Classification of diseases and conditions affecting the periodontium

Classification of diseases is necessary to separate conditions into different categories so aid in clinical and laboratory diagnosis and specific treatment. In light of this fact, a classification can be consistently defined by the differences in the clinical manifestations of diseases and conditions. **The current classification of periodontal diseases is based** on their **extent** (generalized versus localized), **severity** (slight, moderate, or severe), **rate of progression** (aggressive versus chronic), and **localization** (i.e., contained within the gingiva, as in gingivitis, or further involving periodontal bone loss, as in periodontitis).

In **1997** the American Academy of Periodontology responded to the need of developing of new classification of periodontal disease and formed a committee to plan and organize an international workshop to revise the classification system for periodontal diseases. On **1999**, the International Workshop for a Classification of Periodontal Diseases and Condition was held and a new classification was agreed upon **(Table 1).**

Table 1:- The classification of periodontal diseases and conditions in 1999.

1999 Classification of Periodontal Diseases and Conditions	
Gingival Diseases	Plaque-induced gingival diseases Non–plaque-induced gingival lesions
Chronic Periodontitis	Localized Generalized
Aggressive Periodontitis	Localized Generalized
Periodontitis as a Manifestation of Systemic Diseases	
Necrotizing Periodontal Diseases	Necrotizing ulcerative gingivitis (NUG) Necrotizing ulcerative periodontitis (NUP)
Abscesses of the Periodontium	Gingival abscess Periodontal abscess Pericoronal abscess
Periodontitis Associated With Endodontic Lesions	Endodontic–periodontal lesion Periodontal–endodontic lesion Combined lesion

Developmental or Acquired Deformities and Conditions	A- Localized tooth-related factors that predispose to plaque induced gingival diseases or periodontitis. B- Mucogingival deformities and conditions around teeth. C- Mucogingival deformities and conditions on edentulous ridges. D- Occlusal trauma.

The classification of periodontal diseases was studied again in **2017/ 2018**. In November 2017, the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP), discuss the reclassification of periodontal health, periodontal disease, and peri-implant disease **(Table 2).**

Table 2:-The classification of periodontal and peri-implant diseases and conditions in 2017.

2017 classification of periodontal and peri-implant diseases and conditions	
I. Periodontal health, gingival diseases, and conditions	**A-** Periodontal and gingival health. **B-** Gingivitis: dental biofilm induced. **C-** Gingival diseases: non- dental biofilm induced.
II. Periodontitis	**A-** Necrotizing periodontal diseases. **B-** Periodontitis. **C-** Periodontitis as a manifestation of systemic disease.
III. Other conditions affecting the periodontium	**A-** Systemic diseases or conditions affecting the periodontal supporting tissues. **B-** Periodontal abscesses and endodontic- periodontal lesions. **C-** Mucogingival deformities and conditions. **D-** Traumatic occlusal forces. **E-** Tooth and prosthesis- related factors.
IV. Peri-implant diseases and conditions	Peri-implant health. Peri-implant mucositis. Peri-implantitis. Peri-implant soft and hard tissue deficiencies.

The largest change in the reclassification is relating to the diagnosis of periodontitis. The new classification system requires the clinician to not only diagnose periodontitis and whether it is localized or generalized but to also comment on the **stage and grade** of the disease, to reflect on whether the disease is stable, in remission, or unstable, and finally to list the identified risk factors. The distribution of localized or generalized is still based on <30% of sites affected or >30% of sites affected. The **only risk factors** included in the World Workshop paper were diabetes, specifically **uncontrolled diabetes, and smoking**.

What Are the Major Changes in the New Classification System?

1. For the first time, the new classification system defines periodontal health and gingivitis for patient with:

- ☒ **Intact periodontium**
- ☒ **Reduced periodontium due to causes other than periodontitis**
- ☒ **Reduced periodontium due to periodontitis**

2. **"Chronic and aggressive periodontitis"** terminologies have been removed because there is very little evidence to support their existence as separate entities. **The exception is the classical localized juvenile (aggressive) periodontitis**.

Staging ➡ Classify the severity and extent of the disease.

Grading ➡ Indicate the rate of periodontitis progression, responsiveness to therapy, and potential impact on systemic health.

3. A major change has occurred in the classification of mucogingival deformities and conditions. For example, with regard to gingival recessions, the previous classification was more descriptive in nature and involved an assessment of a defect's relation to the mucogingival junction and radiographic assessment of interdental bone. The current classification is evidence-based and classifies recessions based on predictability of recession coverage using contemporary periodontal plastic surgery procedures.

