CLASSIFICATION OF PERIODONTAL DISEASES

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Need for Classification

- Provides us with a framework to come to a diagnosis.
- To provide a foundation to study the etiology, susceptibility traits, pathogenesis, and treatment of diseases in an organized manner.
- To give clinicians a way to organize the health care needs of their patients.
- Assemble similar disease phenotypes in more homogeneous syndromes.

Clinical Characteristics paradigm (1870-1920)

G.V.Black's Classification

1. constitutional gingivitis: Including mercurial gingivitis and scurvy

2. painful form of gingivitis: NUG

3. simple gingivitis. This was associated with the accumulation of debris that eventually led to inflammation

4. calcic inflammation of the peridental membrane: This was associated with calculus deposition. Usually there was an even or generalized pattern of destruction of alveolar bone. The destruction usually occurred slowly.

5. phagedenic pericementitis: Irregular pattern of destruction and not much dental calculus. Destruction of the alveolar bone can occur slowly or rapidly.

Classical Pathology paradigm (1920–1970)

Gottlieb's Classification

- Schmutz pyorrhoea: accumulation of deposits, leading to inflammation, shallow pockets, resorption of alveolar crest.

- Alveolar atrophy or diffuse atrophy: minimal local factors, deep pockets were seen in later stages with loosening of teeth and eventually leading to tooth loss.

- Paradental pyorrhoea: irregularly distributed pockets varying from shallow to extremely deep.

- Occlusal trauma: alveolar bone resorption & tooth mobility due to increased occlusal overload.

Orban's Classification

Inflammation

I. Gingivitis (little or no pocket formation; can include ulcerative form – Vincent's)

A. Local (calculus, food impaction, irritating restorations, drug action, etc.)

B. Systemic

- pregnancy
- diabetes
- other endocrine dysfunctions
- tuberculosis
- syphilis
- nutritional disturbances
- drug action
- allergy
- hereditary
- idiopathic, etc.

II. Periodontitis

A. Simplex (secondary to gingivitis) – bone loss, pockets, abscesses can form: cases have calculus.

B. Complex (secondary to periodontosis) – etiologic factors similar to periodontitis; cases have little, if any calculus.

Degeneration

I. Periodontosis (as a rule attacks young girls and older men; often caries immunity)

A. Systemic disturbances

1. diabetes
2. endocrine dysfunctions
3. blood dyscrasias
4. nutritional disturbances
5. nervous disorders
6. infectious diseases (acute & chronic)

B. Hereditary

C. Idiopathic

Atrophy

I. Periodontal Atrophy (Recession, no inflammation, no pockets; osteoporosis)

A. Local trauma (e.g., from toothbrush)

B. Presenile

C. Senile

D. Disuse

E. Following inflammation

F. Idiopathic

Hypertrophy

I. Gingival Hypertrophy
   A. Chronic irritation (see inflammation)
   B. Drug action (e.g., Dilantin sodium)
   C. Idiopathic (e.g., gingivoma, elephantiasis, fibromatosis)

Traumatism

I. Periodontal Traumatism
   A. Occlusal trauma

Infection/Host response paradigm (1970 to present)

Page & Schroeder's

Prepubertal Periodontitis:

- During or immediately after the eruption of primary teeth
- Localised & generalized (inflammation and clefting of gingival margin. Genetic predisposition). Neutrophils , monocytes from peripheral blood – defective function.
- All primary teeth but permanent teeth may or may not be affected.
- Localised – only some teeth. Not as rapid as generalised

Juvenile Periodontitis:

- Circumpubertal onset
- Lesions are primarily around permanent first molars and or incisors with usually symmetrical distribution
- Clinically gingiva appears completely normal with minimal local factors
- Genetic predisposition
- Neutrophil and monocyte function defects

Rapidly Progressing Periodontitis:

- Generalized, affecting most of the teeth.
- Age of onset – between puberty and 35 yrs.
- Destruction is rapid which gradually slows down spontaneously.
- In active and inactive disease
- Neutrophil and monocyte function defects

Adult Periodontitis:

- 35 yrs
- Accumulation of plaque is consistent with periodontal destruction.
- Neutrophil and monocyte function defects are not usually present.
- Pattern of bone destruction is highly variable.

Acute Necrotizing Ulcerative Gingivo-Periodontitis:

Acute infective condition characterized by deep craters in the interdental bone, either in localized or throughout the mouth.

Pseudo-membrane is seen on the lesion made up of necrotic tissue & bacteria.

The mouth of the pt is foul smelling.

Malnourished or immunocompromised pts.

AAP - 1989

I. Adult Periodontitis

II. Early Onset Periodontitis

- A. Prepubertal Periodontitis
  - 1. Generalized
  - 2. Localized
- B. Juvenile Periodontitis
  - 1. Generalized
  - 2. Localized
- C. Rapidly Progressive Periodontitis

III. Periodontitis Associated with Systemic Disease

IV. Necrotizing Ulcerative Periodontitis

V. Refractory Periodontitis

Drawbacks of AAP 1989

- Age and rate of progression
- Gingivitis or gingival disease category
- Overlap
- Prepubertal periodontitis
- Rapidly progressive periodontitis

