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Development and Evolution of Periodontal Disease Classification

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Abstract

Periodontal diseases have been recognized and treated for at least 5000 years. Clinicians have recognized for many years that there are apparent differences in the presentation of periodontal diseases and have attempted to classify these diseases. Systems of classifications of disease have arisen allowing clinicians to develop structures which can be used to identify diseases in relation to aetiology, pathogenesis and treatment. It allows us to organize effective treatment of our patients 'diseases. Once a disease has been diagnosed and classified, the aetiology of the condition and appropriate evidence-based treatment is suggested to the clinician. Common systems of classification also allow effective communication between health care professionals using a common language. Early attempts at classification were made on the basis of the clinical characteristics of the diseases or on theories of their aetiology. These attempts were unsupported by any evidence base. As scientific knowledge expanded, conventional pathology formed the basis of classification. More recently, this has been followed by systems of classification based upon our knowledge of the various periodontal infections and the host response to them Classification of periodontal

diseases has, however, proved problematic. Over much of the last century clinicians and researchers have grappled with the problem and have assembled periodically to review or develop the classification of the various forms of periodontal disease as research has expanded our knowledge of these diseases. This has resulted in frequent revisions and changes. A classification, however, should not be regarded as a permanent structure. It must be adaptable to change and evolve with the development of new knowledge. It is expected that systems of classification will change over time. This review examines the past and present classifications of the periodontal diseases.

Keywords: Periodontal Diseases, Systemic Factors, Hypoplasia.

Introduction

Periodontal disease is defined as the infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss[1].

The concept of classification systems is considered as uninteresting by many, but it provides us with a framework to come to a diagnosis. The intricacy of periodontal diseases can be understood by classifying various diseases. Its goals are[2]

- 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.

Three paradigms that reflected the understanding of the nature of periodontal diseases were noticed during the evolution of periodontal diseases [3]

(1870–1920): Clinical characteristics paradigm From approximately 1870 to 1920, researchers had insufficient information about the etiopathogenesis of periodontal diseases. There was dispute about the nature of periodontal diseases; whether local or systemic factors caused them. Opinions were divided. Many of the advocates for the etiological role of local factors also acknowledged that in some cases both local and systemic factors are responsible. Various researchers depended on case descriptions and personal interpretations of clinical cases to classify periodontal diseases.

C.G. Davis in 1879[8] believed that there were three distinct forms of destructive periodontal disease:

Gingival recession with minimal or no inflammation. This was due to '... feeble vascular action ...' and trauma from tooth brushing or other sources.

Periodontal destruction was secondary to 'lime deposits. 'The gum retires slowly ... and the alveolar border, deprived of nutrition at the point of pressure, is consentaneously absorbed.' Davis believed that calculus exerted mechanical pressure on the gingiva causing the alveolar bone to resorb because of lack of nutrition.

'Riggs' Disease' the hallmark of which was, '... loss of alveolus without loss of gum.' The perceived problem was a 'necrosed alveolus' or death of the periodontal membrane. we get a disease that is initiated and continued without any visible mechanical irritant in many cases and I believe the death of the peridental membrane, depriving the alveolus of nutrition, accounts for the death and disintegration of the bone; or, as is believed by some, among them Dr Waters, of Boston, the alveolus is destroyed by vegetable parasites.

1886 G.V. Black [9] published his thoughts on the classification of periodontal diseases based on their

clinical characteristics and his understanding of their cause into five separate groups.

Constitutional Gingivitis: including mercurial gingivitis, potassium iodide gingivitis and scurvy.

A Painful Form of Gingivitis: Black described a clinical condition that resembled what is now termed necrotizing ulcerative gingivitis (NUG), but he never used the term.

Simple Gingivitis: This was associated with the accumulation of debris that eventually led to 'calcic inflammation of the peridental membrane.

Calcic Inflammation: of the peridental membrane. This was associated with 'salivary' and/or 'serumal' calculus. Usually there was an even or generalized pattern of destruction of alveolar bone. The destruction usually occurred slowly. Black's description best fits the periodontal disease that is now known as chronic periodontitis.