4. A classification category for peri-implant diseases and conditions has been included for the first time in a periodontal classification system.

Periodontal health ,gingival diseases and conditions

The periodontal health 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. Based on

available methods to assess gingival health and inflammation, which can be simply, objectively accurately defined and graded using a **bleeding on probing score** (BOP%), assessed as the proportion of bleeding sites when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled **(0.25 N)** force to the apical end of the sulcus. **So gingival health can be classified into:**

1- Clinical gingival health on an intact periodontium: - is **characterized by** the absence of bleeding on probing, erythema and edema, patient symptoms, and no attachment and bone loss. **Physiological bone levels range** from 1.0 to 3.0 mm apical to the cemento-enamel junction.

2-Clinical gingival health on a reduced periodontium that include:

A-Stable periodontitis patient: Characterized by an absence (or minimum) bleeding on probing **(less than 10%),** in the presence of reduced clinical attachment and bone levels. while probing pocket depth **≤4 mm** provided that there is no pseudo pockets and no bleeding on probing at site with 4mm pocket depth. However, it should be recognized that successfully treated and stable periodontitis patients **remain at increased** risk of recurrent progression of periodontitis.

B-Non-periodontitis patient (e.g. gingival recession, crown lengthening)

While clinical gingival health on a reduced periodontium is characterized by an absence (or minimum) bleeding on probing **(less than 10%),** with possible presence of reduced clinical attachment and bone levels, with probing pocket depth **≤3 mm.** There is **no** evidence for increased risk of periodontitis.

The transition from periodontal health to gingivitis is **reversible** following treatment that resolves gingival inflammation. The transition to periodontitis results in attachment loss which is **irreversible**. Optimal periodontal therapy can restore gingival health on a reduced periodontium, or may result in mild marginal gingival inflammation at shallow probing pocket depths (≤ 3 mm). However, a history of periodontitis places patients at **high risk** of recurrent periodontitis and such patients require careful site-specific monitoring during periodontal maintenance programs **(Fig.1).**

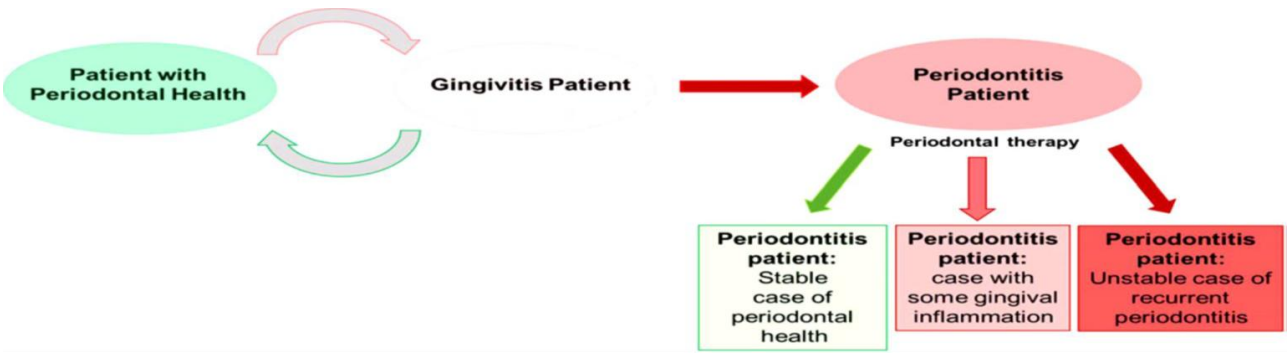

Fig. 1:-The transition from periodontal health to gingivitis and periodontitis.

Gingival diseases

There are broadly two categories of gingival disease:

- ☒ Dental plaque biofilm-induced gingivitis
- ☒ Non–dental plaque-induced gingival diseases

Depending on whether dental plaque biofilm-induced gingival inflammation occurs on an intact or reduced periodontium, or in a patient diagnosed with periodontitis, gingivitis can be further classified as:

- ❖ Gingivitis on an intact periodontium.
- ❖ Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient.
- ❖ Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., recession, crown lengthening).

1- Gingivitis on an intact periodontium

Gingival inflammation associated with **BOP score** ≥10%, **probing pocket depth** ≤ 3mm assuming **no** pseudo pocket , **no** attachment loss and **no** radiographic bone loss.

