ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 4, Issue – 2, March - 2021, Page No. : 218 - 232

Historical and modern classification systems for periodontal diseases

[1]Gowhar Nazir, Private Practice, Khrew, India

Corresponding Author: Gowhar Nazir, Private Practice, Khrew, India

Citation of this Article: Gowhar Nazir, "Historical and modern classification systems for periodontal diseases", IJDSIR- March - 2021, Vol. – 4, Issue - 2, P. No. 218 – 232.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

Periodontal diseases comprise a number of inflammatory conditions that affect the supporting tissues of the teeth. The diagnosis of these common diseases is an important component of oral health care. Over the past decades a number of classification systems for periodontal diseases have been developed to properly diagnose and treat patients and to investigate etiology, pathogenesis and natural history of the diseases and conditions. The 1999 classification of periodontal diseases and conditions was widely used in clinical practice and scientific studies for almost 20 years. Limitations of the 1999 classification like lack of a pathobiology based distinction between the specified categories,non inclusion of peri-implant diseases and conditions and lack of a clear definition of periodontal health and disease led to the development of a new classification systemin 2017. The New Classification was developed in the World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions conducted jointly by the American Academy of Periodontology and the European Federation of Periodontology. This article is a review of the historical and modern classification systems for periodontal diseases.

Keywords: periodontal diseases, diagnosis, classification, peri-implant diseases, gingivitis, periodontitis

Introduction

Periodontal diseases consist of a group of inherited or acquired conditions that affect the supporting tissues of the teeth.[1]Periodontal diseases are probably the most common disease of mankind and periodontitis is a major cause of tooth loss which leads to reduced masticatory ability,poorer dietary intake and compromised aesthetics thereby reducing the quality of life.[2] Moreover, periodontal inflammation is associated with adverse pregnancy outcomes, cardiovascular disease, stroke, pulmonary disease, and diabetes.[1]

Early diagnosis and prompt therapeutic intervention are key to the successful management of periodontal diseases.[2]

Diagnosis is an act of identifying a disease from its signs and symptoms.[3]Periodontal diagnosis is derived from information obtained from the patients medical and dental histories combined with findings from a thorough clinical examination aided, in some instances, by radiographic assessment and laboratory tests.An accurate diagnosis is the first step towards development of a effective treatment plan. In order to reach a correct diagnosis, classification systems for periodontal disease have been developed.[4] Classification systems are designed to arrange periodontal diseases into distinct categories based on current scientific data and to provide a framework for studying etiology, pathogenesis, and treatment of diseases.[4,5] In addition, a classification system makes communication between clinicians, researchers, patients and insurance companies possible by providing a common international language.[6]

A number of classification systems for periodontal diseases have been developed over the past decades.Experts in the field of periodontology have periodically assembled to develop a new classification system or to refine an existing one in order to accommodate advances in knowledge derived from scientific research.[7,8]

The purpose of this review paper is to present historical and modern classification systems for periodontal diseases.

Historical background

Periodontal diseases have afflicted humans for a very long time as indicated by ancient medical documents and human skeletal remains.Description of the periodontal diseases and their treatment is found in ancient Egyptian and Chinese writings some 4000 years ago.[9]

Hippocrates(460–377 BC) discussed the etiology of periodontal diseases and he believed that inflammation of the gums could be caused by accumulations of "pituita" or calculus and prescribed treatment for the same.A major contribution to dentistry and periodontology were from Abu'l-Qasim (Albucasis)(936-1013) as he described the technique of scaling the teeth using a set of instruments that he developed and he had a clear understanding of the major etiologic role of calculus deposits.[10]

In 18th century Pierre Fauchard described etiology of periodontal disease in his book The Surgeon Dentist and termed inflammation of gums as scurvy of the gums.Another major contribution to the understanding of periodontal diseases was made by scottish surgeon and scientist John Hunter (1728–1793) in his books "The Natural History of the Human Teeth" and ''A Practical Treatise on the Diseases of the Teeth''.[11]

In 1806 Joseph Fox published the first classification of periodontal diseases and it had three subdivisions:

• Disease peculiar to the part
• Disease resulting from extrinsic causes
• Disease resulting from the presence of teeth (Fox 1806).[12]

In Europe, the term alveolar pyorrhea_was used to describe any form of periodontal disease unrelated to the aging process and Germany-born physician Frederick H. Rehwinkel (1825–1889) introduced this term to the American dental literature.The term pyorrhea alveolaris was quickly adopted by the dental community and it persisted for a very long time.[11]

John M. Riggs (1811–1885) was the leading authority on periodontal disease in the mid-nineteenth century and the first person to limit his practice to periodontics.Riggs is considered the first specialist in this field and periodontitis in USA was known as "Riggs' disease". He realised that periodontal disease was caused by local factors i.e calculus and advocated conservative treatment by oral prophylaxis and meticulous oral hygiene.He classified periodontal disease based on extent of periodontal destruction as suppurative inflammation of the gums, and Absorption of the gums and alveolar process.[9,10,13]

During this period (i.e late 1800s and early 1900s) there was little knowledge about
etiology and pathogenesis of periodontal diseases. Publications on the subject represented the opinion of a single person who almost always based the classification on clinical manifestations and theoretical explanations of causation without any scientific evidence.[7]

The concept that dominated the classification from 1870 to 1920 has been named as the clinical characteristics paradigm by Gary C Armitage. According to Armitage three dominant paradigmsguided the classification systems for periodontal diseases that reflected the thoughts about the nature of periodontal diseases during a given historical period. These three paradigms were the clinical features paradigm (1870–1920), the classical pathology paradigm (1920–1970), and the Infection/host response paradigm (1970–present). Classification systems in the modern era represent a blend of all three paradigms.[7]

In 1879, C.G. Davis classified periodontal disease into three distinct forms:

- Gingival recession with minimal or no inflammation,
- Periodontal destruction secondary to 'lime deposits and
- 'Riggs Disease'.

A few years later G.V. Black presented the classification of periodontal diseases based on their clinical characteristics into five separate groups:

- constitutional gingivitis; including mercurial gingivitis, potassium iodide gingivitis and scurvy.
- a painful form of gingivitis.
- simple gingivitis.
- calcic inflammation of the peridental membrane.
- phagedenic pericementitis (meaning spreading ulcer or necrosis).(chronic suppurative pericementitis)

During this period, pyorrhea alveolaris was the most commonly used term for periodontal disease.[7]

Gottlieb in the 1920s, based on histopathological studies, introduced a new concept that certain forms of periodontal disease were due to degenerative changes in the periodontium and termed it as diffuse atrophy of alveolar bone. He used this term to describe a form of periodontal disease in young individuals who exhibited massive and localized bone loss around some or all of the permanent incisors and first molars with only minimal or no overt signs of gingival inflammation. Gottlieb's work had a wide spread influence and almost every classification during this period(1920-1970) had a dystrophic, atrophic or degenerative category.

