A new classification for periodontal and peri-implant diseases and conditions

Presented by:

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The consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.

Figure:- Participants of Workgroup 1

CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017

Periodontal Diseases and Conditions

Periodontal Health, Gingival Diseases and Conditions

Chapple, Mealey, et al. 2018 Consensus Rept link

Trombelli et al. 2018 Case Definitions link

- Periodontal Health and Gingival Health
- Gingivitis: Dental Biofilm-Induced
- Gingival Diseases: Non-Dental Biofilm-Induced

Periodontitis

Papapanou, Sanz et al. 2018 Consensus Rept link

Jepsen, Caton et al. 2018 Consensus Rept link

Tonetti, Greenwell, Kornman. 2018 Case Definitions link

- Necrotizing Periodontal Diseases
- Periodontitis
- Periodontitis as a Manifestation of Systemic Disease

Other Conditions Affecting the Periodontium

Jepsen, Caton et al. 2018 Consensus Rept link

Papapanou, Sanz et al. 2018 Consensus Rept link

- 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

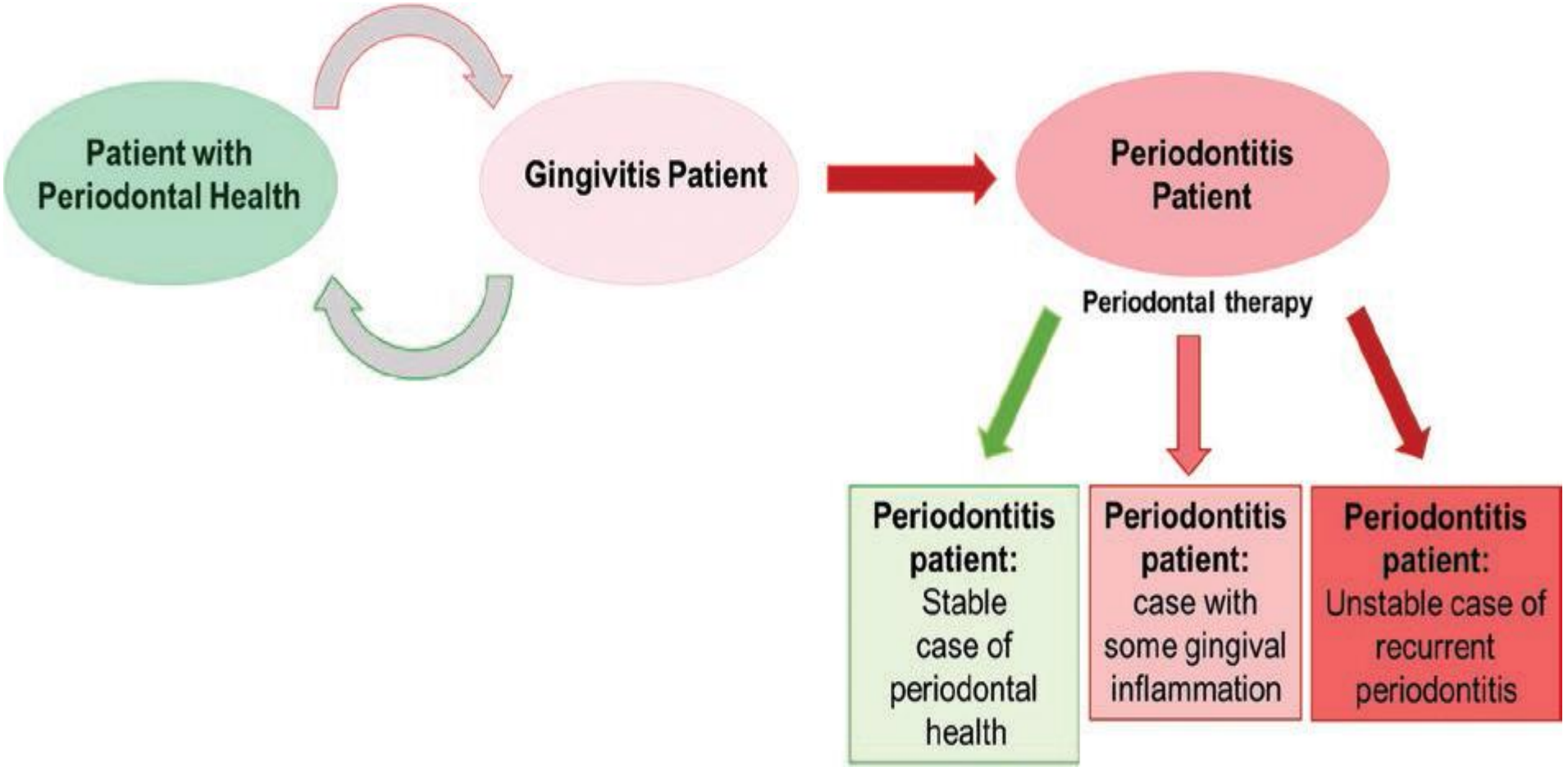
Peri-Implant Diseases and Conditions

Berglundh, Armitage et al. 2018 Consensus Rept link

- Peri-Implant Health
- Peri-Implant Mucositis
- Peri-Implantitis
- Peri-Implant Soft and Hard Tissue Deficiencies

Periodontal health

Periodontal 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.