2-**Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient (remission periodontitis)**

Gingival inflammation associated with **BOP score** ≥10%, **probing pocket depth** ≤ 4mm assuming **no** pseudo pocket, with **presence** of attachment loss and radiographic bone loss, the patient will be diagnosed as **remission periodontitis.**

3- **Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., recession, crown lengthening)**

A patient with a reduced periodontium but without a history of periodontitis (e.g. gingival recession, crown lengthening) and a **BOP score** ≥10% would be diagnosed as a "gingivitis on a reduced periodontium, **probing pocket depth** ≤ 3mm assuming **no pseudo pocket**, with **possible** presence of attachment loss and radiographic bone loss.

The Classification of dental plaque biofilm-induced gingivitis

Dental plaque biofilm-induced gingivitis is defined at the site level as "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. A patient diagnosed as gingivitis as follows: **localized gingivitis**, defined as a patient presenting with a BOP score ≥10% and ≤30%, or **generalized gingivitis**, defined as a patient presenting with a BOP score > 30%.

A. Associated with dental plaque biofilm alone

B. Mediated by systemic or local risk factors: -

i. Systemic risk factors (**modifying factors**)

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

ii. Local risk factors (**predisposing factors**)

(a) Dental plaque biofilm retention factors (e.g., prominent restoration margins), **(b)** Oral dryness.

C. Drug-influenced gingival enlargement

1. Systemic factors

a) Plaque-induced gingivitis exacerbated by sex steroid hormones

Puberty: It is pronounced inflammatory response of gingiva to dental plaque and hormones during the circumpubertal period (11-16) years.

Menstrual cycle: It is pronounced inflammatory response of the gingiva to plaque and hormones immediately prior to ovulation.

Pregnancy: It is pronounced inflammatory response of the gingiva to dental plaque and hormones usually occurring during the second and third trimesters. During pregnancy, the prevalence and severity of gingivitis has been reported to be elevated and frequently unrelated to the amount of plaque present, The features of pregnancy-associated gingivitis are similar to plaque-induced gingivitis, except the propensity to develop signs of gingival inflammation in the presence of a relatively small amount of plaque during pregnancy.

Pregnancy may also be associated with the formation of pregnancy-associated pyogenic granulomas. **pregnancy-tumor:** It is a localized, painless, protuberant, exophytic gingival mass that is attached by a sessile or pedunculated base from the gingival margin or more commonly from an interproximal space resulting from dental plaque and hormones during pregnancy.it is **more common** in the maxilla and may develop as early as the first trimesters, and may regress or completely disappear following parturition.

Oral contraceptive: - Pronounced inflammatory response of the gingiva to plaque and oral contraceptive. The features of gingivitis associated with oral contraceptive in premenopausal women are similar to plaque-induced gingivitis. The condition is reversible following discontinuation of the drug.

b. Hyperglycemia: It is inflammatory response of the gingiva to plaque aggravated by poorly controlled plasma glucose levels.

c. Leukemia: Pronounced inflammatory response of the gingiva to plaque resulting in increased bleeding and enlargement subsequent to leukemia. Gingival bleeding is a **common sign** in patients with leukemia and it is the initial oral sign and/or symptom in **17.7%** and **4.4%** of patients with acute and chronic leukemia respectively. Gingival enlargement initially begins at the interdental papilla followed by marginal and attached gingiva.

d. Smoking: is **one of the major** lifestyle/behavioral risk factors for periodontitis, but which also has profound effects upon the gingival tissues. Systemic circulatory uptake of components of cigarette smoke as well as local uptake are reported to induce microvascular vasoconstriction and fibrosis. This can mask clinical signs of gingivitis, such as bleeding on probing, despite a significant underlying pathological inflammatory cell infiltrate.

e. Gingival diseases modified by malnutrition: It is known that malnourished individuals have a compromised host defense system that may affect the susceptibility to infection. **Ascorbic acid-deficiency gingivitis**: Inflammatory response of the gingiva to plaque aggravated by chronically low ascorbic acid levels. **The classic clinical signs of scurvy** describe the gingiva as being bright red, swollen, ulcerated and susceptible to hemorrhage. It is common in certain population, with restricted diets (e.g. infants from low socio economic families and institutionalized elderly).

f. Pharmacological agents: - May include drugs that reduce salivary flow, drugs that impact endocrine function, and drugs that may induce gingival enlargement and pseudo-pocketing.

2. Oral factors enhancing plaque accumulation.

a) The local contributing factors such as **prominent subgingival restoration margins, orthodontic appliance**.

b) Hyposalivation. Oral dryness is a clinical condition often associated with symptoms of xerostomia. Oral dryness manifesting as a lack of salivary flow, availability, or changes in quality of saliva, leading to reduced cleansing of tooth surfaces is associated with reduced dental plaque biofilm removal and enhanced gingival inflammation. **Common causes** include medications that have anti-parasympathetic action, Sjögrens syndrome when the salivary acini are replaced by fibrosis following autoimmune destruction, and mouth breathing in people who may have enhanced gingival display and/or an incompetent lip seal.