Gottlieb classified periodontal diseases into four types: Schmutz-Pyorrhoe, Paradental-Pyorrhoe, alveolar atrophy or diffuse atrophy and occlusal trauma.[3,7]

In 1925 the term periodontitis was introduced by McCall & Box to designate the inflammatory disease of the periodontium. Box differentiated between Simplex periodontitis(due to local etiological factors) and Complex periodontitis (due to systemic etiologic factors).[3]

Becks (1929/1931) classified periodontal diseases into two groups and termed diseases of inflammatory origin as paradentitis and those of dystrophic form as paradentosis.[3]

Orban & Weinmann adopted this nomenclature and coined the term periodontosis to designate this degenerative type of periodontal disease.In 1942,based on the recommendations of the nomenclature committee of the American Academy of Periodontology, Orban proposed a classification according to pathologic point of view into Inflammation, Degeneration, Atrophy, Hypertrophy, and Traumatism categories.[14]

The American Academy of Periodontology organized a meeting in 1957 where the classification of periodontal diseases was revisited. Diseases were classified into Inflammatory processes and Dystrophic processes.The Inflammatory processes consisting of gingivitis(chronic, acute, necrotizing, fibrotic, desquamative, ulcerative, bullous) and periodontitis(simplex, complex).The Dystrophic processes consisting of occlusal traumatism, diffuse atrophy, gingivosis and periodontosis.[15]

The 1996 World Workshop in Periodontics concluded that the term periodontosis should be abandoned as there is insufficient evidence to identify it as a specific disease entity.[16]

The first individual to identify bacteria as the cause of periodontal disease was German dentist Adolph Witzel (1847–1906).However, Willoughby D. Miller(1853–1907) was the first true oral microbiologist.[10]He advocated that complex group of bacteria in presence of predisposing factors and local irritation cause periodontal disease what was later known as the nonspecific plaque hypothesis. However, the concept that bacteria play an important etiologic role in periodontal diseases was accepted nearly hundred years later.[7]

During the mid-1960s, microbiological studies provided scientific basis of the infectious nature of periodontal diseases. Of particular importance were the classical studies of human experimental gingivitis performed in 1965–1968 by Loe et al.which provided convincing evidence of the bacterial origin of periodontal diseases.Study by Michael G. Newman and colleagues reported that a complex and variable subgingival microbiota could be isolated from periodontosis lesions confirming that the disease was an infection.[17]

Butler in 1969 introduced the term "juvenile periodontitis" to replace periodontosis.[3]

In 1977 the World Workshop organized by American Academy of Periodontology eliminated the term periodontosis and juvenile periodontitis was adopted as a new term to describe the periodontal condition of young individuals with severe periodontal bone loss.[7] At the end of workshop periodontal diseases were categorized into juvenile periodontitis and chronic marginal periodontitis.[11]

Page & Schroeder in 1982 classified periodontitis into prepubertal, juvenile, rapidly progressive, adult periodontitis, and acute necrotizing ulcerative gingivo-periodontitis (ANUG / P). Except ANUG/P age of onset was an important distinguishing factor and it was adopted in all the subsequent classification systems.[3]

In 1986 the American Academy of Periodontology (AAP) recommended the following classification[3] :

I Juvenile periodontits
A Prepubertal periodontitis
B Localized juvenile periodontitis
C Generalized juvenile periodontitis
II Adult periodontis
III Necrotizing ulcerative gingivo-periodontitis
IV Refractory periodontitis.

JB Suzuki in 1988 proposed the following classification[8]:

Forms of gingivitis
1. Plaque-associated gingivitis
2. ANUG
3. Steroid hormone-influenced gingivitis
4. Medication-influenced gingival overgrowth
5. Other forms of gingivitis

Forms of periodontitis
1.Adult periodontitis
2.Rapidly progressive periodontitis:
type A
3.Rapidly progressive periodontitis:
type B
4.Juvenile periodontitis
5.Postjuvenile periodontitis
6.Prepubertal periodontitis

As a refinement to the previous classification, AAP at the 1989 World Workshop in Clinical Periodontics recommended a new classification system[7] .The disease category "Early-Onset Periodontitis" (EOP) was introduced to designate a group of dissimilar destructive periodontal diseases that affected young patients (i.e., prepubertal, juvenile, and rapidly progressive periodontitis) if they exhibited significant attachment loss in the presence of little local factors (plaque and calculus) and were less than 35 years of age[5,18].Periodontal disease were categorized as following[3]:

I Adult periodontitis
II Early onset periodontitis
A Prepubertal periodontits
1 Generalized
2 Localized
B Juvenile periodontitis
1 Generalized
2 Localized
C Rapidly progressive periodontitis
III Periodontitis associated with systemic diseases
IV Necrotizing ulcerative periodontitis
V Refractory periodontitis.

This classification depended on the age of onset and rate of disease progression.Periodontitis associated with systemic disease was added as a new category.[7]

One of the main limitations of the 1989 classification system was lack of a category for gingival diseases. Other shortcomings include inappropriate emphasis on age of onset of disease and rates of progression, a significant overlap between disease categories and inadequate classification criteria.[5]

In 1993 Ranney provided a simplified classification system consisting of Gingivitis, adult periodontitis, early onset periodontitis, necrotizing ulcerative periodontitis, and periodontal abscess eliminating 'Periodontitis Associated with Systemic Disease'and refractory peridontitis categories.[19] This classification system was widely accepted as it was easy to use.However it was not possible to claasify all the periodontal conditions with this classification system.[7]

In the same year European Workshop on Periodontology suggested a simple classification distinguishing between early onset periodontitis, adult periodontitis and necrotizing periodontitis[7]

In order to address the limtations associated with 1989 classification system, an International Workshop for a Classification of Periodontal Diseases and Conditions was held in 1999 and a new classification was agreed upon.[5]