Phagedenic Pericementitis (Phagedenic Ω Spreading Ulcer or Necrosis): This condition shared many features with 'calcic inflammation of the peridental membrane' but there was an irregular pattern of destruction and not much dental calculus. Destruction of the alveolar bone can occur slowly or rapidly. In a later publication Black replaced the term 'phagedenic pericementitis' with 'chronic suppurative pericementitis'[10].

(1920–1970): Classical pathology paradigm During this time a new concept developed that periodontal diseases can be 2 :-inflammatory and non-inflammatory ('degenerative' or 'dystrophic'). This was based on the observation that certain forms of periodontal diseases were due to degenerative changes in the periodontium such as cementopathia. As a result, most of the classification systems in this era included disease categories such as 'dystrophic', 'atrophic', or 'degenerative'. Around 1970, a different paradigm began to dominate thoughts about the nature of periodontal diseases. Also, the observation that a patient with hypophosphatasia who had a premature loss of anterior deciduous teeth also harbored Porphyromonas gingival is in the subgingival flora, suggested that something other than hypoplasia of cementum might have contributed to the periodontal destruction. Classification systems of the period were dominated by the 'Classical Pathology' paradigm which is based on the 'principles of general pathology' as articulated by Orban et al[11].

Inflammation

II. 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.

III. 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

II. 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

Fig. 1: Classification of Periodontal Diseases Following the ''Classical Pathology'' Paradigm (Orban 1942)[11]

(1970–present): Infection/ host response paradigm

After the publication of Robert Koch's postulates (1876), researchers stressed the infectious nature of periodontal diseases. W.D. Miller, in particular, was an early proponent of the infectious nature of periodontal diseases. He stated three factors that were to be taken into consideration in every case of pyorrhea alveolar: (1)

predisposing circumstances, (2) local irritation, and (3) bacteria. Miller also recognized that certain systemic conditions (e.g. diabetes, pregnancy) could modify the course of the disease. The next major discovery in periodontal microbiology was the preliminary demonstration in 1976–1977 of microbial specificity at sites with periodontosis. This finding, coupled with the demonstration in 1977–1979 that neutrophils from patients with juvenile periodontitis (periodontosis) had defective chemotactic and phagocytic activities, marked the beginning of the dominance of the Infection/Host Response paradigm.

Classification systems in the modern era represent a blend of all three paradigms since there is a certain amount of gravity to some of the earliest thoughts about the nature of periodontal diseases. History reveals that people in the past opposed the modification of these entities. They were adamant about accepting a particular classification despite it having many flaws. But in the true sense, they should be periodically modified based on modern thinking and concepts.

The next major landmark in the classification of periodontal diseases emerged from the 1989 World Workshop in Clinical Periodontics where a new classification of periodontitis based on the Infection/ Host Response paradigm was suggested[17] (Fig.2). The classification was a refinement of one that had been proposed by Page & Schroeder in 1982[18] and a similar one that had been adopted by the AAP in 1986 one that had been adopted by the.

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

Fig. 2: World Workshop in Clinical Periodontics (1989) Classification [17]

The concept that the rate of progression might be a useful criterion upon which to base a disease category may be flawed. The rate at which periodontitis progresses are highly variable and depends on such factors as

1. Innate and acquired host susceptibility[19].
2. Composition and quantity of the subgingival flora[20]
3. The nature of genetically determined host–bacterial interactions[21]

European Workshop in Periodontology (1993) Classification

This classification, being simple, was agreed upon by most clinicians and research scientists throughout the world.

Adult Periodontitis: Begins at the 4th decade of life, slow rate of progression of disease.

Early onset Periodontitis: Begins before the 4th decade of life, rapid rate of progression of disease, altered host response is seen.

