Lecture: **Dr: Raghad Fadhil**

ATTEMPTS AT CLASSIFICATION:

Classification of disease is necessary to try to separate conditions into distinct categories so as to aid clinical and laboratory diagnosis and specific treatment. The criteria for separating diseases in this way should ideally be based on etiology, histopathology and, where appropriate, genetics rather than age of onset and rates of disease progression. Over the last three decades there have been four major attempts to classify periodontal disease.

Major changes were made in the 1999 classification of periodontitis, which has been in use for the last 19 years. Periodontitis was reclassified as chronic, aggressive (localized and generalized), necrotizing and as a manifestation of systemic disease.

The workshop in 2017 agreed on a classification framework for periodontitis further characterized based on a multidimensional staging and grading system that could be adapted over time as new evidence emerges

Classification of periodontal diseases and conditions (2017):

1. **Periodontal health and gingival diseases and condtions**
    a. **Periodontal health and gingival health**
    b. **Dental biofilm induced gingivitis**
    c. **Non-dental biofilm induced gingival disease**
2. **Periodontitis**
    a. **Periodontitis**
    b. **Necrotizing periodontal diseases**
    c. **Periodontitis as a manifestation of systemic disease**
3. **Other conditions affecting the periodontium**
    a. **Periodontal abscess and endodontic periodontal lesions**
    b. **Mucogingival deformity and conditions**
    c. **Traumatic occlussal force**
    d. **Tooth and prosthetic related factors**

4. **Peri-implant disease and conditions**
    a. **Peri- implant health**
    b. **Peri-implant mucositis**
    c. **Peri-implantitis**
    d. **Peri-implant soft and hard tissues deficiency**

1. **Periodontal health and gingival diseases and conditions**

a. Periodontal health and gingival health: is defined by absence of clinically detectable inflammation. There is a biological level of immune surveillance that is consistent with clinical gingival health and homeostasis. Clinical gingival health can be restored following treatment of gingivitis and periodontitis. However, the treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis, and accordingly, must be closely monitored.

Based on available methods to assess gingival health and inflammation, which can be simply, objectively and 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 in to:

• Clinical gingival health on an intact periodontium

Clinical gingival health on an intact periodontium is characterized by the absence (or minimum) of bleeding on probing (less than 10%), absence of patient symptoms, and attachment and bone loss.

• Clinical gingival health on a reduced periodontium that include:

○ **Non-periodontitis patient (e.g. 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. In non-periodontitis patients, there is no current evidence for increased risk of periodontitis.

○ **Stable periodontitis patient:** the clinical gingival health in stable periodontitis patients is characterized by an absence (or minimum) bleeding on probing (less than 10%), *in the presence of interproximal clinical attachment loss*. while probing pocket depth ≤4 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.

A. **Dental 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 by reducing levels of dental plaque at and apical to the gingival margin". 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%. Depending on whether dental 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 associated with BOP score ≥10%, probing pocket depth ≤ 3mm assuming no pseudo pocket , no attachment loss and no radiographic bone loss.

• **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.

• **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 (Note that recurrent periodontitis cannot be ruled out in this case).

The classification of dental biofilm induced gingivitis:

A. Associated with bacterial dental biofilm only

B. Mediated by systemic or local risk factors

1. Systemic conditions

a) Sex steroid hormones 1) Puberty 2) Menstrual cycle 3) Pregnancy 4) Oral contraceptives

b) Hyperglycemia

c) Leukemia

d) Smoking

e) Malnutrition

2. Oral factors enhancing plaque accumulation

a) Prominent subgingival restoration margins

b) Hyposalivation

C. Drug-influenced gingival enlargements

There are common characteristics to all gingival diseases associated with Dental plaque induced gingival diseases:-

1. Gingivitis is a clinical diagnosis. While emerging technologies are starting to shed light on the microbiological, molecular, and pathophysiological characteristics of gingivitis, definitive knowledge is not sufficient to supersede current clinical parameters.

2. The clinical signs of inflammation are erythema, edema, pain (soreness), heat, and loss of function, these may manifest clinically in gingivitis as: a. Swelling, seen as loss of knife-edged gingival margin and blunting of papillae b. Bleeding on gentle probing c. Redness d. Discomfort on gentle probing

3. 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

4. Radiographs cannot be used to diagnose gingivitis.

5.Reversibility of the disease by removing the etiology .

1. Gingival disease associated with dental biofilm only:-

It is called plaque induced gingivitis and it is inflammation of the gingiva resulting from dental plaque only,

2. mediated by systemic or local risk factors:-

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 frank 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, except for the propensity to develop signs of gingival inflammation in the presence of relatively little plaque in women taking these hormones. 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 begin 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).

2. Oral factors enhancing plaque accumulation.

a) The local contributing factors can be defined as a local feature that may influence the presentation of the disease, 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 classification of Non- dental biofilm induced gingival lesions:

1 Genetic/developmental disorders Hereditary gingival fibromatosis **2 Specific infections** a. Bacterial origin Neisseria gonorrhoeae (gonorrhea) Treponema pallidum (syphilis) Mycobacterium tuberculosis (tuberculosis) Streptococcal gingivitis (strains of streptococcus) b. Viral origin Coxsackie virus (hand-foot-and-mouth disease) Herpes simplex 1/2 (primary or recurrent) Varicella-zoster virus (chicken pox or shingles affecting V nerve) c. Fungal Candidosis **3 Inflammatory and immune conditions and lesions** a. Hypersensitivity reactions Contact allergy Plasma cell gingivitis Erythema multiforme b.Autoimmune diseases of skin and mucous membranes Pemphigus vulgaris Pemphigoid Lichen planus Lupus erythematosus c.Granulomatous inflammatory conditions (orofacial granulomatosis) Crohn's disease Sarcoidosis	**4 Reactive processes** Epulides Fibrous epulis **5 Neoplasms** a. Premalignant Leukoplakia Erythroplakia b. Malignant Squamous cell carcinoma Leukemia Lymphoma **6 Endocrine, nutritional, and metabolic diseases** Vitamin deficiencies **7 Traumatic lesions** a. Physical/mechanical insults Toothbrushing-induced gingival ulceration Factitious injury (self-harm) b. Chemical (toxic) insults Etching Chlorhexidine Acetylsalicylic acid c. Thermal insults Burns of mucosa **8 Gingival pigmentation** Smoker's melanosis 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, The hereditary gingival fibromatosis is genetically derived fibrotic gingival enlargement.

2.Specific infection

- **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.

- **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 gingivostomatitisI
    2) Recurrent oral herpes
b) Oral Epstein- Barr virus lesions
c) Varicella- Zoster infections
d) Other

- **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 d) Other

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 diseases**
   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