The 1999 AAP classification system is comprehensive enlisting over 40 gingival diseases and
seven major categories of destructive periodontal diseases.[4]The terms adult periodontitis and early onset periodontitis were replaced with chronic periodontitis and aggressive periodontitis, respectively, to eliminate the age dependent criteria.[5]In addition, refractory periodontitis, rapidly progressive periodontitis(RPP) and prepubertal periodontitis were eliminated as separate disease categories because of their extraordinary heterogeneity[7] and Periodontitis associated with Systemic Disease was replaced with periodontitis as a manifestation of systemic diseases.[5]

The disease category of Necrotizing Ulcerative Periodontitis was replaced With Necrotizing Periodontal Diseases. Abscesses of the periodontium , periodontitis associated with endodontic lesions and developmental or acquired deformities and conditions were added as new categories.[5]

Classification of Periodontal Diseases and Conditions(1999)[10]

(I) Gingival Diseases
Plaque-induced gingival diseases
Non—plaque-induced gingival lesions
(II) Chronic Periodontitis
Localized
Generalized
(III) Aggressive Periodontitis
Localized
Generalized
(IV) Periodontitis as a Manifestation of Systemic Diseases
(V) Necrotizing Periodontal Diseases
Necrotizing ulcerative gingivitis (NUG)
Necrotizing ulcerative periodontitis (NUP)
(VI) Abscesses of the Periodontium
Gingival abscess
Periodontal abscess
Pericoronal abscess
(VII) Periodontitis Associated with Endodontic Lesions
Endodontic-periodontal lesion
Periodontal-endodontic lesion
Combined lesion
(VIII) Developmental or Acquired Deformities and Conditions
Localized tooth-related factors that predispose to plaque-induced gingival diseases or periodontitis
Mucogingival deformities and conditions around teeth
Mucogingival deformities and conditions on edentulous ridges
Occlusal trauma

Other classification systems for periodontal diseases proposed by various authors are enlisted in Table 1.[20]

Current classification system

The 1999 classification of periodontal diseases has provided a workable framework that has been used to classify periodontal diseases and conditions for the past 19 years in both clinical practice and scientific research. Since then new evidence emerging from population studies, basic science investigations, and prospective studies has made the revision of the present classification mandatory. Moreover, the 1999 classification had some short comings including overlap and lack of clear pathobiology based distinction between the disease categories, lack of a category for perimplant diseases and conditions, difficulties in clinical implementation, diagnostic imprecision and lack of definition for periodontal health and disease.[21,22]Classification systems should be viewed as flexible structures that allow modification based on current thinking and new knowledge.[7]

Van der Veldenin year 2000 proposed a new classification system based on nominalistic disease concept. This classification was based on extent, severity, age, and clinical characteristics.[3] According to Armitage, this would be a return to the domination of the Clinical Characteristics paradigm that reigned from approximately 1870 to 1920 when we knew little about the nature of periodontal diseases.[7]

In 2017, the American Academy of Periodontology and the European Federation of Periodontology co-sponsored the World Workshop on the Classification of Periodontal

and Peri-implant Diseases and Conditions to update the 1999 classification system and address it's limitations (Table 2).[22]

The objectives of 2017 world workshop were to revisit the current classification system, incorporate current knowledge relevant to its etiology and pathogenesis and propose a new classification framework along with case definitions.[22]

Periodontal health and Gingival health

Periodontal health is defined as a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease.[23]

Periodontal health should be based upon the absence of disease, as assessed clinically, associated with gingivitis, periodontitis, or other periodontal conditions.It may include patients who have had a history of successfully treated gingivitis or periodontitis, or other periodontal conditions, who are able to maintain their dentition free from inflammation(table 3).[23]

A definition of periodontal health is critical to have a common reference point for assessing periodontal disease and determining meaningful treatment outcomes.[24]

Four levels of periodontal health have been proposed depending on the state of the periodontium (structurally and clinically sound or reduced) and the relative treatment outcomes: (1) pristine periodontal health, with a structurally sound and uninflamed periodontium with normal support; (2) clinical periodontal health, with a structurally and clinically sound (intact) periodontium; (3) periodontal disease stability, with a reduced periodontium, and (4) periodontal disease remission/control,with a reduced periodontium.[24]

The term pristine clinical health is characterized by no attachment loss, no bleeding on probing (BoP), no sulcular probing >3 mm and no redness, clinical swelling/edema, or pus. The term clinically healthy should refer to tissue that demonstrates an absence, or very low level, of clinical indicators of inflammation such as BoP and inflammatory markers in gingival crevicular fluid.[24]

Gingival diseases and conditions

Gingivitis is characterized by signs and symptoms of inflammation confined to the gingiva without clinical attachment loss(CAL). [23]

There are two broad categories of gingival diseases:

• Dental plaque biofilm-induced gingivitis

• Non–dental plaque-induced gingival diseases

Dental plaque-induced gingivitis

Dental plaque-induced gingivitis may arise on an intact periodontium or on a reduced periodontium in either a non-periodontitis patient or in a currently stable periodontitis patient(Table 4).Gingivitis can be defined as mild, moderate and severe based on percentages (e.g. mild = < 10%, moderate = 10%-30%, severe = > 30% sites) or Grades (e.g. grade 1 to 5 in 20% quintiles for % sites bleeding on probing). It can further be defined as localized(10%-30% bleeding sites) and generalized(> 30% bleeding sites).[23]

Dental plaque-induced gingivitis is classified as following:

A. Associated with biofilm alone

B. Mediated by either Systemic Risk Factors or Local Risk Factors

i. Systemic Risk Factors (modifying factors)

(a) Smoking, (b)Hyperglycemia, (c)Nutritional factors, (d)Pharmacological agents, (e) Sex steroids hormones (Puberty, menstrual cycle, pregnancy, oral contraceptives) (f) Hematological conditions .

ii. Local Risk Factors (predisposing factors)

(a) Dental plaque biofilm retaining factors(e.g.,prominent restoration margins)

(b) Oral dryness

C. Drug-influenced gingival enlargement

Non-dental plaque biofilm–induced.

Non-dental plaque biofilm-induced gingival diseases consist of a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal.Such lesions may be

manifestations of a systemic disorder or may represent a lesion localized to the oral tissue.

