Research Paper

Critical review of 2017 classification of periodontal and peri-implant diseases and conditions .

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Abstract - *This new classification (2017) of periodontal and peri-implant diseases will have a major impact on clinical practice with respect to periodontal and implant specialties. One of the major changes was the removal of the Aggressive and Chronic Periodontitis terms and replaced by a single category "Periodontitis". Introduction of staging and grading, similar to being used in oncology for many years is bound to facilitate multidimensional periodontal diagnostic classification. This enables the clinician to give individualized diagnosis and tailor made treatment plans for every patient. However, severity and extent of disease is based on the measurable extent of destroyed and damaged tissue. Furthermore, complexity is determined by assessing factors that may influence disease control and managing long term function and aesthetics.*
Key words- *Classification, Periodontitis, Severity, Complexity, Staging, Grading, Peri-implant health, peri-implantitis.*

I.   Introduction

Classification is the systematic collection of data or knowledge and its arrangement in sequential manner in order to facilitate its understanding or knowledge.It is essential for identification of the etiology and understanding of the pathology. Diagnosis, determining prognosis and treatment planning facilitates communication among the clinician, researchers, educators, students, epidemiologist and public health workers.

Salient features and important changes in the new 2017 Classification of Periodontal and peri-implant diseases and conditions (when compared to the 1999 AAP Classification)

1. Recategorization of types of Periodontitis. Chronic and aggressive periodontitis are grouped under a single category – Periodontitis.
2. Three forms of periodontitis have been defined – Periodontitis, necrotizing periodontitis and periodontitis as a manifestation of systemic conditions.
3. Development of staging and grading system for periodontitis.
4. Staging is the severity of periodontitis and complexity of its management.
5. Grading is the estimation of the rate and likelihood of progression of periodontitis.
6. Inclusion of classification for peri-implant conditions and diseases in classification of periodontal diseases.
7. Hard and soft tissue implant site deficiencies also included.
8. Periodontal health and gingival inflammation in a reduced periodontium after completion of successful treatment of a patient with periodontitis have been characterized.
9. This classification accepts that gingivitis patients can revert to a state of periodontal health but periodontitis patients remain periodontitis for life.

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10. Systemic diseases like diabetes mellitus acts as modifier of periodontitis. They can alter the disease occurrence and severity.
11. Periodontal biotype is replaced by periodontal phenotype.
12. Biologic width replaced by supracrestal attached tissues.

2017 Classification

The World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions proposed the new classification system. The workshop was co-sponsored by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) and included expert participants from all over the world. Planning for the conference, which was held in Chicago on November 9 to 11, 2017, began in early 2015.[1]The new classification was formulated under 4 categories - periodontal health & gingival diseases, Periodontitis, other conditions affecting the periodontium and Peri-implant health and diseases.The classification under these categories was evidence based, on the basis of meta analyses and population studies from all over the world.

Periodontal Health, Gingival Diseases and Conditions			Periodontitis		
Periodontal Health and Gingival Health	Gingivitis: Dental Biofilm-Induced	Gingival Diseases: Non-Dental Biofilm-Induced	Necrotizing Periodontal Diseases	Periodontitis	Periodontitis as a Manifestation of Systemic Disease

Other Conditions Affecting the Periodontium				
Systemic diseases or conditions affecting the periodontal supporting tissues	Periodontal Abscesses and Endodontic-Periodontal Lesions	Mucogingival Deformities and Conditions	Traumatic Occlusal Forces	Tooth and Prosthesis Related Factors

Periimplant health and Diseases			
Peri-implant health	Peri-implant Mucositis	Peri-implantitis	Periimplant hard & soft tissue deficiency

Periodontal Health, Gingival Diseases And Conditions

Periodontal health is classified as –
A. Clinical health on an intact periodontium
B. Clinical gingival health on a reduced periodontium
(i) Stable periodontitis patient
(ii) Non-periodontitis patient

Clinical gingival health on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss. Physiological bone levels range from 1.0 to 3.0 mm apical to the cemento-enamel junction.

There is a biological level of immune surveillance, manifesting as a predominantly neutrophilic infiltrate that is consistent with clinical gingival health.

