Classification of periodontitis:

Periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic dental plaque biofilms. Periodontitis is characterized by progressive destruction of the tooth-supporting apparatus. Its primary features include the loss of periodontal tissue support manifest through clinical attachment loss (CAL) and radiographically assessed alveolar bone loss, presence of periodontal pocketing and gingival bleeding (Papapanou et al., 2018). If untreated, it may lead to tooth loss, although it is preventable and treatable in the majority of cases.

Recently, based on pathophysiology, three clearly different forms of periodontitis have been identified according to new classification system proposed by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) in 2017:

1. Periodontitis.

2. Periodontitis as a direct manifestation of systemic diseases

3. Necrotizing periodontitis

I. Periodontitis: was classified according to different form of staging and grading. Staging relies on the standard dimensions of severity and extent of periodontitis at presentation**. Periodontitis cases defined as (Tonetti et al., 2018) :(1) Interdental CAL is detectable at ≥2 non-adjacent teeth, or (2) Buccal or oral CAL ≥3 mm with pocketing >3mm is detectable at ≥ 2teeth**

The extent

The extent and distribution for each stage described as molar/ incisor pattern or localized if the involved sites < 30% or generalized if the involved site ≥30%

Staging:

Stage I periodontitis: Stage I periodontitis is the borderland between gingivitis and periodontitis and represents the early stages of attachment loss, the severity of this stage associated with clinical attachment loss about 1-2mm and radiographic bone loss less than 15% in the coronal third.

Stage II periodontitis: represents established periodontitis. The severity of this stage associated with clinical attachment loss about 3-4mm and radiographic bone loss more than 15% and less than 33%in the coronal third.

Stage III periodontitis: This stage associated with clinical attachment loss ≥5mm and radiographic bone loss extending to the middle third of the roots, which mean the percentage of bone loss more than 33% and less than 66%.

Advanced stage IV periodontitis: This stage is characterized by the presence of deep periodontal lesions that extend to the apical portion of the root. This stage associated with clinical attachment loss ≥5mm and radiographic bone loss extending to apical third of the roots in which bone loss more than 66%.

Grades of periodontitis:

Irrespective of the stage at diagnosis, periodontitis may progress with different rates in individuals, may respond less predictably to treatment in some patients, and may or may not influence general health or systemic disease. **Grading or rate of progression is assigned by dividing the maximum amount of radiographic bone loss in percentage terms on patient's age in years**

Grade A periodontitis: if the result was less than 0.5 so the grade is A (for example, bone loss 25% in a 60-year-old or less than 30% in an 80-year-old)

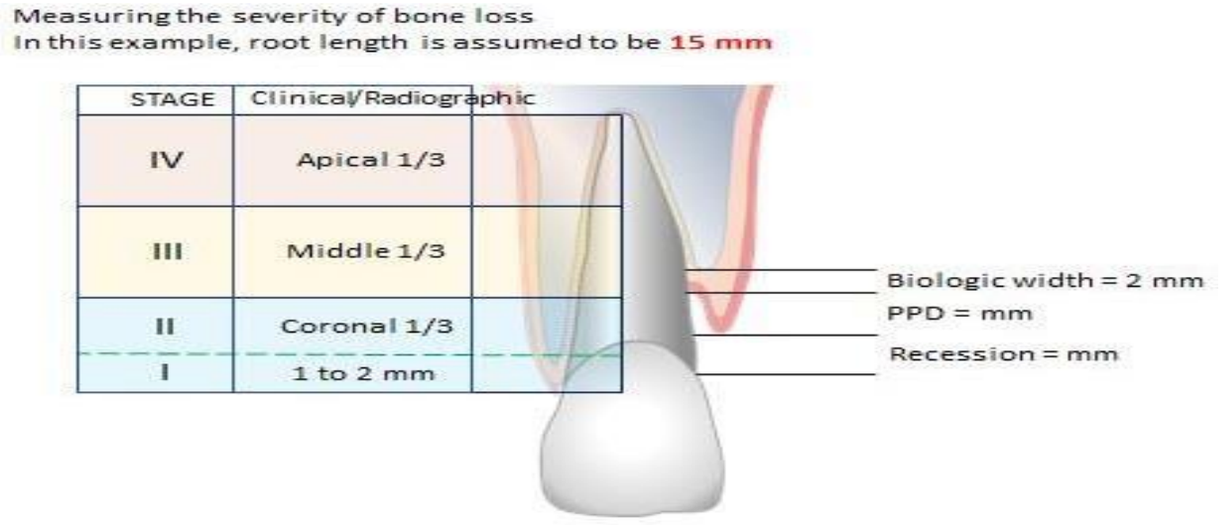
Grade C periodontitis: if the result was more than 1 (for example, bone loss more than 30% in a 28-year-old or more than 50% in a 49-year-old)

Grade B periodontitis: all other situations (the result between 0.5-1)

Important note: clinically the radiographic bone loss in percentage can be calculated (in case there is no radiograph) if we have clinical attachment loss by measuring (recession + pocket depth) using the worse tooth, adding the biological width (~2 mm) to give an estimate of the distance that the bone crest is from the CEJ, If an average root length assumed 15 mm or calculated by doubling the crown length, the percentage of bone loss calculated by using the following formula

Percentage of bone loss = (clinical attachment loss +2) *100/ root length (assumed 15mm) the result can be us ed to estimate percentage bone loss for grading.