Non-dental plaque biofilm-induced gingival diseases include the following[24]:

A. Genetic/developmental disorders

B. Specific infections

C. Inflammatory and immune conditions

D. Reactive processes

E. Neoplasms

F. Endocrine, nutritional and metabolic diseases

G. Traumatic lesions

H. Gingival pigmentation

Classification of Periodontitis

Based on current knowledge on pathophysiology, periodontitis is categorized into three forms: periodontitis**,** necrotizing periodontitis and periodontitis as a manifestation of systemic disease.[24]

Differential diagnosis is based on history and the specific signs and symptoms of necrotizing periodontitis and the presence or absence of an uncommon systemic disease that definitively alters the host immune response.[24]

Periodontitis

The diseases previously recognized as "chronic" or "aggressive" are now grouped under a single category periodontitis.[24] Periodontitis is characterized by microbially-associated, host-mediated inflammation that results in the destruction of supporting structures of the teeth.[25]

A patient is diagnosed as a periodontitis case in clinical practice if:

1. Interdental CAL is detectable at ≥2 non-adjacent teeth, or

2. Buccal or oral CAL ≥3 mm with pocketing >3 mm is detectable at ≥2 teeth and the observed CAL cannot be ascribed to non-periodontal causes.[25]

Periodontitis is further characterized based on a multidimensional staging and grading system.

Staging is largely dependent upon the severity of disease at presentation as well as on the complexity of disease management and involves four categories (stages 1 through 4)(Table 5).[24]

Grading provides information about 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.Grading includes three levels (grade A – low risk, grade B – moderate risk, grade C – high risk for progression)(Table 6).[24,25]

Necrotizing periodontitis

Necrotizing periodontitis is categorized as a separate disease entity based on evidence from: i) a distinct pathophysiology characterized by prominent bacterial invasion and ulceration of epithelium; ii) rapid and full thickness destruction of the marginal soft tissue resulting in characteristic soft and hard tissue defects; iii) prominent symptoms; and iv) rapid resolution in response to specific antimicrobial treatment.[25] Clinical features of Necrotizing periodontitis include history of pain, presence of ulceration of the gingival margin and/or fibrin deposits at sites with loss of gingival papillae, and, in some cases, denudation of the marginal alveolar bone.[25]

In the new classification Necrotizing periodontitis is categorized under Necrotizing periodontal diseases(NPD) which are a group of conditions that share a common characteristic of necrosis of the gingival or periodontal tissues. NPD include Necrotizing gingivitis(NG), Necrotizing periodontitis(NP) and Necrotizing stomatitis(NS)(Table 7).[26]

NPD are bacterial infections associated with compromised host immune response. Studies have suggested that NG and NP may represent different stages of the same disease and may even progress to more severe forms such as necrotizing stomatitis (NS) and noma.[27]

Periodontitis as a manifestation of systemic disease

Systemic disease is defined as a disease that affects multiple organs or tissues or that affects the body as a whole.[28]

The new classification of periodontal diseases and conditions also includes systemic disorders and certain medications that affect the periodontal supporting tissues and that the primary diagnosis should be the systemic disease according to International Statistical Classification of Disease (ICD).[22,25]

These systemic disorders affecting the periodontal attachment apparatus include:

1a. Rare systemic diseases that affect the course of periodontitis (e.g., Papillon Lefevre Syndrome, leucocyte adhesion deficiency, and hypophosphatasia) and have a major impact resulting in the early presentation of severe periodontitis. These conditions are grouped as "Periodontitis as a Manifestation of Systemic Disease",and classification should be based on the primary systemic disease(Table 8).[22,29]

These diseases and conditions include genetic disorders that affect the host immune response or affect the connective tissues, metabolic and endocrine disorders, and inflammatory conditions.[29]

1b. Common systemic disorders that affect the course of periodontitis (e.g., diabetes mellitus) resulting in increased occurrence and severity.

Periodontitis associated with Diabetes is not a distinct disease,but diabetes should be considered as an important modifying factor of periodontitis, and is included in a clinical diagnosis of periodontitis as a descriptor.[29]

2. Systemic diseases that affect the periodontal supporting tissues independently of dental plaque biofilm induced inflammation and are categorized separately in the new classification system as Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues (e.g., squamous cell carcinoma, Langerhans cell histiocytosis).[22,28]

Other conditions affecting the periodontium

Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues:

Systemic diseases that affect the periodontal supporting tissues independently of dental plaque biofilm induced inflammation (e.g., squamous cell carcinoma, Langerhans cell histiocytosis) resulting in breakdown of periodontal tissues are grouped as **"Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues"**.[22,29] The majority of these lesions arise from the deeper periodontal tissue and are diagnosed by biopsy and histopathologic examination. Systemic diseases under this category include Neoplasms and Other disorders that may affect periodontal tissue(table 8).[28]

Periodontal abscesses(PA)

Periodontal abscesses are acute periodontal lesions characterised by an ovoid swelling in the gingiva along the lateral part of the root,BOP, pain and increased tooth mobility.

A periodontal abscess may develop in a pre-existing periodontal pocket or at a previously periodontally healthy commonly associated with a history of impaction or harmful habits (Table 9).[27]

Periodontal abscesses (PA) are common dental emergencies requiring prompt treatment and they can cause rapid tissue destruction with a negative impact on the prognosis of the affected tooth.Depending upon the etiological factors different abscesses may occur in in the periodontal tissues, such as pulp necrosis (endodontic, periapical or dentoalveolar abscesses), periodontal infections (gingival or periodontal abscess), pericoronitis (pericoronal abscess), trauma, surgery, or foreign body impaction.[27]

Endodontic-Periodontal lesions (EPL)

EPL are clinical conditions defined by a pathological communication between the pulpal and periodontal tissues at a given tooth involving both the pulp and may occur in acute or chronic forms.[27] The primary etiology of these lesions might be associated with endodontic and/or periodontal infections or trauma and/or iatrogenic factors.The most common signs and symptoms associated with a tooth affected by an EPL are deep periodontal pockets reaching or close to the apex,negative or altered response to pulp vitality tests,bone resorption in the apical or furcation region, spontaneous pain or pain on palpation and percussion, purulent exudate, tooth mobility, sinus tract, crown, and gingival color alterations. These conditions severely compromise the prognosis of the tooth and require multidisciplinary evaluation, diagnosis, and treatment.[27]

EPLs are classified according to signs and symptoms that have direct impact on their prognosis and treatment, such as presence or absence of fractures and perforations, and presence or absence of periodontitis(Table 10).[27]

Mucogingival deformities and conditions around teeth

Normal mucogingival condition is defined as the absence of pathosis like gingival recession, gingivitis and periodontitis.[29]

Mucogingival defects are a group of conditions that affect a large number of patients particularly adults(Table 11).[30]

Amongst Mucogingival deformities lack of keratinized tissue and gingival recession are the most common and occur in patients with both high and low standards of oral hygiene .[30]

Gingival recession is defined as an apical shift of the gingival margin caused by

different conditions/pathologies and is associated with hypersensitivity, the development of root surface alterations in the form of caries and noncarious cervical lesions and impaired esthetics.[29]

In the 2017 classification system periodontal biotype is replaced with periodontal phenotype to describe the combination of gingival phenotype (three-dimensional gingival volume) and the thickness of the buccal bone plate (bone morphotype).[29] A new treatment-oriented classification of recession proposed by Cairo et al. categorizes recession into three types based on interdental CAL:

• **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 is less than or equal to the buccal attachment loss.