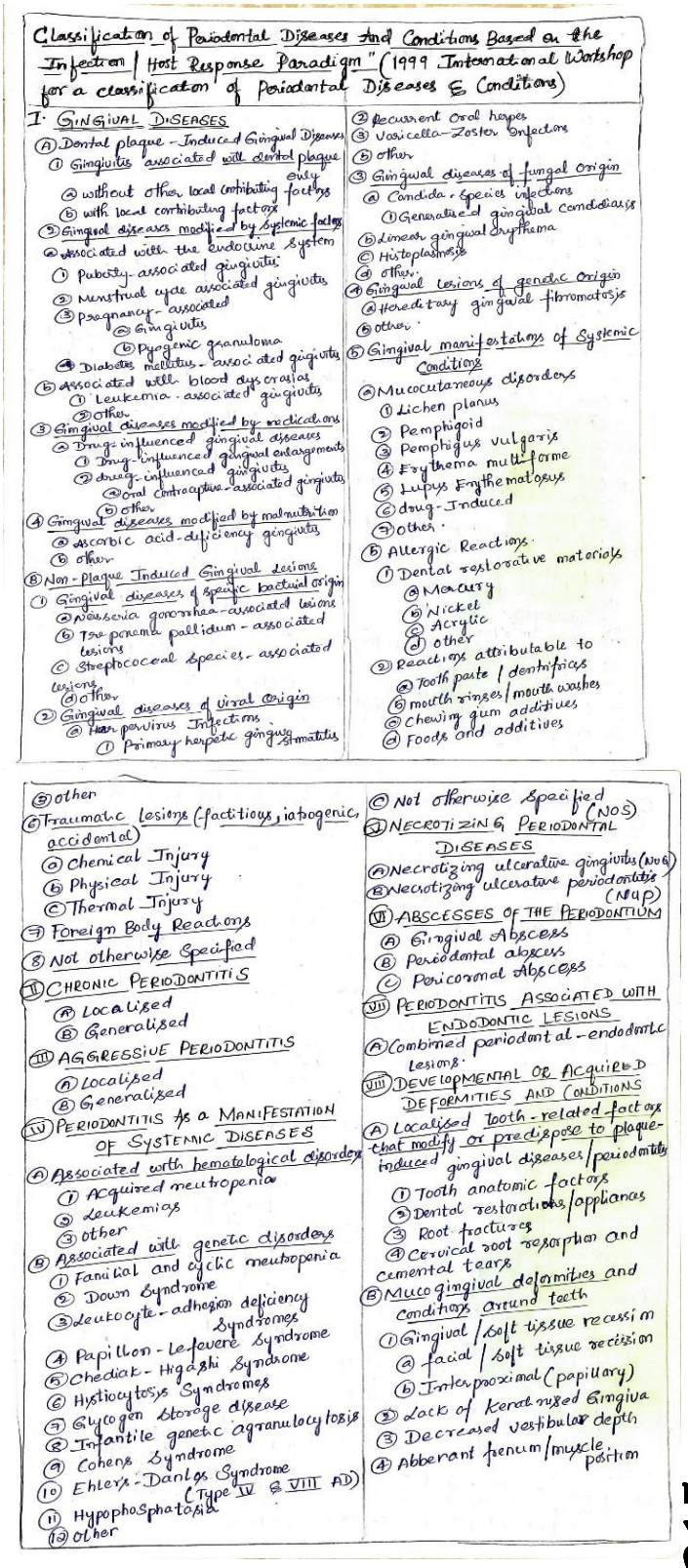
Necrotizing Periodontitis: Tissue necrosis with clinical attachment and bone loss is seen.

Elaboration of the broad spectrum of periodontal diseases encountered in clinical practice was absent in the 1993 European classification. Thus during the 1996 World Workshop in Periodontics, the need for a revised classification system for periodontal diseases was stressed. In 1997, the American Academy of Periodontology responded to this need and formed a committee to plan and organize an international workshop to revise the classification system for periodontal diseases. The International Workshop for a Classification of Periodontal Diseases and Conditions was held and a new classification was agreed upon in 1999.