. A summary of how this is calculated can be seen



Status 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 if 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.

○ **Remission periodontitis (Gingival inflammation on a successfully treated periodontitis patient)**

Gingival inflammation associated with BOP score ≥10%, probing pocket depth ≤ 4mm assuming no pseudo pocket, no BOP at 4mm 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).

○**Unstable periodontitis:** Probing pocket depth equal or more than 5 mm or BOP at 4mm pocket

Risk factors:

1. **Diabetes mellitus**
2. **Smoking**
3. **Family history**

So, the final diagnostic statement for periodontitis should include the followingterms

Extent, Periodontitis, Stage, Grade, Status, Risk factor

II. Periodontitis as a manifestation of systemic diseases :

A variety of systemic diseases and conditions can affect the course of periodontitis or have a negative impact on the periodontal attachment apparatus.

1. Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation

I. Genetic disorders

a. Diseases associated with immunologic disorders: like Down syndrome, Leukocyte adhesion deficiency syndromes, Papillon-Lefèvre syndrome

b. Diseases affecting the oral mucosa and gingival tissue: like Plasminogen deficiency

c. Diseases affecting the connective tissues: Ehlers-Danlos syndromes (types IV, VIII)

d. Metabolic and endocrine disorders: like Glycogen storage disease and Hypophosphatasia

II. Acquired immunodeficiency diseases: including Acquired neutropenia and HIV infection

III. Inflammatory diseases: Inflammatory bowel disease

2. Other systemic disorders that influence the pathogenesis of periodontal diseases

Diabetes mellitus, Obesity, Osteoporosis, Arthritis (rheumatoid arthritis, osteoarthritis), Emotional stress and depression, Smoking (nicotine dependence)

3. Systemic disorders that can result in loss of periodontal tissues independent of periodontitis

a. Neoplasms:

-Primary neoplastic diseases of the periodontal tissues like Oral squamous cell carcinoma

-Secondary metastatic neoplasms of the periodontal tissues

b. Other disorders that may affect the periodontal tissues like Langerhans cell histiocytosis, Giant cell granulomas Hyperparathyroidism

III. Necrotizing periodontal diseases:

A. Necrotizing ulcerative gingivitis

B. Necrotizing ulcerative periodontitis

C. Necrotizing stomatitis

Necrotizing ulcerative gingivitis: This is an infection characterized by gingival necrosis presenting as 'punched-out' papillae, with gingival bleeding, and pain. Fetid breath and pseudomembrane formation may be secondary diagnostic features. *Fusiform bacteria*, *prevotella intermedia*, and *spirochetes* have been associated with gingival lesions. Predisposing factors may include emotional stress, poor diet, cigarette smoking, and HIV infection.

Necrotizing ulcerative periodontitis: This is an infection characterized by necrosis of gingival tissues, periodontal ligament, and alveolar bone. These lesions are most observed in individuals with systemic conditions including HIV infection, severe malnutrition, and immunosuppression.

Necrotizing stomatitis: is a very severe and aggressive form of necrotizing periodontal disease showing extensive oral cavity tissue and bone destruction.

In necrotizing stomatitis, after the oral mucosal membranes are destroyed, the entire mouth is involved due to spread of deep infection.

Other condition affecting the periodontium:

a. Periodontal abscess and endodontic periodontal lesion

I. **PERIODONTAL ABSCESSES** (PA): PA can specifically be defined as a localized accumulation of pus located within the gingival wall of the periodontal pocket, with an expressed periodontal breakdown occurring during a limited period, and with easily detectable clinical symptoms.

Periodontal abscess can be classified as:

1. **Periodontal abscess in periodontitis patients:**

In periodontitis patients, a PA could represent a period of disease exacerbation, favored by the existence of tortuous pockets, presence of furcation involvement or a vertical defect, in which the marginal closure of the pocket could lead to an extension of the infection into the surrounding periodontal tissues. In addition, changes in the composition of the subgingival microbiota, with an increase in bacterial virulence, or a decrease in the host defense, could also result in an inefficient capacity to drain the increased suppuration.