• **Recession Type 3 (RT3)**: Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss is greater than the buccal attachment loss.[29]

Although a lack of keratinized tissue is considered to be a predisposing factor for the development of gingival recessions and inflammation, optimal oral hygiene prevents attachment loss even with minimal amounts of keratinized tissue.However,patients with thin periodontal biotypes, suboptimal oral hygiene, and requiring restorative/ orthodontic treatment are more prone to gingival recession.[30]

Mucogingival diagnoses include:

a)Mucogingival condition with gingival recessions

b) Mucogingival condition without gingival recessions[29]

Traumatic occlusal forces

The term Excessive occlusal force is renamed as traumatic occlusal force and is defined as any occlusal force resulting in injury of the teeth and/or the periodontal attachment apparatus. The presence of traumatic occlusal forces may be indicated by one or more of the following: fremitus, tooth mobility, thermal sensitivity, excessive occlusal wear, tooth migration, discomfort/pain on chewing, fractured teeth, radiographically widened periodontal ligament space, root resorption,and hypercementosis.[29]

discomfort/pain on chewing, fractured teeth, radiographically widened periodontal ligament space, root resorption,and hypercementosis.[29]

Occlusal trauma is a term used to describe the injury to the periodontal attachment apparatus caused by traumatic occlusal forces.

Primary occlusal trauma is injury resulting in tissue changes from traumatic occlusal forces applied to a tooth or teeth with normal periodontal support.

Secondary occlusal trauma is injury resulting in tissue changes from normal or traumatic occlusal forces applied to a tooth or teeth with reduced support(Table 12).[29]

Since occlusal trauma is a histologic term, its clinical diagnosis is made by various clinical and radiographic indicators (Table13).[31]

Evidence from animal and human studies indicate that traumatic occlusal force do not initiate periodontitis or alters it's progression. There is no evidence that traumatic

occlusal forces cause non-carious cervical lesions or gingival recession.[29,31]

Evidence from various studies indicate that certain orthodontic forces can adversely affect the periodontium and result in root resorption, pulpal disorders, gingival recession and alveolar bone loss while as good plaque control in teeth with a reduced but healthy periodontium can undergo successful tooth movement without compromising the periodontal support.[29]

Prostheses and tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

Several conditions associated with fabrication and presence of dental prostheses[32] and tooth-related factors may predispose to diseases of the periodontium (table 14).[29]

Placement of restoration margins within the supracrestal tissue attachment(bologic width), hypersensitivity reactions to the prosthesis dental material,procedures adopted for the fabrication of dental restorations and fixed prostheses and tooth related factors like cervical enamel projections,enamel pearls,developmental grooves, tooth fractures, root fractures, root resorption, tooth malposition,root proximity and open contacts may predispose to the initiation and progression of gingivitis and periodontitis especially in patients with poor plaque control and those who do not attend periodic maintenance appointments.[32]

Abnormal dentoalveolar relationships associated with altered passive tooth eruption may be clinically associated with the formation of pseudopockets and esthetic concerns.[29]

Peri-implant diseases and conditions

Osseointegrated dental implants are a well known therapeutic modality for the replacement of absent or lost teeth. Functional implants and their restorations may be subject to mechanical and biological complications. Biological complications associated with dental implants are mostly inflammatory conditions induced by the accumulation of bacterial biofilm and include peri-implant mucositis and peri-implantitis.[33]

The 2017 world workshop developed a new category for peri-implant health,peri-implant mucositis, peri-implantitis and relevant aspects of implant site conditions and deformities so that the clinician may assign a proper diagnosis and select a proper treatment modality in cases where disease is present.[22,33,34]

Peri-implant health

Peri-implant tissues include both soft and hard tissues that occur around Osseo integrated dental implants. Knowledge of the characteristics of healthy peri-implant tissues is important for the recognition of disease.[35] The healthy peri-implant mucosa is characterized by absence of clinical signs of inflammation (i.e. erythema and swelling) including lack of bleeding on probing and absence of further bone loss following initial healing. Peri-implant health can exist around implants with normal or reduced bone support. However, probing depths could vary depending on the height of the soft tissue at the implant location and should be ≤5.0 mm .[33]

Peri-implant mucositis

Peri-implant mucositis is an inflammatory lesion of the soft tissues surrounding an endosseous implant in the absence of loss of supporting bone characterized by signs of inflammation such as redness, swelling, line or drop of bleeding within 30 seconds following probing and suppuration. Peri-implant mucositis is the precursor of peri-implantitis and is primarily caused by biofilm accumulation at the implant–mucosa interface. Peri-implant mucositis a reversible condition and complete resolution of the lesion occurs upon institution of biofilm removal measures.[33,36]

Peri-implantitis

Peri-implantitis is a plaque-associated pathological condition characterized by inflammation in the connective tissue around dental implants and progressive loss of supporting bone. BOP with or without suppuration, increased probing depths compared to baseline measurements and progressive bone loss identified on radiographs represents peri-implantitis.[37]In the absence of initial radiographs and probing depths, peri-implantitis is diagnosed by radiographic evidence of bone level ≥3 mm and/or probing depths ≥6 mm in conjunction with profuse bleeding. **patients** Patients diagnosed with peri-implant mucositis may develop peri-implantitis, especially in the absence of regular maintenance care.[34]

Risk factors/imdicators of peri-implantitis include a history of chronic periodontitis, poor plaque control measures, and lack of regular maintenance care after implant therapy.[37]