Ranney's Suggestion

European Workshop in Periodontology, 1993

- Adult periodontitis
- Early-onset periodontitis
- Necrotizing periodontitis

AAP - 1999

Classification of Diseases Affecting Periodontium

1. Gingival Diseases
2. Chronic Periodontitis
   - Localized
   - Generalized
3. Aggressive Periodontitis
   - Localized
   - Generalized
4. Periodontitis as a modification of Systemic Diseases
5. Necrotizing Periodontal Disease
   - Necrotizing Ulcerative Gingivitis (NUG)
   - Necrotizing Ulcerative Periodontitis (NUP)
6. Abscesses of the Periodontium
   - Gingival Abscess
   - Periodontal Abscess
   - Pericoronal Abscess
7. Periodontitis associated with Endodontic Lesions
   - Endodontic – Periodontal lesions
   - Periodontal – Endodontic lesions
   - Combined Lesions
8. Developmental or Acquired Deformities & Conditions
   - Localized Tooth Related Factors That Predispose To Plaque Induced Gingival Diseases or Periodontitis
   - Mucogingival Deformities & Conditioning Around Teeth
   - Mucogingival Deformities & Conditions on Edentulous Ridges
   - Occlusal Trauma

Gingival Diseases

A. Dental Plaque Induced Gingival Diseases

I. Gingivitis Associated with Dental Plaque Only

a. Without Local Contributing Factors

b. With Local Contributing Factors

II. Gingival Diseases Modified by Systemic Factors

a. Associated with Endocrine System
   - Puberty Associated Gingivitis
   - Menstrual Cycle Associated Gingivitis
   - Pregnancy Associated
     - Gingivitis
     - Pyogenic Granuloma
   - Diabetes Associated Gingivitis

b. Associated with Blood Dyscrasias

- Leukemia Associated Gingivitis
- Others

III. Gingival Diseases Modified by Medications

a. Drug Influenced Gingival Enlargement

b. Drug Influenced Gingivitis
   - Oral Contraceptives Associated Gingivitis
   - Others

IV. Gingival Diseases Modified by Malnutrition

- Ascorbic Acid Deficiency Gingivitis
- Others

B. Non Plaque Induced Gingival Diseases

I. Gingival Diseases of Bacterial Origin

- Neisseria Gonorrhea
- Treponema Pallidum
- Streptococcal Species
- Others

II. Gingival Diseases of Viral Origin

- Herpes virus Infections
  - Primary herpetic Gingivostomatitis
  - Recurrent Oral Herpes
  - Varicella Zoster
- Others

III. Gingival Diseases of Fungal Origin

- Candida Species Infections
- Linear Gingival Erythema
- Histoplasmosis
- Others

IV. Gingival Diseases of Genetic Origin

- Hereditary Gingival Fibromatosis
- Others

V. Gingival Manifestations of Systemic Conditions

- Mucocutaneous Lesions
  - Lichen Planus
  - Pemphigoid
  - Pemphigus Vulgaris
  - Erythema Multiforme
  - Lupus Erythematous
  - Drug Induced
  - Others
- Allergic Reactions
  - Dental Restorative Materials
    - Mercury
    - Nickel
    - Acrylic
    - Others
  - Reactions attributable to
    - Toothpastes/dentifrices
    - Mouth rinses/mouthwashes
    - Chewing gum additives
    - Foods & additives

VI. Traumatic Lesions

- Chemical Injury
- Physical Injury
- Thermal Injury

VII. Foreign Body Reactions

VIII. Not Otherwise Specified

Highlights of AAP 1999

- Addition of gingival disease component
- Replacement of Early onset with aggressive
- Replacement of Adult with chronic
- Elimination of prepubertal periodontitis
- Elimination of Refractory periodontitis
- Replacement of Necrotizing ulcerative periodontitis with Necrotizing periodontal conditions

- Addition of Periodontal abscesses & Periodontitis associated with endodontic lesions
- Addition of Developmental and acquired deformities & conditions

Essentialistic or nominalistic disease concepts

Table 1. Classification based on the extent of the disease. If teeth are missing, the class description should still reflect the clinical image of the patient. Therefore it was decided for cases with ≤ 14 teeth to omit the class semi-generalized and to change the number of teeth for the generalized class to 8–14

	Permanent / mixed dentition No. of teeth present		**Primary dentition**
	n **≥ 14**	***n*** **≤ 14**	
Incidental	1 tooth	1 tooth	1 tooth
Localized	2–7 teeth	2–7 teeth	2–4 teeth
Semi-generalized	8–13 teeth	–	5–9 teeth
Generalized	≥ 14 teeth	8–14 teeth	≥ 10 teeth

Table 2. Classification based on the severity of disease per tooth. The mean estimated root length based on the literature is approximately 12 mm (21); in the case of incidental disease, the severity category at that particular tooth is mentioned

Minor	bone loss ≤ 1/3 of the root length or attachment loss ≤ 3 mm
Moderate	bone loss > 1/3 and ≤ 1/2 of the root length or attachment loss 4–5 mm
Severe	bone loss > 1/2 of the root length or attachment loss ≥ 6 mm

Table 3. Classification based on age. If in patients classified as adult periodontitis it can be demonstrated on the basis of documentation that they already had moderate or severe periodontitis before the age of 36 years, the disease is classified as early onset periodontitis

Early onset periodontitis	
Prepubertal periodontitis	≤ 12 years
Juvenile periodontitis	13–20 years
Postadolescent periodontitis	21–35 years
Adult periodontitis	≥ 36 years

Table 4. Classification based on clinical characteristics. Periodontitis associated with systemic diseases, i.e. periodontitis in subjects suffering from general diseases, or periodontitis in subjects using medication, which enhance the rate and severity of periodontal breakdown is not identified as a specific class of periodontitis. However, the association with such a condition should be added to the diagnosis.

Necrotizing periodontitis	interdental gingival necrosis, bleeding and pain
Rapidly progressive periodontitis	documented rapid breakdown (at any age), i.e. rapidly progressive periodontitis patients showing a progression of ≥ 1 mm interproximal attachment / bone loss per year at affected sites
Refractory periodontitis	documented, no or minimal pocket depth reduction at single rooted teeth after proper initial therapy and/or further attachment loss despite the proper execution of various treatment modalities

Thank you