1999 Classification of Periodontal Diseases and Conditions

Problems, inconsistencies, and deficiencies associated with the 1989 classification led many clinicians and investigators to call for a revision of the currently used system. This resulted in a 1999 international workshop on the classification of periodontal diseases and the classification has been mentioned in figure 3{a, b, c} [22]. One of the goals of this workshop was to correct the problems associated with the 1989 system. There were six major problems with the 1989 classification that needed to be addressed:

- It did not include a gingivitis or gingival disease category.
  - The periodontitis categories had non validated age dependent criteria.
  - There was extensive crossover in rates of progression of the different categories of peri- heterogeneous category.
  - There was extensive overlap in the clinical characteristics of the different categories of periodontitis.
  - 'Refractory Periodontitis' was a heterogeneous category.
  - 'Prepubertal Periodontitis' was a heterogeneous category



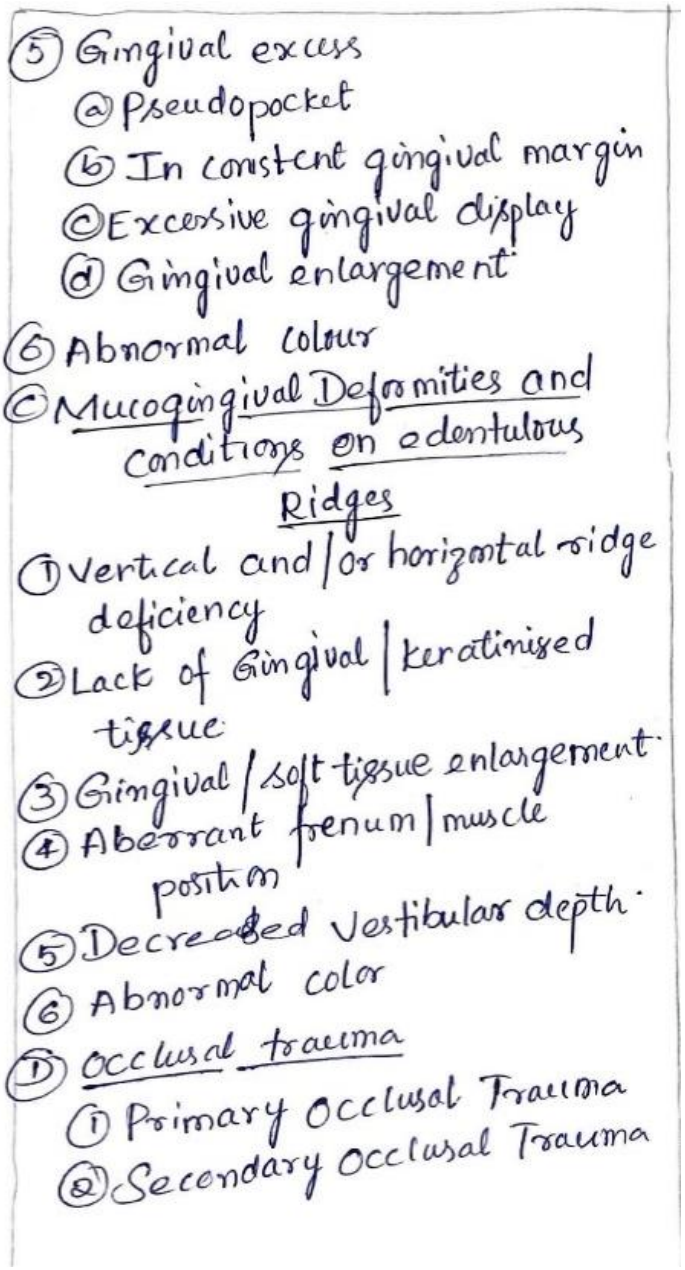


Fig. 3[a, b, c]: Classification of Periodontal Diseases and Conditions Based on the ‘‘Infection/Host Response Paradigm’’ (1999 International Workshop for a Classification of Periodontal Diseases and Conditions)[22]

What features of the 1999 classification prompted a new classification?

- It did not serve as a therapeutic guide (unlike Angle's Classification of malocclusion)
- Categorizing aggressive & chronic periodontitis – cumbersome & confusing (required assessment of rate of progression spread over multiple visits & chronic periodontitis exacerbation phase may also show rapid progression)
- Current evidence does not support the distinction between chronic and aggressive periodontitis as separate clinical entities (similar microbiology, pathogenesis, and histopathology).
- Confusion in diagnosing a case of plaque-induced gingival inflammation on a reduced but healthy periodontium – periodontitis or gingivitis?
- Categories of gingival disease modified by medication & diabetes mellitus exist but no such periodontitis class exists.
- No mention of peri-implant diseases

