individuals generally have a low caries rate. The cause of the disease is believed to be genetically based with a possible defect in the immune system related to depressed neutrophil chemotaxis and phagocytosis. This disease is not plaque induced; however, bacterial organisms have been associated with the disease. LAP is dominated by the Gram-negative rod, *Actinobacillus actinomycetemcomitans*. Other commonly isolated bacteria include *Actinomyces naeslundii*,



Figure 3
Localized Aggressive Periodontitis

Fusobacterium nucleatum, and *Campylobacter rectus*. Treatment includes mechanical debridement with scaling and root planing, systemic antibiotics (Tetracycline), and periodontal surgery. (See Figure 3)

Generalized aggressive periodontitis is similar to LAP in that presents in otherwise healthy children and teenagers with a rapid destruction of periodontal attachment over a short period of time. However, it differs from LAP in that destruction involves most, if not all, the teeth. Similar to LAP, GAP is thought to be associated with a depressed neutrophil chemotaxis and pathogenic bacteria. Another clinical difference from LAP is that a significant amount of clinical inflammation along with heavy plaque and calculus formation are present with this type of periodontal infection. In most instances, if left untreated disease will progress to loss of all teeth; however, on rare occasions, progression of attachment loss may suddenly cease. The bacterial organisms are also different from the localized form and include *Porphyromonas gingivalis*, *Prevotella intermedia*, *Eikenella corrodens*, and *Actinobacillus actinomycetemcomitans*. Treatment includes improved home plaque biofilm control, scaling and root planing, systemic antibiotic therapy such as Tetracycline, and periodontal surgery.

Necrotizing Periodontal Diseases

Under the current classification system, necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP) are placed under the category "Necrotizing Periodontal Diseases". This classification gave workshop participants a dilemma because there is still so much to be learned about these two diseases. For example, NUG and NUP may both be manifestations of systemic diseases such as AIDS which would categorize them with periodontal diseases as a manifestation on systemic diseases. Other factors such as cigarette smoking and stress have also been linked with these infections. In the end, the committee decided against placing these conditions with other diseases for the time being and allowed them to remain a separate disease entity until further information was discovered.

One of the dilemmas facing researchers is whether or not NUG and NUP are two separate diseases or simply different presentations of the same disease. Both NUG and NUP are related to a diminished systemic resistance to bacterial infection in the periodontal tissues.

Necrotizing ulcerative gingivitis generally infects persons between the age of 15-25 years of age, particularly students and military recruits during periods of elevated stress and limits itself to the gingiva. NUG was commonly referred to as "trench mouth" during World War I because of how widespread it was among the soldiers in the trenches and was attributed to the stress, filthy living conditions, and lack of oral hygiene.⁶ Clinical signs and symptoms include sudden onset, pain, and fiery red gingiva with spontaneous bleeding, gray pseudomembrane, excessive salivation, fever, and swollen lymph nodes. Feter oris, or fetid breath odor, and necrosis of interdental papillae creating the cratered, "punched-out" papillae are two characteristics uniquely associated with NUG and NUP. The etiology of the disease is believed to be predisposing factors that are present with pathogenic bacteria. These factors include severe stress, gingivitis, smoking, and radical change in eating or sleeping habits. Treatment consists of debridement of the necrotic tissues which can be painful for the patient, along with irrigation and pain control; rigorous home care including daily mechanical plaque removal, and rinsing with Chlorhexidine as well as systemic antibiotics and antifungal therapy if indicated. Nutritional counseling and smoking cessation are also important in the management of this disease. Following treatment, patients should be placed on a more frequent maintenance therapy recall. (See Figure 4)

Necrotizing ulcerative periodontitis differentiates itself when NUG extends to involve the attachment apparatus and bone loss is apparent. This infection is now considered to be a periodontal disease, NUP, instead of gingival. The patient population distinctive of NUP tends to include individuals with systemic conditions such as HIV/AIDS, severe malnutrition, and

immunosuppression. NUP is a massive tissue-destroying process that extends from acute NUG.⁶ The clinical manifestations of NUP include all of the previously discussed signs and symptoms of NUG as well as rapid gingival recession; rapid, irregular bone loss, delayed wound healing, bone sequestra and the spread of infection to adjacent oral mucosa. The etiology of NUP is also thought to be the same or similar to the etiology discussed above with NUG. Treatment modalities are also identical with the exception of the possibility of the removal of bone sequestra. Over 100 different bacteria have been identified with NUG and NUP. Necrotizing periodontal diseases are of a fuso-spirochetal origin with gram-negative rods accounting for 50% of all bacteria including *Prevotella intermedia*, *Fusobacterium* and *Selenomonas*.