3.Drug-influenced gingival enlargement: Gingival enlargement resulting in whole or in part of gingiva from systemic drug use. Drugs that may cause gingival overgrowth include anticonvulsant (e.g. phenytoin), immunosuppressant (e.g. cyclosporine A), and calcium channel blockers (e.g. nifedipine, verapamil). The common clinical characteristics of drug-influenced gingival enlargement include:-

1)Variation in interpatient and intrapatient pattern (genetic predisposition).

2) Predilection for anterior gingiva.

3) Higher prevalence in children and younger age group.

4) Onset within 3 months of use.

5) Change in the gingival contour leading to modification of gingival size.

6) Enlargement first observed at the interdental papilla.

7) Change in gingival color.

8) Increased gingival exudate.

9) Bleeding upon provocation.

10) Pronounced inflammatory response of gingiva in relation to the plaque present.

11) Reduction in dental plaque can limit the severity of the lesion

The diagnostic criteria for gingivitis

The clinical signs of inflammation are erythema, edema, pain (soreness), heat, and loss of function.

These may have marked clinically in gingivitis as:

A. Swelling, seen as loss of knife-edged gingival margin
B. Bleeding on gentle probing
C. Redness
D. Discomfort on gentle probing

The symptoms a patient may report include:

A. Bleeding gums (metallic/altered taste)
B. Pain (soreness)
C. Halitosis
D. Difficulty eating
E. Appearance (swollen red gums)
F. Reduced oral health–related quality of life

Notes: -

- ☒ **Radiographs cannot be used to diagnose gingivitis**
- ☒ **Localized gingivitis is defined as 10%-30% bleeding sites; generalized gingivitis is defined as > 30% bleeding sites.**

The classification of non–dental plaque biofilm -induced Gingival diseases

A. Genetic/developmental disorders

i. Hereditary gingival fibromatosis

B. Specific infections

i. Bacterial origin

(a) Neisseria gonorrhoeae (gonorrhea)

(b) Treponema pallidum (syphilis)

(c) Mycobacterium tuberculosis (tuberculosis)

(d) Streptococcal gingivitis

ii. Viral origin

(a) Coxsackie virus (hand-foot-and-mouth disease)

(b) Herpes simplex I & II (primary or recurrent)

(c) Varicella zoster (chicken pox & shingles)

(d) Human papilloma virus (squamous cell papilloma; verruca vulgaris)

iii. Fungal origin

(a) Candidosis

(b) Other mycoses, e.g., histoplasmosis, aspergillosis

C. Inflammatory and immune conditions

i. Hypersensitivity reactions

(a) Contact allergy

(b) Plasma cell gingivitis

(c) Erythema multiforme

ii. Autoimmune diseases of skin and mucous membranes

(a) Pemphigus vulgaris

(b) Pemphigoid

(c) Lichen planus

(d) Lupus erythematosus

Systemic lupus erythematosis

Discoid lupus erythematosis

iii. Granulomatous inflammatory lesions (orofacial granulomatoses)

(a) Crohn's disease

D. Reactive processes

i. Epulides

(a) Fibrous epulis

(b) Calcifying fibroblastic granuloma

(c) Vascular epulis (pyogenic granuloma)

(d) Peripheral giant cell granuloma

E. Neoplasms

i. Pre-malignancy
(a) Leukoplakia
(b) Erythroplakia
ii. Malignancy
(a) Squamous cell carcinoma
(b) Leukemic cell infiltration
(c) Lymphoma
Hodgkin
Non-Hodgkin

F. Endocrine, nutritional & metabolic diseases
i. Vitamin deficiencies
(a) Vitamin C deficiency (scurvy)

G. Traumatic lesions
i. Physical/mechanical trauma
(a) Frictional keratosis
(b) Mechanically induced gingival ulceration
ii. Chemical (toxic) burn
iii. Thermal burn

H. Gingival pigmentation
i. Melanoplakia
ii. Smoker's melanosis
iii. Drug-induced pigmentation **(antimalarials, minocycline)**
iv. Amalgam tattoo

The origin of gingival inflammation in this group is different from that of the routine plaque-associated gingivitis. It is **not caused** by plaque and usually does not disappear after plaque removal.