Gingival recession was placed under multiple categories complicating the diagnosis

- treated periodontitis case with recession
- **Further CAL loss** – present- periodontitis; Absent – gingivitis
- Toothbrush trauma induced - non-plaque-induced traumatic lesion
- Due to anatomical variation-mucogingival deformities and conditions
- No proper acknowledgement of risk factors (diabetes &smoking).
- Terms localized and generalized were introduced with arbitrary cut-off 30%, & gives ambiguous results. InLocalised Aggressive Periodontitis, if all the incisors & 1st molars are involved (12 teeth), 12/32= 37.5%- but>30% must fall into generalised)

Classification of Periodontal &Peri-Implant Diseases and Conditions 2017

However, challenges in differentiating the aggressive periodontitis from chronic periodontitis and emergence of new scientific evidences such as peri-implant health and diseases were the major rationale for the new

classification workshop of 2017. The American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) cosponsored a workshop held in Chicago on November 9 to 11, 2017. It included periodontal and dental implant academicians from all over the world. Four consensus reports and 19 review papers related to periodontology and implant dentistry were evaluated by a committee from the AAP and EFP. The experts were assigned with updating the 1999 classification of periodontal diseases and conditions and developing a new classification for peri-implant diseases and conditions. The establishment of periodontal clinical case definitions and the provision of diagnostic criteria for periodontal and peri-implant diseases were prioritized. This updated version was introduced and named as ‘‘New classification scheme for periodontal and peri-implant diseases and conditions: Introduction and key changes from the 1999 classification”

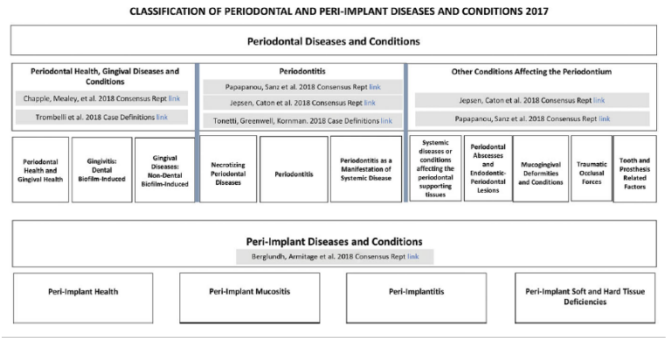


Fig. 4: Classification of Periodontal &Peri-implant diseases and conditions 2017

The workshop addressed unresolved issues with the previous classification by identifying the difference between presence of gingival inflammation at one or more sites and the definition of a gingivitis case. It agreed that bleeding on probing should be the primary parameter to set thresholds for gingivitis[23,24]. The workshop also characterized periodontal health and gingival inflammation in a reduced periodontium after completion of successful treatment of a patient with periodontitis. Specific definitions were agreed to with regard to cases of gingival health or inflammation after completion of periodontitis treatment based on bleeding on probing and depth of the residual sulcus/pocket. This distinction was made to emphasize the need for a more comprehensive maintenance and surveillance of the successfully treated patient with periodontitis. Itwas accepted that a patient with gingivitis can revert to a state of health, but a periodontitis patient remains a periodontitis patient for life, even following successful therapy, and requires life-long supportive care to prevent recurrence of disease[25].The workshop also reorganized the broad spectrum of non-plaque induced gingival diseases and conditions based on primary etiology.

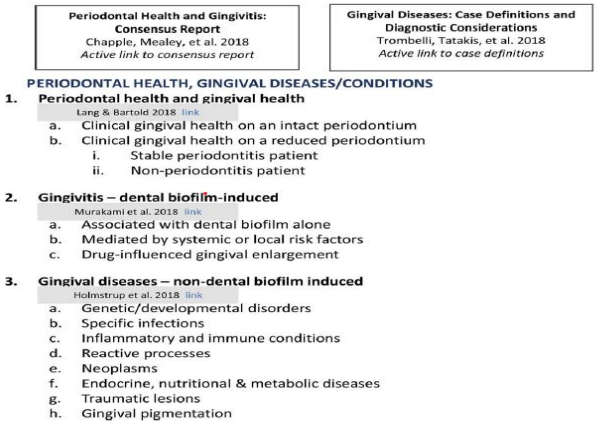


Fig. 5: Periodontal Health, Gingival diseases and Conditions

The 1993 European Workshop determined that the classification should be simplified and proposed grouping of periodontitis into two major headings: adult and early onset periodontitis[26] Periodontitis was reclassified as chronic, aggressive (localized and generalized), necrotizing and as a manifestation of systemic disease. Since the 1999 workshop, substantial new information has emerged from population studies, basic science investigations, and the evidence from prospective studies evaluating environmental and