Conclusions

Research is ongoing in the field of periodontal disease and our knowledge and understanding continues to change. New technology and developments are sure to be discovered through this research in the future. Periodontology is always changing so as clinicians, it is imperative that we remain current in our knowledge of the diseases as well as the etiologies, pathogenesis, and new treatment modalities as they become available. That being said, this current classification system could find itself changing in the future as we continue to learn more about the disease.¹

The focus of this article is to help clinicians acquaint themselves with the new classifications of periodontal diseases introduced by the Academy of Periodontology in 1999 as well as the disease entities themselves. Although the majority of the cases of periodontal diseases seen in general dental practices today fall under the category of plaque-induced gingivitis and chronic periodontitis, it is important for dentists and dental hygienists to be familiar with the different categories of periodontal diseases in the chance that a patient presents to your office for treatment.

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Fig 4 Necrotizing Ulcerative Periodontitis, Photo courtesy of Dr. Hiram R. Fry, DDS

An Overview of Gingival and Periodontal Diseases

- Today, the theory that is accepted as the current etiological basis for periodontal disease is
 - Calculus theory
 - Non-specific plaque theory
 - Specific plaque theory
 - Host-bacterial interaction theory
- In 1999 the International Workshop for a Classification of Periodontal Diseases and Conditions introduced a new classification system for periodontal diseases. This replaced the previous classification system that had been in use since 1802.
 - Both statements are true.
 - Both are false.
 - The first is true and the second is false.
 - The first is false and the second is true.
- Major changes in the new classification system include all of the following EXCEPT
 - Addition of a section on gingival diseases
 - Replacement of the classification of "Adult Periodontitis" with "Chronic Periodontitis"
 - Placement of NUG and NUP under classification of periodontal diseases as a manifestation of systemic diseases.
 - Removal of a separate category for refractory periodontitis
- The bacteria present in the oral cavity during health are primarily
 - Gram-negative, facultative anaerobic cocci and rods
 - Gram-negative aerobic rods
 - Gram-positive, facultative cocci and rods
 - Gram-positive, aerobic rods
- Which of the following is the most common form of gingival diseases encountered in the dental office today?
 - Necrotizing ulcerative gingivitis
 - Plaque-induced gingivitis
 - Pregnancy gingivitis
 - Gingival diseases modified by malnutrition
- Systemic factors can affect how a person's immune system reacts to the presence of bacterial plaque. Many of these factors in and of themselves are not responsible for causing gingivitis but can intensify it in certain susceptible individuals resulting in the development of the disease.
 - Both statements are true.
 - Both are false.
 - The first is true and the second is false.
 - The first is false and the second is true.
- Pregnancy gingivitis is most common during which trimester of pregnancy due to the increase in hormone levels which can result in an increase of gingival inflammation?
 - First
 - Second
 - Third
 - All of the above
- All of the following medications can cause gingival hyperplasia EXCEPT
 - Cyclosporine
 - Phenytoin
 - Verapamil
 - Nitroglycerine
- Which of the following bacteria is predominant in chronic periodontitis?
 - Porphyromonas gingivalis*
 - Streptococcus mutans*
 - Eikenella corrodens*
 - Staphylococcus aureus*
- All of the following diseases are examples of aggressive periodontitis EXCEPT
 - Prepubertal periodontitis
 - Rapidly progressive periodontitis
 - Localized aggressive periodontitis
 - Chronic periodontitis
- Localized aggressive periodontitis occurs in otherwise healthy children and teenagers and affects which teeth?
 - Primary canines and second molars
 - Permanent incisors and first molars
 - Permanent canines and first premolar
 - Primary incisors and canines
- Generalized aggressive periodontitis is similar to localized aggressive periodontitis in all the following ways EXCEPT
 - Occurs in otherwise healthy children and teenagers
 - Rapid destruction of periodontal attachment over a short period of time
 - Associated with neutrophil chemotaxis disorder
 - Associated with significant amount of clinical inflammation along with heavy plaque and calculus formation
- The periodontal disease that often results in the loss of both primary and secondary dentition is known as
 - Prepubertal periodontitis
 - Rapidly progressive periodontitis
 - Localized aggressive periodontitis
 - Chronic periodontitis
- In addition to all the signs and symptoms of NUG, patients with NUP may also experience
 - Rapid gingival recession
 - Rapid, irregular bone loss
 - Bone sequestra
 - All of the above
- Necrotizing ulcerative gingivitis is limited to the gingiva. When NUG extends to involve the attachment apparatus, it is considered to differentiate into the periodontal disease, NUP.
 - Both statements are true.
 - Both are false.
 - The first is true and the second is false.
 - The first is false and the second is true.

SELF INSTRUCTIONAL REGISTRATION FORM: AN OVERVIEW OF GINGIVAL AND PERIODONTAL DISEASES

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