The Periodontal Disease Classification System of the American Academy of Periodontology — An Update

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Abstract

Until recently, the accepted standard for the classification of periodontal diseases was the one agreed upon at the 1989 World Workshop in Clinical Periodontics. This classification system, however, had its weaknesses. In particular, some criteria for diagnosis were unclear, disease categories overlapped, and patients did not always fit into any one category. Also, too much emphasis was placed on the age of disease onset and rate of progression, which are often difficult to determine. Finally, no classification for diseases limited to the gingiva existed. In 1999, an International Workshop for a Classification of Periodontal Diseases and Conditions was organized by the American Academy of Periodontology to address these concerns and to revise the classification system. The workshop proceedings have been published in the Annals of Periodontology. The major changes to the 1989 proceedings and the rationale for these changes are summarized here. In addition, the potential impact of these changes is discussed.

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Periodontology (AAP) went from 2 main periodontal disease categories to 5 (Table 1). The 1989 periodontal disease classification was a significant improvement over previous classifications. In particular, the effect of systemic disease on periodontal health was recognized and added as a category. Also, more criteria for early-onset diseases were added. However, the 1989 classification had its shortcomings including: lack of a category for strictly gingival diseases; overlap between disease categories; difficulty in fitting certain patients into any of the existing categories; similarity of microbiological and host response features in purportedly different disorders; an emphasis on age of onset that became a problem as patients aged into a new category; and some unclear classification criteria.

A new periodontal disease classification system was recommended by the 1999 International Workshop for a Classification of Periodontal Disease and Conditions² (**Table 2**) and has been accepted by the AAP. This new classification has numerous subcategories; only the major categories will be discussed here. A summary paper of the workshop by Dr. Gary Armitage is available on the AAP Web site at

http:www.perio.org/resources-products/classification.htm. The entire workshop proceedings² can be ordered from the AAP by calling 1-800-282-4867.

Changes to the Periodontal Classification System

Addition of a gingival disease component (Table 2, Section I)

Gingivitis represents a spectrum of diseases whose onset is commonly attributed to the presence of bacteria, but there are other forms of gingivitis that are not primarily plaque-related. Systemic diseases such as diabetes and leukemia can exacerbate plaque-associated gingivitis, as can endocrine changes (puberty, pregnancy), medications (nifedipine, cyclosporin and phenytoin) and malnutrition (vitamin C deficiency). The 1999 classification includes categories for all these forms of dental plaque-induced gingivitis.

Non-plaque induced gingival lesions can result from specific bacterial pathogens such as *Neisseria gonorrhea*, from viral infections and from fungal infections. These gingival diseases are classified differently from plaque-associated gingivitis. Mucocutaneous disorders (e.g., lichen planus,

Table 1 Evolution of the AAP periodontal disease classification system¹

1977	1986	1989
I. Juvenile Periodontitis	I. Juvenile Periodontitis	I. Early-Onset Periodontitis
II. Chronic Marginal Periodontitis	A. PrepubertalB. Localized juvenile periodontitisC. Generalized juvenile periodontitis	A. Prepubertal periodontitis1. Localized2. Generalized
	II. Adult PeriodontitisIII. Necrotizing Ulcerative Gingivo-PeriodontitisIV. Refractory Periodontitis	B. Juvenile periodontitis

pemphigoid), allergic reactions (e.g., restorative materials, toothpastes, gum), trauma (chemical, physical or thermal) as well as disorders of genetic origin such as hereditary gingival fibromatosis can also cause non-plaque-induced gingival lesions.

Replacement of "Adult Periodontitis" with "Chronic Periodontitis" (Table 2, Section II)

The reported prevalence of periodontal disease varies depending on the criteria (depth of pockets or clinical attachment level and number of teeth involved), but it is generally accepted that 8-13% of North Americans have periodontal bone loss.3 Adult periodontitis has traditionally been defined as having its onset after the age of 35 years; approximately 18% of this population has periodontal bone loss.³ However, the age-dependent nature of the adult periodontitis designation was felt to be somewhat arbitrary as similar bone loss patterns can also be seen in adolescents and even in the primary dentition of children. Another difficulty lay in the fact that the age at which a patient presents for treatment does not necessarily reflect the age at which the disease began. Workshop participants concluded that the term adult periodontitis was misleading and should be replaced with the term chronic periodontitis because there is no histopathological uniqueness nor natural determination point as to when disease onset is most likely to occur. "Chronic" periodontitis refers to progression of the disease over time without treatment and does not suggest that the disease is "untreatable."