systemic risk factors [27]. The analysis of this evidence has prompted the 2017 workshop to develop a new classification framework for periodontitis

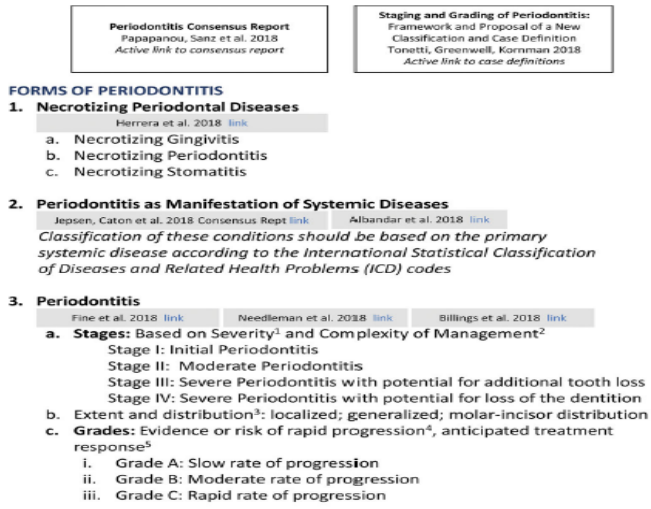


Fig. 6: Forms of Periodontitis

Staging is largely dependent upon the severity of disease at presentation as well as on the complexity of disease management, while grading provides supplemental information about biological features of the disease, including a history based analysis of the rate of disease progression, assessment of the risk for further progression, anticipated poor outcomes of treatment, and assessment of the risk that the disease

or its treatment may negatively affect the general health of the patient[27] Staging involves four categories (stages 1 through 4) and is determined after considering several variables including clinical attachment loss, amount and percentage of bone loss, probing depth, presence and extent of angular bony defects and furcation involvement, tooth mobility, and tooth loss due to periodontitis. Grading includes three

levels (grade A – low risk, grade B – moderate risk, grade C – high risk for progression) and encompasses, in addition to aspects related to periodontitis progression, general health status, and other exposures such as smoking or level of metabolic control in diabetes. Thus, grading allows the clinician to incorporate individual patient factors into the diagnosis, which are crucial to comprehensive case management. For a complete description of the new classification scheme for periodontitis, the reader is directed to the consensus report on periodontitis[27]

Systemic Diseases Associated With Loss of Periodontal Supporting Tissues [28, 29]