Intact periodontium	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets)[a]	≤3 mm	≤3 mm
Bleeding on probing[a]	<10%	Yes (≥ 10%)
Radiological bone loss	No	No

Clinical gingival health following treatment of gingivitis on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss.
Clinical gingival health following successful treatment of periodontitis is characterized by an absence of bleeding on probing, erythema, edema, and patient symptoms in the presence of reduced clinical attachment and bone levels.
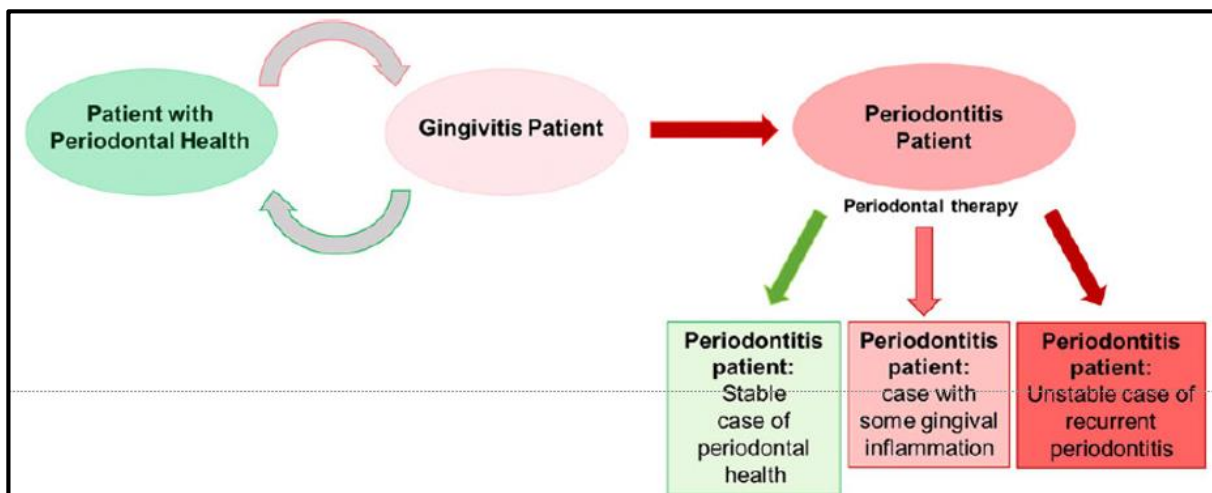
Stable periodontitis patient - Periodontal stability is characterized by successful treatment through control of local and systemic risk factors, resulting in minimal (< 10% of sites) BOP, no probing depths of 4 mm or greater that bleed on probing, optimal improvement in other clinical parameters and lack of progressive periodontal destruction.

Gingivitis
It is of two types – Dental plaque biofilm induced gingivitis and Non-dental plaque biofilm induced gingivitis.
Dental plaque biofilm induced gingivitis is an inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune-inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone). Such inflammation remains confined to the gingiva and does not extend beyond the mucogingival junction and is reversible by reducing levels of dental plaque at and apical to the gingival margin.
Current epidemiological data on the prevalence of gingivitis suffer from the lack of a universally adopted case definition and vary as widely as 6% to 94%, due to the use of indices that measure gingival inflammation at individual sites rather than considering the patient's mouth as a whole. Therefore, mild localized clinical inflammation is reported to affect almost 95% of the population, a figure that would incorrectly suggest gingivitis as being a variation of "normality" and thus consistent with the spectrum of "clinical health" rather than being a disease. – Increased prevalence.
An increasingly extensive manifestation of disease employed in a case definition, causes expression of a lowered prevalence.

Criteria for epidemiological purposes

Condition/ Disease	BOP	Probing Depth
Gingival health in stable & reduced periodontium	< 10% bleeding sites	≤ 3mm
Gingivitis on an intact periodontium	> 10% bleeding sites	≤ 3mm
Gingivitis on a reduced periodontium without h/o periodontitis	> 10% bleeding sites	≤ 3mm
Localized Gingivitis	10% - 30% bleeding sites	
Generalized Gingivitis	>30% bleeding sites	

Periodontitis

In 2015, AAP task force on 1999 classification, suggested that there was difficulty in applying the major and minor criteria for diagnosis of aggressive periodontitis in daily clinical practice.

They also suggested that there is a considerable overlap in the diagnostic criteria which acts as a barrier for application of the classification system.

The workshop suggested that rates of disease progression should not be used to exclude patients from receiving the diagnosis of chronic periodontitis.

There has been a difficulty in correctly differentiating between aggressive and chronic periodontitis clinically.

These difficulties formed the rationale for the new classification of periodontitis.

Necrotizing periodontitis is considered a separate entitiy, as it has a distinct pathophysiology characterized by bacterial invasion and ulceration of epithelium, rapid destruction of soft tissue, hard and soft tissue defects, & rapid resolution to specific antimicrobial regimen.