1. Genetic/developmental disorders:

Hereditary gingival fibromatosis is genetically derived fibrotic gingival enlargement.

2.Specific infection

A-Bacterial origin:

a. Neisseria gonorrhea-associated lesions

b. Treponema pallidum-associated lesions

c. Streptococcal species-associated lesions.

d. Others. These conditions induced by exogenous bacterial infection other than common component of dental plaque.

B-Viral origin: These are acute manifestations of viral infections of oral mucosa, characterized by redness and multiple vesicles that easily rupture to form painful ulcers affecting the gingiva. These infections may be accompanied by fever, malaise, and regional lymphadenopathy.

a) Herpes virus infections:

1) Primary herpetic gingivostomatitis

2) Recurrent oral herpes

b) Oral Epstein- Barr virus lesions

c) Varicella- Zoster infections

C-Fungal origin: These gingival manifestation of fungal infections are characterized by white, red, or ulcerative lesions associated with several predisposing conditions.

Candida species infections:

a) Generalized gingival candidiasis

b) Linear gingival erythema

c) Histoplasmosis

d) Others

In the generalized gingival candidiasis, the most common species that causes this condition is **candida albicans**. In otherwise healthy individuals, oral candidiasis rarely manifests in the gingiva, but in immunocompromized patients like HIV-seropositive. Infection show erythema of the marginal gingiva and this condition is called linear gingival erythema which characterized by linear erythematous band limited to free gingiva and not respond to plaque removal.

3. Inflammatory and immune conditions and lesions:

A. Hypersensitivity reactions

- Contact allergy
- Plasma cell gingivitis:
- Erythema multiforme

Are gingival manifestations of immediate or delayed hypersensitivity responses. The allergy that is occur called contact allergy and there is clinical manifestations on the oral mucosa after a period of 12-48 hours following contact with the allergen. The lesions that affect the gingiva resemble oral lichen planus or leukoplakia. They are reddish or whitish and sometimes ulcerated but these lesions resolve after removal of the material. **Reactions attributable** to: **a)** Tooth pastes/dentifrices **b)** Mouth rinses/mouthwashes **c)** Chewing gum additives **d)** Foods and additives. Dental restorative materials: **a)** Mercury **b)** Nickel **c)** Acrylic.

B. Autoimmune diseases of skin and mucous membranes: Mucocutaneous disorders: These oral manifestations of disorders of the skin and mucous membrane present as erosions, vesicles, bullae, ulcers, and desquamative lesions. The lesions may be erythematous, white, or striated in appearance

1)Lichen planus **2)**Pemphigoid **3)** Pemphigus vulgaris **4)**Drug-induced Erythema multiforme **5)** Lupus erythematosus **6)** Other

4. Reactive process: Epulis is a term often applied to exophytic processes originating from the gingiva. Usually there are no symptoms, although the reactive processes are thought to represent an exaggerated tissue response to limited local irritation or trauma, and they are classified according to their histology. True epulides include:

- ✓ **Fibrous epulis**
- ✓ **Calcifying fibroblastic granuloma**
- ✓ **Pyogenic granuloma (vascular epulis)**
- ✓ **Peripheral giant cell granuloma (or central**)

5. Neoplasm

- **a)** Premalignant lesions Leukoplakia ,Erythroplakia.
- **b)** Malignant Squamous cell carcinoma, Leukemia, Lymphoma.

6. Endocrine, nutritional, and metabolic disease:- Vitamin deficiencies (vit C deficiency).

7. Traumatic lesions: These are self-inflicted (factitious), accidental, or iatrogenic injuries. They may be present as localized gingival recession, abrasions, ulceration, and burns. The lesions may be edematous, erythematous, or white in appearance. Lesions may exhibit combinations of several of these clinical features.

a. Chemical injury b. Physical injury c.Thermal injury

A -Traumatic lesions induced by chemicals: These traumatic lesions can be caused by local application of certain chemicals such as aspirin, cocaine, pyrophosphates, detergents (e.g., sodium lauryl sulfate), smokeless tobacco, betel nut, and bleaching agents.

B -Traumatic lesions caused by physical injury:- These traumatic lesions may be accidental or result from inappropriate oral hygiene procedures, inadequate dental restorations, poorly designed dental appliances, and orthodontic bands and devices.

C -Thermal trauma may occur from burns to the oral mucosa involving the gingiva. Common causes are hot coffee, pizza and melted cheese, dental treatment involving improper Handling of hot impression material, hot wax --- etc.

8. Gingival pigmentation Smoker's melanosis, Amalgam tattoo