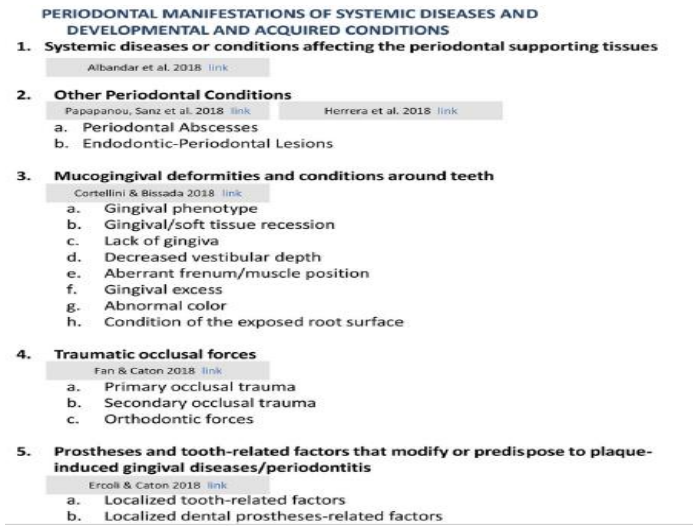


Fig. 7: Other condition affecting the periodontium

The new classification of periodontal diseases and conditions also includes systemic diseases and conditions that affect the periodontal supporting tissues. It is recognized that there are rare systemic disorders, such as Papillon Lefèvre Syndrome, that generally result in the early presentation of severe periodontitis. Such conditions are grouped as “Periodontitis as a Manifestation of Systemic Disease”, and classification should be based on the primary systemic disease [28]. Other systemic conditions, such as neoplastic diseases, may affect the periodontal apparatus independent of dental plaque biofilm induced periodontitis, and such clinical findings should also be classified based on the primary systemic disease and be grouped as “Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues”. There are, however, common

systemic diseases, such as uncontrolled diabetes mellitus, with variable effects that modify the course of periodontitis. These appear to be part of the multifactorial nature of complex diseases such as periodontitis and are included in the new clinical classification of periodontitis as a descriptor in the staging and grading process[28,29]. Although common modifiers of periodontitis may substantially alter disease occurrence, severity, and response to treatment, current evidence does not support a unique pathophysiology in patients with diabetes and periodontitis[29].

A New Classification for Peri-Implant Diseases and Conditions [30]

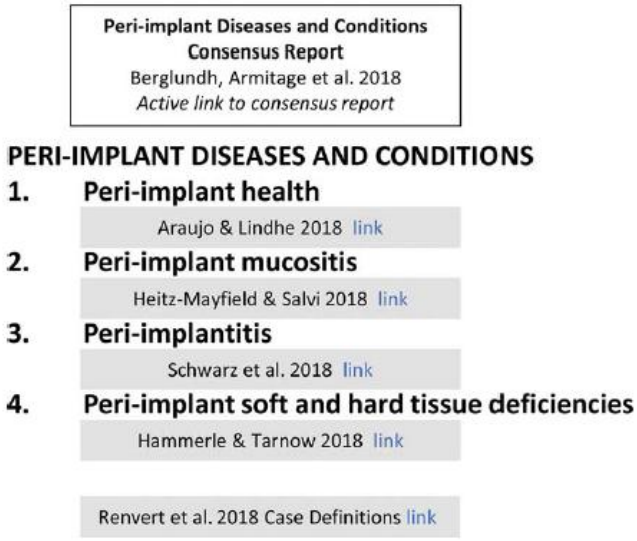


Fig. 8: Peri Implant Diseases and Conditions

A new classification for peri-implant health, 27 peri-implant mucositis[31] and peri-implantitis[32] was developed by the workshop. An effort was made to review all aspects of peri-implant health, diseases, and relevant aspects of implant site conditions and deformities to achieve a consensus for this classification that could be accepted worldwide. Case definitions were developed for use by clinicians for individual case management and also for population studies [31,32].

Other salient features of the new classification were as follow:

Introduction of the term ‘‘gingival pigmentation”.

Identifying smoking and diabetes as the major potential risk factors that can alter the staging of periodontal disease.

Recognition of ‘‘Periodontitis as a manifestation of systemic disease” such as Papillon Life` vre Syndrome.

Systemic conditions affecting the periodontium when not related to dental plaque will be considered as ‘‘Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues”.

Management protocol of gingival recession based on the Inter proximal attachment loss.

The term periodontal phenotype replaced the periodontal biotype and supracrestal attachment is the new term replacing the biological width.

Introduction of the term traumatic occlusal force.

Conclusion

In the past 130years classification systems for periodontal diseases have evolved based on the understanding of the nature of these diseases at the time the classifications were proposed. One consistent feature of the development of classification systems is the guaranteed controversy surrounding any suggested revisions to the previously accepted system of nomenclature Although classification systems for periodontal diseases currently in use are firmly based on and dominated by the Infection/ Host Response paradigm, some features of the older paradigms are still valid and have been retained. Since it is probable that essentially all dentists and periodontists in the world are convinced that most periodontal diseases are infections, it is unlikely that the Infection/Host Response paradigm will be replaced in the near future Concluding, that in the common pursuit towards building a better periodontal disease classification “Coming Together is a

Beginning, keeping together is progress and working together is success"…

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