Different subgroups could be distinguished:

a. Acute exacerbation:

○ In untreated periodontitis. ○ In “refractory” periodontitis. ○ In maintenance phase.

b. After different treatments:

○ Scaling and root planning or professional prophylaxis: dislodged calculusfragments could be pushed into the tissues, or inadequate scaling could allow

calculus to remain in deep pocket areas, whereas the coronal part would occlude the normal drainage. ○ Surgical periodontal therapy: associated with the presence of foreign bodies such as membranes for regeneration or sutures. ○ Systemic antimicrobial intake, without subgingival debridement, in patients with severe periodontitis could also cause abscess formation, probably related to an overgrowth of opportunistic bacteria.

○ Use of other drugs: e.g., nifedipine.

2. Periodontal abscess in non- periodontitis patients PA can also occur in previously healthy sites, because of:

• Impaction of foreign bodies: dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls.

• Harmful habits (biting wire, nail biting, clenching) could favor abscess formation because of subgingival impaction of foreign bodies or to coronal closure of the pocket

• Orthodontic factors, such as inadequate orthodontic forces or a cross-bite, have been reported to favor PA development.

• Gingival enlargement.

• Alterations of the root surface, including: ○ Severe anatomic alterations, such as invaginated tooth, dens evaginatus (grooves) or odontodysplasia. ○ Minor anatomic alterations, such as cemental tears, enamel pearls or developmental grooves. ○ Iatrogenic conditions, such as perforations. ○ Severe root damage: vertical root fracture or cracked tooth syndrome extending through the root. ○ External root resorption.

II. ENDO-PERIODONTAL LESIONS (EPL)

Clinical presentation

EPL are clinical conditions involving both the pulp and periodontal tissues and may occur in acute or chronic forms. When they are associated with a recent traumatic or iatrogenic event (e.g. root fracture or perforation), the most common manifestation is an abscess accompanied by pain. However, EPL, in subjects with periodontitis, normally present slow and chronic progression without evident symptoms. The most common signs and symptoms associated with a tooth affected by an EPL are deep periodontal pockets reaching or close to the apex and negative or altered response to pulp vitality tests. The other signs and symptoms reported, in order of prevalence, are 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

The main and obvious routes of communication are

1.the apical foramina 2. Lateral canals 3. Dentinal tubules

TABLE 3 Classification of endo-periodontal lesions

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

Etiology and risk factors

Primary etiology and established EPL is always associated with varying degrees of microbial contamination of the dental pulp and the supporting periodontal tissues. Nonetheless, the primary etiology of these lesions might be associated with

A. endodontic and/or periodontal infections

Endo-periodontal lesions associated with endodontic and periodontal infections They might be triggered:

(1) by a carious lesion that affects the pulp and, secondarily, affects the periodontium.

(2) by periodontal destruction that secondarily affects the root canal.

(3) or by both events concomitantly. The latter type occurs less frequently and is usually referred to as a “true-combined” or “combined” lesion. These lesions may develop in subjects with periodontal health or disease.

(B) trauma and/or iatrogenic factors.

These conditions usually have a poor prognosis as they affect the tooth structure. The most common lesions in this category were:

(1) root/pulp chamber/furcation perforation (e.g. because of root canal instrumentation or to tooth preparation for post retained restorations

(2) external root resorption (e.g., because of trauma)

(3) pulp necrosis (e.g., because of trauma) draining through the periodontium

b. Mucogingival deformities or conditions around teeth:

1. gingival 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
    d. Gingival thickness
    e. Gingival width
3. Lack of keratinized gingiva
4. Decreased vestibular depth
5. Aberrant frenum/muscle position
6. Gingival excess
    a. Pseudopocket
    b. Inconsistent gingival margin
    c. Excessive gingival display
    d. Gingival enlargement
7. Abnormal color

c. Tooth and prosthetic related factors:

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 indirect restorations

3. Hypersensitivity/toxicity reactions to dental materials.

Several conditions exist in teeth that may predispose the periodontium to disease. In certain cases, these factors may contribute to the initiation of periodontal disease. While the etiology of periodontal disease is bacterial, factors that enhance bacterial accumulation or allow ingress of bacteria into the periodontium should be considered in the classification of periodontal diseases.

d.Traumatic occlusal force

- **Occlusal trauma :**

  Periodontal tissue injury due to occlusal force.
- **Primary occlusal trauma:**

  Trauma occurred in intact periodontal tissue due to excessive occlusal load.
- **Secondary occlusal trauma:**

  Trauma occurred in reduced periodontal tissue due to excessive or normal occlusal load.

e. Peri-implant diseases and conditions

- **1. peri-implant health**
  Absence of erythema , swelling and bleeding.
- **2. peri-implant mucositis .**
  Signs of inflammation and no bone loss beyond remodeling level.
- **3. peri-implantitis .**
  Signs of inflammation and bone loss.
  4. Peri implant soft tissues and hard tissues deficiency