Additional elements in classification of periodontitis:

1.   Severity - The degree of periodontal breakdown present at diagnosis has long been used as the key descriptor of the individual case of periodontitis. Also included in the 1999 classification as slight, moderate and severe based on CAL.

2.   Complexity of Management - Factors such as probing depths, type of bone loss, furcation status, tooth mobility, missing teeth, bite collapse, and residual ridge defect size increase treatment complexity and need to be considered and should ultimately influence diagnostic classification.

3.   Extent - The number and the distribution of teeth with detectable periodontal breakdown, represented in 1999 Classification as localized and generalized and has now been replaced as – localized, generalized, molar-incisor relationship.

4.   Rate of Progression - One of the most important aspects for a classification system is to properly account for variability in the rate of progression of periodontitis. The major concern regarding this criterion is how to assess the rate of progression at initial examination in the absence of direct evidence (e.g. an older diagnostic quality radiograph allowing comparison of marginal bone loss over time).

5.   Risk Factors - Improved knowledge of how risk factors affect periodontitis (higher severity and extent at an earlier age) and treatment response indicate that risk factors should be considered in the classification of periodontitis.

Severity

STAGE I PERIODONTITS :

Patients with stage I periodontitis have developed periodontitis in response to persistence of gingival inflammation and biofilm dysbiosis.

Diagnosis at this stage enables early intervention and monitoring, more cost effective treatment options - mechanical plaque removal, pharmacological agents, oral hygiene aids.

Diagnosis of Stage I periodontitis at an early age, may indicate a heightened susceptibility to disease onset.

STAGE II PERIODONTITIS :

Stage II represents established periodontitis in which a carefully performed clinical periodontal examination identifies the characteristic damages that periodontitis has caused to tooth support.

Treatment plan - standard treatment principles involving regular personal and professional bacterial removal and monitoring of the condition.

STAGE III PERIODONTITIS:
Significant damage to the attachment apparatus and, in the absence of advanced treatment, tooth loss may occur.
Treatment – bacterial removal & surgical periodontal therapy.
Even though there may be tooth loss, or possibility for tooth loss masticatory function is preserved, and treatment of periodontitis does not require complex rehabilitation of function.
*STAGE IV PERIODONTITIS :*
Periodontitis causes considerable damage to the periodontal support and may cause significant tooth loss, and this translates to loss of masticatory function.
Without proper control of the periodontitis and adequate rehabilitation, the dentition is at risk of being lost. Case management may require stabilization/ restoration of masticatory function along with periodontal treatment.
Complexity score is based on the local treatment complexity assuming the wish/need to eliminate local factors and takes into account factors like presence of vertical defects, furcation involvement, tooth hypermobility, drifting and/or flaring of teeth, tooth loss, ridge deficiency and loss of masticatory function. Besides the local complexity, it is recognized that individual case management may be complicated by medical factors or comorbidities.
CAL is the initial determinant of severity. With presence of additional factors from complexity dimension, the staging can be changed.
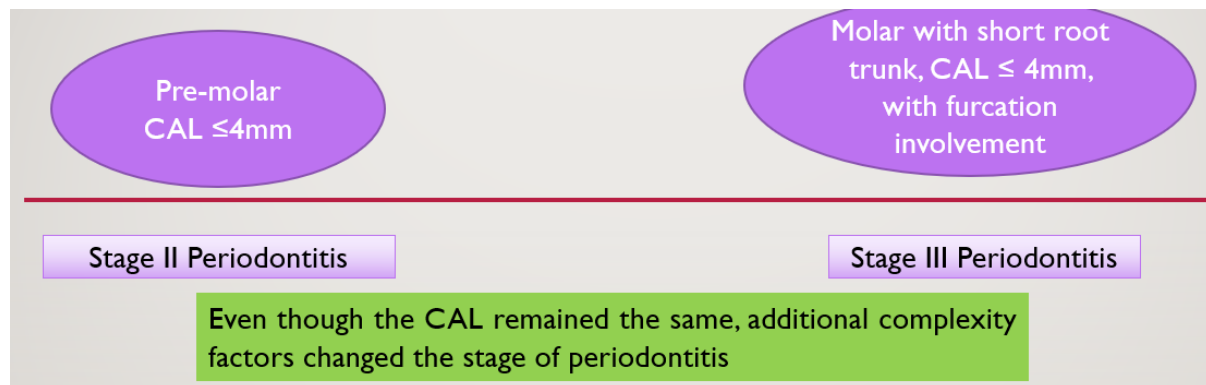
If a stage shifting complexity factor(s) were eliminated by treatment, the stage should not retrogress to a lower stage since the original stage complexity factor should always be considered in maintenance phase management. EXCEPT - Successful periodontal regeneration that may, through improvement of tooth support, effectively improve CAL and RBL of the specific tooth.