Chronic periodontitis is characterized as occurring mostly in adults, but it can be seen in younger people. Destruction is consistent with the amount of plaque present and other local factors (i.e., anatomic and other factors that retain plaque next to a tooth such as overhanging restorations, open contacts and palato-radicular grooves); subgingival calculus is also commonly found. In general the disease progresses slowly but there may be bursts of destruction. In addition, the rate of disease progression can be modified by local factors, systemic diseases and such extrinsic factors as smoking.

Chronic periodontitis has been further classified as localized or generalized depending on whether < 30% or > 30% of sites are involved. Severity is based on the amount of clinical

attachment loss (CAL) and is designated as slight (1-2 mm CAL), moderate (3-4 mm CAL) or severe (> 5 mm CAL).

Elimination of "Refractory Periodontitis" as a Separate Entity

Refractory periodontitis refers to continued attachment loss in spite of adequate treatment and proper oral hygiene. Many factors appear related to a lack of response to periodontal therapy, including extent of disease prior to therapy, type of therapy provided (nonsurgical vs. surgical, with or without antibiotics, etc.), tooth type and furcation involvement, species and strains of microflora, degree of host response (particularly immune response), and whether the patient smokes. Given that these factors can affect treatment response, it was felt that refractory periodontitis was likely not a separate entity and the category was discontinued.

The term recurrent periodontitis is used to indicate a return of periodontitis and not a separate disease. Potentially, any patient with a past history of periodontitis can develop recurrent periodontitis if adequate oral hygiene is not maintained. Accumulation of dental plaque due to poor oral hygiene or lack of routine periodontal debridement contribute to the development of recurrent periodontitis.

Replacement of "Early-Onset Periodontitis" with "Aggressive Periodontitis" (Table 2, Section III)

There are forms of periodontal disease that clearly differ from chronic periodontitis. In the 1989 classification, patients were placed into the early-onset category if they exhibited significant attachment loss in the presence of little local factors (plaque and calculus) and were less than 35 years of age. It is true that this disease often occurs in people under 35 years of age, but it may also affect older patients. The workshop participants concluded that the term early-onset periodontitis was too restrictive and recommended it be replaced with "aggressive periodontitis." Diagnosis of aggressive periodontitis is made on clinical, radiographic and historical findings which show rapid attachment loss and bone destruction, and possible familial aggregation of disease. Except for periodontal disease, patients are systemically healthy. Other features that may be present are periodontal tissue destruction that is

Table 2 Abbreviated version of the 1999 classification of periodontal diseases and conditions²

- I. Gingival Diseases
 - A. Dental plaque-induced gingival diseases
 - B. Non-plaque-induced gingival lesions
- II. Chronic Periodontitis

(slight: 1-2 mm CAL; moderate: 3-4 mm CAL;

severe: > 5 mm CAL)

A. Localized

B. Generalized (> 30% of sites are involved)

III. Aggressive Periodontitis

(slight: 1-2 mm CAL; moderate: 3-4 mm CAL;

severe: > 5 mm CAL)

A. Localized

B. Generalized (> 30% of sites are involved)

- IV. Periodontitis as a Manifestation of Systemic Diseases
 - A. Associated with hematological disorders
 - B. Associated with genetic disorders
 - C. Not otherwise specified
- V. Necrotizing Periodontal Diseases
 - A. Necrotizing ulcerative gingivitis
 - B. Necrotizing ulcerative periodontitis
- VI. Abscesses of the Periodontium
 - A. Gingival abscess
 - B. Periodontal abscess
 - C. Pericoronal abscess
- VII. Periodontitis Associated With Endodontic Lesions
 A. Combined periodontic-endodontic lesions
- VIII. Developmental or Acquired Deformities and Conditions
 - A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
 - B. Mucogingival deformities and conditions around teeth
 - Mucogingival deformities and conditions on edentulous ridges
 - D. Occlusal trauma

greater than would be expected given the level of local factors, elevated levels of *Actinobacillus actinomycetemcomitans* or *Porphyromonas gingivalis*, phagocyte abnormalities and increased production of prostaglandin E_2 and interleukin-1 β .

Aggressive periodontitis was also subcategorized into localized and generalized forms to replace localized and generalized juvenile periodontitis. Classification is similar to chronic periodontitis in terms of number of teeth involved and severity of attachment loss.