Hard and soft tissue implant site deficiencies

Diminished dimensions of the alveolar process/ridge and soft tissue as a result of normal healing following tooth loss leads to hard and soft-tissue deficiencies at implant sites.[22,38]Tissue deficiencies at implant sites may occur from a number of reasons including natural resorption processes following tooth extraction, trauma, infectious diseases such as periodontitis, peri-implantitis, endodontic infections, growth and development, expansion of the sinus floor, anatomical preconditions, mechanical overload, thin soft tissues, lack of keratinized mucosa,

Mal positioning of implants, migration of teeth, lifelong growth, and systemic diseases. Tissue deficiencies around implants can lead to various complications and compromise implant survival. Knowledge of the etiology of hard and soft-tissue deficiencies is necessary to develop appropriate therapeutic measures with the aim of improving clinical outcomes of implant therapy.[38]

Conclusion

1. Periodontal diseases are among the most common diseases affecting the mankind. Early diagnosis and prompt treatment. Is essential for successful management of periodontal diseases. Classification systems have been developed to facilitate diagnosis and treatment of periodontal diseases as well as to provide a framework for studying disease prevalence, natural history, etiology and pathogenesis. Moreover a classification system provides a common international language for communication amongst clinicians, patients and researchers. Over the past hundred years many classification systems for periodontal diseases have been proposed and updated to keep pace with the advances in knowledge derived from scientific research. The classification should be considered as a flexible system to be regularly updated as new knowledge about the disease emerges. The 1999 Classification of Periodontal Disease and Conditions has been used for the past 17 years in clinical practice and scientific research. However 1999 classification had it's shortcomings like implementation difficulties, lack of clear diagnostic criteria and overlap between disease categories. Since then emergence of new knowledge and better understanding of the periodontal disease led to the revision of the 1999 classification systems in 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions co-sponsored by the American Academy of Periodontology and the European Federation of Periodontology.Several important changes have been made like recategorization of the three forms of periodontitis, introduction of staging and grading system to designate disease severity and susceptibility and a new category for peri-implant diseases and conditions. The staging and grading system helps to visualize the past disease experience,current status and risk of future disease progression.Clear definitions of periodontal health and disease have been provided and a new classification for gingival recession has been introduced.
2. The New Classification system has been developed by an international group of experts and will be adopted by students, clinicians and scientists throughout the world.

References

1. Pihlstrom B L, Michalowicz B S, Johnson N W. Periodontal diseases. Lancet 2005; 366: 1809–1820
2. Tonetti MS, Jepsen S, Jin L, Otomo-Corgel J. Impact of the global burden of periodontal diseases on health, nutrition and wellbeing of mankind: A call for global action. J Clin Periodontol. 2017;00:1–7
3. van der Velden U .Purpose and problems of periodontal disease classification. Periodontology 2000 2005;39:13–21
4. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. Periodontol 2000 2004: 34: 9–21.
5. Armitage GC. Development of a Classification System for Periodontal Diseases and Conditions. Ann Periodontol. 1999 Dec; 4(1):1-6.
6. Hoath, Wiebe, Garcia Fulle De Owen, et al. Current status of the classification of periodontal diseases. Can J Dent Hyg 2016;50(3): 140-144
7. Armitage GC. Classifying periodontal diseases – a longstanding dilemma. Periodontol 2000 2002: 30: 9–23.
8. Suzuki J B. Diagnosis and classification of the periodontal diseases. Dent Clin N Am 1988;32:195-216.
9. Löe H.Periodontal diseases: a brief historical perspective.Periodontology 2000 1993;2:7-12.
10. Newman MG, Carranza FA, Takei H, Klokkevold PRThe Historical Background of Periodontology in Carranzas clinical Periodontology. 10th ed. Elsevier health sciences; 2006.P 39-57.
11. Dentino A, Lee S, Mailhot J , Hefti AF. Principles of periodontology Periodontology 2000 .2013;61:16–53
12. Eaton K,Ower P.Practical periodontics Leo Briggs Classification of Periodontal Diseases 2015:83
13. Gold SI. Periodontics. The past Part (II). The development of modern periodontics Journal of Clinical Periodontology.1985;12:171-189
14. Orban B. Classification and nomenclature of periodontal diseases. (Based on pathology, etiology, and clinical picture). J Periodontol 1942: 13: 88–91.
15. Arthur-Jean Held, Periodontology. From its origins up to 1980: a survey. 1989 Springer Basel AG p124.
16. Armitage GC, Cullinan MP . Comparison of the clinical features of chronic and aggressive periodontitis. Periodontol 2000 2010: 53: 12–27.
17. Armitage GC, Robertson PB. The biology, prevention, diagnosis and treatment of periodontal diseases Scientific advances in the United States.JADA 2009;140(9 suppl):36-43.
18. Wiebe CB, Putnins EE. The Periodontal Disease Classification System of the American Academy of Periodontology —An Update. J Can Dent Assoc 2000; 66:594-7.
19. Ranney RR. Classification of periodontal diseases. Periodontol 2000 1993: 2: 13–25.
20. Stephen B Weinmann , Philip R Geron A chronological classification of periodontal disease: a review J Int Acad Periodontol 2011;13(2):31-9.
21. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: Consensus report of workgroup 2 of the

2017 world workshop on the classification of periodontal and peri-implant diseases and conditions. J Periodontol. 2018;89(S1):S173–82.P164
22. Caton J, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and periimplant diseases and conditions – Introduction and key changes from the 1999 classification. J Clin Periodontol. 2018;45(Suppl 20):S1–S8.Chapple ILC, Mealey BL, et al. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45(Suppl 20):S68–S77.
23. Lang NP, Bartold PM. Periodontal health. J Clin Periodontol. 2018;45(Suppl 20):S9–S16
24. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. J Clin Periodontol. 2018;45(Suppl 20):S149–S161.
25. Papapanou PN, Sanz M, et al. Periodontitis: Consensus report of Workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45(Suppl 20):S162–S170.
26. Herrera D, Retamal-Valdes B, Alonso B, Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. J Clin Periodontol. 2018;45(Suppl 20):S78–S94.
27. Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: Case definitions and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20):S171–S189.
28. Jepsen S, Caton JG, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45(Suppl 20):S219–S229
29. Cortellini P, Bissada NF. Mucogingival conditions in the natural dentition: Narrative review, case definitions, and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20):S190–S198.
30. Fan J, Caton JG. Occlusal trauma and excessive occlusal forces: Narrative review, case definitions, and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20):S199–S206.
31. Ercoli C, Caton JG. Dental prostheses and tooth-related factors. J Clin Periodontol. 2018;45(Suppl 20):S207–S218.
32. Renvert S, Persson GR, Pirih FQ, Camargo PM. Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations. J Clin Periodontol. 2018;45(Suppl 20): S278–S285
33. Berglundh T, Armitage G, et al. Periimplant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45(Suppl 20):S286–S291.
34. Araujo MG, Lindhe J. Peri-implant health. J Clin Periodontol. 2018;45(Suppl 20):S230–S236.
35. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. J Clin Periodontol. 2018;45 (Suppl 20):S237–S245
36. Schwarz F, Derks J, Monje A, Wang H-L. Peri-implantitis. J Clin Periodontol. 2018;45(Suppl 20):S246–S266.
37. Hämmerle CHF, Tarnow D. The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review. J Clin Periodontol. 2018;45(Suppl 20):S267–S277.