Grading
Periodontitis may progress with different rates in individuals, may respond less predictably to treatment in some patients, and may or may not influence general health or systemic disease. Earlier grade of periodontitis progression was incorporated into the classification system by defining specific forms of periodontitis with higher rates of progression. Needleman et al(2018)[2]in a review indicated that there is no evidence to suggest that such forms of periodontitis have a unique pathophysiology, rather the complex interplay of risk factors in a multifactorial disease model may explain the phenotypes of periodontitis in exposed patients. Thus, a biological grade (risk or actual evidence of progression) of periodontitis was incorporated.
Periodontitis progression can be evaluated –
Directly – by previous radiographic records
Indirectly – RBL as % of root length/ age
The objective of grading is to use whatever information is available to determine the likelihood of the case progressing at a greater rate than is typical for the majority of the population or responding less predictably to standard therapy.
Systemic diseases & conditions affecting periodontitis
Diabetes - There are no characteristic phenotypic features that are unique to periodontitis in patients with diabetes mellitus. Thus, diabetes-associated periodontitis is not a distinct disease. But, diabetes is an important modifying factor of periodontitis and hence has been included as a grading factor in classification of periodontitis.

Obesity -   Chaffe et al (2010)[3]&Suvan et al (2015)[4] performed meta-analysis that indicated statistically significant positive association between obesity and periodontitis, but longitudinal studies are lacking in this respect.

Osteoporosis – Penoni et al (2017)[5] in a systematic review concluded that postmenopausal women with osteoporosis or osteopenia exhibit a modest but statistically significant greater loss of periodontal attachment compared with women with normal bone mineral density.

Rheumatoid arthiritis – Fuggle et al (2016)[6] in a meta-analysis found a statistically significant but weak positive association between rheumatoid arthritis and periodontitis, which needs to be confirmed with longitudinal studies.

Smoking - smoking has a major adverse effect on the periodontal supporting tissues, increasing the risk of periodontitis. There are no unique periodontal phenotypic features of periodontitis in smokers, thus, smoking-associated periodontitis is not a distinct disease. But, tobacco smoking is an important modifying factor of periodontitis, hence, has been included in the classification.

Mucogingival conditions around the natural dentition

It has been suggested to use the periodontal phenotype to describe the combination of gingival phenotype (three-dimensional gingival volume) and the thickness of the buccal bone plate (bone morphotype).

Biotype is described as group of organs having the same specific genotype (genetic expression) and phenotype is the appearance of an organ based on a multifactorial combination of genetic traits and environmental factors (its expression includes the biotype).

Chambarone et al (2016)[7] in a meta analysis stated that thin phenotype is at an increased risk of gingival recession.

Recession - Recession is defined as an apical shift of the gingival margin caused by different conditions/pathologies.   It   is   associated   with   clinical   attachment   loss.
The most commonly used classification system for gingival recession is Millers classification (1985).[8]

Shortcomings of miller's classification

The reference point for classification is MGJ, difficulty in identifying the MGJ creates difficulties in the classification between Class I and II. There is no mention of presence of keratinized tissue. Amount and pattern of bone loss have not been described for Class III and IV recessions, and does not exactly specify the level of interdental papilla and amount of loss, hence, a clear picture of severity of recession is hard to project. Cases, which have interproximal bone loss and the marginal recession that does not extend to MGJ cannot be classified either in Class I because of interproximal bone or in Class III because the gingival margin does   not   extend   to   MGJ.
For Class III and IV recessions, it is difficult to classify a tooth, where adjacent tooth/ teeth are missing , as there is   a   lack   of   reference   point   (gingival   margin)   for   recession.
Recession of interdental papilla alone cannot be classified according to the Miller's classification. It is difficult to classify recession on the palatal surface due to lack of MGJ. Miller stated that 100% coverage can be anticipated in Class I and II recessions, partial root coverage in Class III, and no root coverage in Class IV. Pini‑ Prato (2011) stated that anticipation of 100% root coverage does not mean that it will occur. Root coverage % ranging from 9% to 90% has been reported by different authors in Class I and II recessions using different techniques. Recession coverage depends upon various prognostic factors.