Further Subclassification of "Periodontitis as a Manifestation of Systemic Diseases" (Table 2, Section IV)

Systemic diseases that affect immune function, inflammatory response and tissue organization can modify the onset and progression all forms of periodontal disease. The 1989 classification was expanded to include subcategories for hematological disorders (acquired neutropenia, leukemias and other), genetic disorders (familial and cyclic neutropenia, Down syndrome, leukocyte adhesion deficiency syndromes,

Papillon-Lefèvre syndrome, Chediak-Higashi syndrome, histiocytosis syndromes, glycogen storage disease, infantile genetic agranulocytosis, Cohen syndrome, Ehlers-Danlos syndrome types IV and VIII, hypophosphatasia and other) and other disorders "not otherwise specified." This category acknowledges that management of the periodontal disease should be carried out in conjunction with management of the systemic disease. It is expected that other diseases will eventually be added as future investigations demonstrate the effect of systemic diseases on periodontal tissue health and periodontal disease progression.

Replacement of "Necrotizing Ulcerative Periodontitis" with "Necrotizing Periodontal Diseases" (Table 2, Section V)

The category includes necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP). Both appear to be related to diminished systemic resistance to bacterial infection and may only differ in terms of tissue, with NUP extending into periodontal attachment.

Addition of Categories for "Periodontal Abscess" and "Periodontic-Endodontic Lesion" (Table 2, Sections VI and VII)

No changes were made to the definition of these diseases; they were simply added to the classification system. The term periodontic-endodontic lesion is not based on the initial etiology of the lesion but simply indicates there is both a periodontic and an endodontic component.

Addition of a Category for "Developmental or Acquired Deformities and Conditions" (Table 2, Section VIII)

This category includes local factors associated with teeth and restorations, mucogingival deformities around teeth and on edentulous ridges as well as occlusal trauma as we have traditionally defined them.

Discussion

Many of the 1999 changes to the classification system were simply semantic ones that allowed for a more continuous organization of conditions rather than a separation of periodontitis from other diseases or disorders also affecting the periodontium. For example, periodontal abscesses, combined periodontic-endodontic problems, mucogingival deformities and occlusal trauma all remain unchanged except that they have been ordered in the classification system. Likewise, NUG and NUP were combined under the category of necrotizing periodontal diseases with no changes to their definitions.

One of the most significant changes included the addition of a detailed section on gingival diseases and lesions. Another important change was the discontinuation of terms related to age of presentation and rate of progression of the diseases. It was felt that these criteria were rather ambiguous since it is often impossible to determine when periodontal disease starts or how fast it progresses if previous dental records are not available. The fact that disease progression can be either slow and constant or episodic, and the finding that similar disease presentations are found at most ages, provided additional evidence for removing these terms. The term adult periodontitis was therefore replaced with chronic periodontitis. The criteria for chronic periodontitis remain similar to those used for adult periodontitis but the age-dependent terminology has been removed. It was acknowledged that chronic periodontitis is most prevalent in adults, but can also occur in adolescents.

In the past, there was considerable overlap between earlyonset periodontal disease and periodontal disease in children and adolescents who had systemic diseases and syndromes associated with periodontal disease progression. Early-onset periodontitis has now been replaced with aggressive periodontitis. Use of this category implies that the patient is systemically healthy but has periodontal disease. All syndromes and systemic diseases which predispose a patient to periodontal disease would be classified under the category of "periodontitis as a manifestation of systemic disease." The continuing difficulty is with patients who have a subclinical systemic disease whose only symptom is the periodontal component. Cases that fall between aggressive periodontitis and systemic diseases are likely to remain a challenge to classify. This category is the most likely to see considerable additions in the future.

Refractory periodontitis is no longer considered a specific disease. Rather, use of this wording refers to any type of destructive periodontal disease that demonstrates additional attachment loss despite therapeutic and patient efforts to stop disease progression. Rather than being a specific type of periodontal disease, it is now a descriptor of any form of periodontal disease.

Conclusion

The 1999 classification system has been approved by the AAP, is now official terminology for that organization, and will be used in accredited graduate periodontal programs and board examinations. The Parameters of Care⁴ approved by the AAP have adopted the new classification and future publications will use it as their standard. Since many of the 1999 workshop participants were from Europe and Asia as well as North America, it is anticipated that the proposed classification will be adopted in most parts of the world. The new classification has not resulted in any changes in insurance codes for the billing of periodontal treatment. ❖

References

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C D A R E S O U R C E C E N T R E

Copies of the Consensus Reports from the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions can be obtained from the CDA Resource Centre, tel.: 1-800-267-6354 or (613) 523-1770, ext. 2223; fax: (613) 523-6574; e-mail: info@cda-adc.ca.

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