Legend Tables

Table 1: Classification systems for periodontal diseases proposed by various authors.[20]

Kantorowicz---1924
McCall and Box---1925
Simonton---1927
Haupl and Lang---1927
Jaccard---1930/1933
Roy---1935
Robinson---1935
Weski---1937
Isadore Weinmann---1934-1957
Thoma and Goldman---1937
Fish---1944/1952
Hine and Hine---1944
Hulin---1949
Held---1949 Pucci---1950
Lyons---1951
Miller---1950
Kerr---1951
Goldman---1956
McCall---1956
Robinson---1959
Carranza---1959
Ray---1962
Glickman---1964
Drum---1975

Table 2: Classification of Periodontal and Peri-Implant Diseases and Conditions 2017

CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017[22]										
Periodontal Diseases and Conditions										
Periodontal Health,Gingival Diseases and Conditions			Periodontitis			Other Conditions Affecting the Periodontium				
Periodontal Health and Gingival Health	Gingivitis: Dental Biofilm-Induced	Gingival Diseases: Non-Dental Biofilm-Induced	Necrotizing Periodonal Diseases	Periodontitis	Periodontitis as a Manifestation of Systemic Disease	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
Peri-Implant Diseases and Conditions										
Peri-Implant Health			Peri-Implant Mucositis			Peri-Implantitis			Peri-Implant and Hard Tissue Deficiencies	

Table 3: Periodontal Health[23]

Intact periodontium	Reduced periodontium: Non- periodontitis patient	Reduced periodontium: Successfully treated Stable- periodontitis patient
Probing attachment loss No Bleeding on probing <10% Probing pocket depths ≤3 mm (assuming no pseudo pockets) Radiological bone loss No	Probing attachment loss Yes Bleeding on probing <10% Probing pocket depths ≤3 mm (all sites and assuming no pseudo pockets) Radiological bone loss Possible	Probing attachment loss Yes Bleeding on probing <10% Probing pocket depths ≤4 mm(no site ≥ 4 mm with BOP (all sites and assuming no pseudo pockets) Radiological bone loss Yes

Table 4: Dental plaque-induced Gingivitis[23]

intact periodontium	Reduced periodontium: Non- periodontitis patient	Reduced periodontium: Successfully treated Stable- periodontitis patient
Probing attachment loss No Bleeding on probing Yes(≥10%) Probing pocket depths ≤3 mm (assuming no pseudo pockets) Radiological bone loss No	Probing attachment loss Yes Bleeding on probing <10% Probing pocket depths ≤3 mm (all sites and assuming no pseudo pockets) Radiological bone loss Possible	Probing attachment loss Yes Bleeding on probing <10% Probing pocket depths ≤3 mm (all sites and assuming no pseudo pockets) Radiological bone loss Yes

Table 5: Periodontitis stage[25]

Periodontitis stage		Stage 1	Stage 2	Stage 3	Stage 4
Severity	Interdental CAL at site of greatest loss	1-2 mm	3-4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15%-33%)	Extending to middle or apical third of the root	Extending to middle or apical third of the root
	Tooth loss	No tooth loss due to periodontitis		Tooth loss due to periodontitis of ≤4	Tooth loss due to periodontitis of ≥5
Complexity	Local	Maximum probing depth ≤4 mm Mostly horizontal bone loss	Maximum probing depth ≤5 mm Mostly horizontal bone loss	In addition to stage II complexity: – Probing depth ≥6 mm – Vertical bone loss ≤3 mm – Furcation involvement Class II or III – Moderate ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: – Masticatory dysfunction – Secondary occlusal trauma (tooth mobility degree ≥2) – Severe ridge defect – Bite collapse, drifting, flaring – Less than 20 remaining teeth (10 opposing pairs)
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern			

Table 6: Periodontitis grade[25]

Periodontitis grade			Grade A: Slow rate of progression	Grade B: Moderate rate of progression	Grade C: Rapid rate of progression
Primary criteria	Direct evidence of progression	Longitudinal data (Radiographic bone loss or CAL)	Evidence of no loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/ incisor pattern; lack of expected response to standard bacterial control therapies)
	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
Grade modifiers		Diabetes	Normoglycemic/no diagnosis of diabetes	HbAlc <7.0% in patients with diabetes	HbAlc ≥7.0% in patients with diabetes
Risk of systemic impact of periodontitis	Inflammatory burden	High sensitivity CRP (hsCRP)	<1mg/L	1 to 3 mg/L	>3 mg/L
Biomarkers	Indicators of CAL/bone loss	Saliva, gingival crevicular fluid,serum			

Table 7: Classification of necrotizing periodontal diseases (NPD)[26]

Category	Patients	Predisposing conditions	Clinical condition
Necrotizing periodontal diseases in chronically, severely compromised patients	In adults	HIV+/AIDS with CD4 counts < 200 and detectable viral load	NG, NP, NS, Noma. Possibleprogression
		Other severe systemic conditions(immunosuppression)	
	In children	Severe malnourishments	
		Extreme living conditions	
		Severe (viral) infections	
Necrotizing periodotal diseases in temporarily and/or moderately compromised patients	In gingivitis patients	Uncontrolled factors: stress, nutrition, smoking, habits	Generalized NG. Possibleprogression to NP
		Previous NPD: residual craters	
		Local factors: root proximity, toothmalposition	Localized NG. Possibleprogression to NP
		Common predisposing factors forNPD	NG. Infrequent progression
	In periodontitis patients		NP. Infrequent progression

Table 8: Classification of systemic diseases and conditions that affect the periodontal supporting tissues.[29]