Use of classification system by Cairo et al (2011)[10] was proposed –
Recession Type 1 (RT1): Gingival recession with no loss of interproximal attachment. Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth.
• Recession Type 2 (RT2): Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal Sulcus/pocket) is less than or equal to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).
• Recession Type 3 (RT3): Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is higher than the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).

Traumatic occlusal forces and occlusal trauma

Traumatic occlusal forces are the excessive forces to denote that the forces exceed the adaptive capacity of the individual person or site.

Occlusal trauma is a lesion in the periodontal ligament, cementum and adjacent bone caused by traumatic occlusal forces. It is a histologic term; however, a clinical diagnosis of occlusal trauma may be made in the presence of one or more of the following: progressive tooth mobility, adaptive tooth mobility (fremitus), radiographically widened periodontal ligament space, tooth migration, discomfort/pain on chewing, and root resorption.

Orthodontic forces have been added to the category of traumatic occlusal forces. Ericsson et al (1977)[11] performed an animal study and suggested that suggests that certain orthodontic forces can adversely affect the periodontium and result in root resorption, pulpal disorders, gingival recession and alveolar bone loss.

The term biological width has been replaced by the term Supracrestal attachment tissues.

Biological width is a clinical term whereas supracrestal attachment tissues, is a histological term used to describe tissues that are histologically composed of the junctional epithelium and supracrestal connective tissue attachment.

Peri-implant health

The diagnosis of peri-implant health requires:

Visual inspection demonstrating the absence of peri implant signs of inflammation: pink as opposed to red, no swelling as opposed to swollen tissues, firm as opposed to soft tissue consistency. Lack of bleeding on probing.

Probing pocket depths could differ depending on the height of the soft tissue at the implant location. An increase in probing depth over time, however, conflicts with peri implant health.

Absence of further bone loss following initial healing, which should not be ≥2 mm.

Peri – implant diseases

Various meta analyses and systematic reviews were considered to provide a criteria for peri-implant mucositis and peri-implantitis.

Wahlstrorm et al (2010)[12] described periimplantitis as Bone level change > 2 mm after first year in function + BOP and/or SUP + PD ≥4 mm and mucositis - BOP + PD < 4 mm+ no bone loss after first year on function. Daubert et al (2015)[13] described implantitis - Bone level change > 2 mm after remodeling + BOP and or SUP + PD ≥4 mm and mucositis - BOP and/or gingival inflammation + no bone level change after remodeling. Tenenbaum et al (2017)[14] described implantitis - Bone level change > 4.5 mm from platform + BOP + PD ≥5 mm and mucositis - BOP + no bone level change from platform.

Peri-implant mucositis

Visual inspection demonstrating the presence of periimplantsigns of inflammation: red as opposed to pink, swollen tissues as opposed to no swelling, soft as oppose to firm tissue consistency.

Presence of profuse (line or drop) bleeding and/or suppuration on probing.

An increase in probing depths compared to baseline.

Absence of bone loss beyond crestal bone level changes resulting from the intialremodeling.

Peri-implantitis

Evidence of visual inflammatory changes in the periimplant soft tissues combined with bleeding on probing and/or suppuration.

Increasing probing pocket depths as compared to measurement obtained at placement of the supra-structure.

Progressive bone loss in relation to the radiographic bone level assessment at 1 year following the delivery of the implant-supported prosthetics reconstruction.

In the absence of initial radiographs and probing depths, radiographic evidence of bone level ≥3 mm and/or probing depths ≥6 mm in conjunction with profuse bleeding represents peri-implantitis.

Guidelines for epidemiological studies

Epidemiological studies should ideally include previous examinations performed after the first year of loading. In the absence of previous radiographic examinations, bone levels ≥3 mm apical of the most coronal portion of the intra-osseous part of the implant together with bleeding on probing are consistent with the diagnosis of peri-implantitis. The criteria for diagnosis of peri-implant health and mucositis remain the same. Disadvantage of 2017 classification is that it is very extensive and application in daily clinical practise is difficult, especially for the general dentist.

The extensivity also limits its use for epidemiological purposes.

II.   Conclusion:

The World Workshop Classification of Periodontal Diseases and Conditions provides a contemporary and future proof system for classifying periodontal status of undiagnosed patients.

The major novelty is the introduction of staging and grading for periodontitis and the deletion of the term aggressive periodontitis.

The staging and grading system is designed primarily to capture and distinguish a patient's history of periodontal tissue destruction and patient's historical rate of disease progression.

The classification although in accordance with the clinical variations of the periodontium, it is an entity that requires constant evolution and updating.

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