Classification	Disorders	ICD-10 code
1	Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation	
1.1	Genetic disorders	
1.1.1	Diseases associated with immunologic disorders	
	Down syndrome	Q90.9
	Leukocyte adhesion deficiency syndromes	D72.0
	Papillon-Lefèvre syndrome	Q82.8
	Haim-Munk syndrome	Q82.8
	Chediak-Higashi syndrome	E70.3
	Severe neutropenia	
	– Congenital neutropenia (Kostmann syndrome)	D70.0
	– Cyclic neutropenia	D70.4
	Primary immunodeficiency diseases -Chronic granulomatous disease	 D71.0
	– Hyperimmunoglobulin Esyndromes	D82.9
	Cohen syndrome	Q87.8
1.1.2.	Diseases affecting the oral mucosa and gingival tissue	
	Epidermolysis bullosa	
	– Dystrophic epidermolysis bullosa	Q81.2
	– Kindler syndrome	Q81.8
	Plasminogen deficiency	D68.2
1.1.3	Diseases affecting the connective tissues	
	Ehlers-Danlos syndromes (types IV,VIII)	Q79.6
	Angioedema (C1-inhibitor deficiency)	D84.1
	Systemic lupus erythematosus	M32.9
1.1.4	Metabolic and endocrine disorders	
	Glycogen storage disease	E74.0
	Gaucher disease	E75.2
	Hypophosphatasia	E83.30
	Hypophosphatemic rickets	E83.31
	Hajdu-Cheney syndrome	Q78.8
1.2	Acquired immunodeficiency diseases	
	Acquired neutropenia	D70.9
	HIV infection	B24
1.3	Inflammatory diseases	
	Epidermolysis bullosa acquisita	L12.3
	Inflammatory bowel disease	K50, K51.9,K52.9
2	Other systemic disorders that influence the pathogenesis of periodontal diseases	
	Diabetes mellitus	E10 (type1), E11 (type 2)
	Obesity	E66.9
	Osteoporosis	M81.9
	Arthritis (rheumatoid arthritis, osteoarthritis)	M05, M06, M15-M19
	Emotional stress and depression	F32.9
	Smoking (nicotine dependence)	F17
	Medications	
3	Systemic disorders that can result in loss of periodontal tissues independent of periodontitis	
3.1	Neoplasms	
	Primary neoplastic diseases of the periodontal tissues	
	– Oral squamous cell carcinoma	C03.0 – 1
	– Odontogenic tumors	D48.0
	– Other primary neoplasms of the periodontal tissues	C41.0
	Secondary metastatic neoplasms of the periodontal tissues	C06.8
3.2	Other disorders that may affect the periodontal tissues	
	Granulomatosis with polyangiitis	M31.3
	Langerhans cell histiocytosis	C96.6
	Giant cell granulomas	K10.1
	Hyperparathyroidism	E21.0
	Systemic sclerosis (scleroderma)	M34.9
	Vanishing bone disease (Gorham-Stout syndrome)	M89.5

Table 9: Classification of periodontal abscesses based on the etiologic factors involved

Periodontal abscess in periodontitis patients (in a pre-existing periodontal pocket)	Acute exacerbation	Untreated periodontitis	
		Non-responsive to therapy periodontitis	
		Supportive periodontal therapy	
	After treatment	Post scaling	
		Post surgery	
		Post-medication	Systemic antimicrobials
			Other drugs: nifedipine
Periodontal abscess in non-periodontitis patients (not mandatory to have a pre-existing periodontal pocket)	impaction		Dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls
	Harmful habits		Wire or nail biting and clenching
	Orthodontic factors		Orthodontic forces or a cross-bite
	Gingival overgrowth		
	Alteration of root surface	Severe anatomic alterations	Invaginated tooth, dens evaginatus or odontodysplasia
		Minor anatomic alterations	Cemental tears, enamel pearls or developmental grooves
		Iatrogenic conditions	Perforations
		Severe root damage	Fissure or fracture, cracked tooth syndrome
		External root resorption	

TABLE 10. Classification of endo-periodontal lesions[27]

Endo-periodontal lesion with root damage	Root fracture or cracking	
	Root canal or pulp chamber perforation	
	External root resorption	
Endo-periodontal lesion without root damage	Endo-periodontal lesion in periodontitis patients	Grade 1 – narrow deep periodontal pocket in 1 tooth surface
		Grade 2 – wide deep periodontal pocket in 1 tooth surface
		Grade 3 – deep periodontal pockets in > 1 tooth surface
	Endo-periodontal lesion in non-periodontitis patients	Grade 1 – narrow deep periodontal pocket in 1 tooth surface
		Grade 2 – wide deep periodontal pocket in 1 tooth surface
		Grade 3 – deep periodontal pockets in > 1 tooth surface

Table 11: Mucogingival deformities and conditions around teeth[30]

1.Periodontal biotype
- a. thin scalloped
- b.thick scalloped
- c.thick flat

2. gingival/soft tissue recession
- a. facial or lingual surfaces
- b. interproximal (papillary)
- c. severity of recession(Cairo RT 1,2,3)
- d. gingival thickness
- e. gingival width
- f. presence of NCCL/cervical caries
- g.patient aesthetic concern(Smile Esthetic Index)
- h. presence of hypersensitivity

3. lack of keratinized gingiva
4. decreased vestibular depth
5. aberrant frenum/muscle position
6. gingival excess
- a. pseudo-pocket
- b. inconsistent gingival margin
- c. excessive gingival display
- d. gingival enlargement

7. abnormal color

Table 12: Classification of traumatic occlusal forces on the periodontium[29]

1. Occlusal trauma
A. Primary occlusal trauma
B. Secondary occlusal trauma
C. Orthodontic forces

Table 13: Proposed clinical and radiographic indicators of occlusal trauma

1. Fremitus	7. Thermal sensitivity
2. Mobility	8. Discomfort/pain on chewing
3. Occlusal discrepancies	9. Widened periodontal ligament space
4. Wear facets	10. Root resorption
5. Tooth migration	11. Cemental tear
6. Fractured tooth	

Table 14: Classification of factors related to teeth and to dental prostheses that can affect the periodontium

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis 1. Tooth anatomic factors 2. Root fractures 3. Cervical root resorption, cemental tears 4. Root proximity 5. Altered passive eruption
B. Localized dental prosthesis-related factors 1. Restoration margins placed within the supracrestal attached tissues 2. Clinical procedures related to the fabrication of indirectrestorations 3. Hypersensitivity/toxicity reactions to dental materials