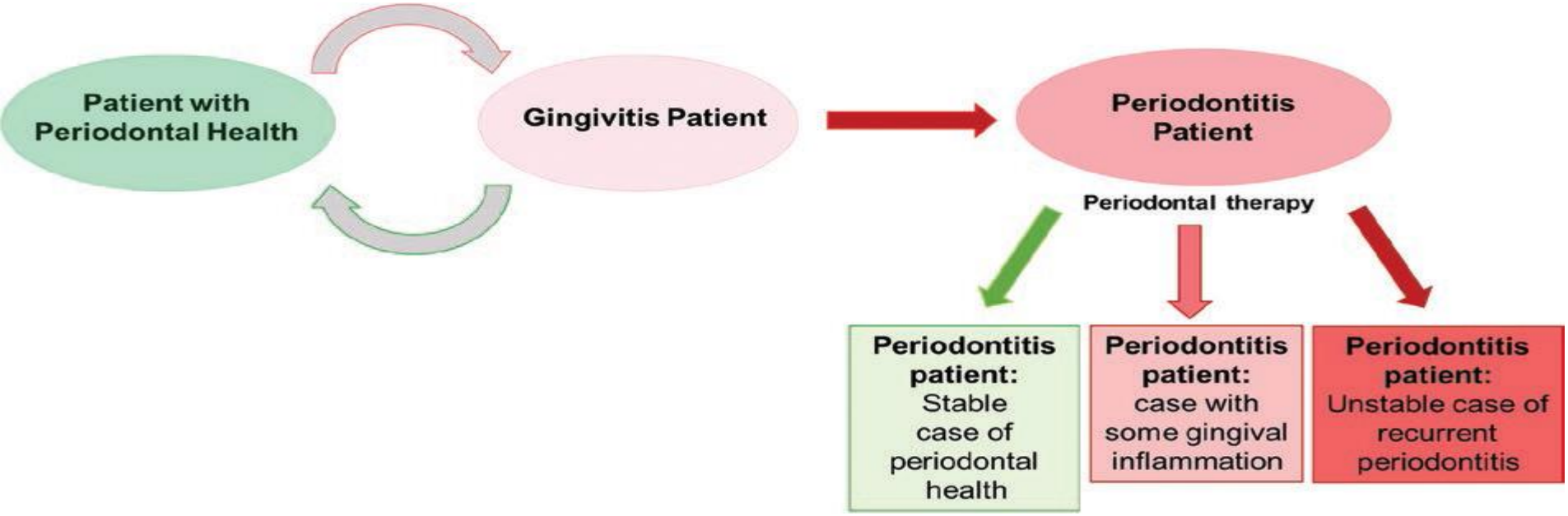
Periodontal health

What is the biology of clinical gingival health?

Clinical gingival health is generally associated with an inflammatory infiltrate and a host response consistent with homeostasis.

How do we classify clinical gingival health?

- **Clinical gingival health on an intact periodontium**
- **Clinical gingival health on a reduced periodontium**
  - **Stable periodontitis patient**
  - **Non-periodontitis patient (e.g. recession, crown lengthening)**



Note:-"intact periodontium" within this consensus, an absence of detectable attachment and/or bone loss is implicit.

PERIODONTAL HE ALTH

clinical features of gingival health

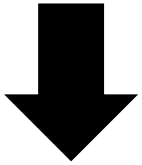
Clinical features of gingival health on an intact periodontium:-

Clinical gingival health on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss.

Physiological bone levels range from 1.0 to 3.0 mm apical to the cemento-enamel junction.

Clinical features of gingival health on a reduced periodontium:-

Clinical gingival health on a reduced periodontium is characterized by an absence of bleeding on probing, erythema, edema and patient symptoms in the presence of reduced clinical attachment and bone levels.



However, it should be recognized that successfully treated and stable periodontitis patients remain at increased risk of recurrent progression of periodontitis.

In non-periodontitis patients,there is no current evidence for increased risk of periodontitis.

Assessment of gingival inflammation

Based on available methods to assess gingival inflammation, a gingivitis case can be simply, objectively and accurately defined and graded using :-

bleeding on probing score (BOP%),

assessed as the proportion of bleeding sites(dichotomous yes/no evaluation) when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled (~0.25 N) force to the apical end of the sulcus at six sites (mesio-buccal, buccal, disto-buccal, mesiolingual,lingual, disto-lingual) on all teeth present.

------Limitations of these clinical criteria arise from a lack of standardized periodontal probes (e.g. probe dimensions, taper), examiner variability (probe pressure, angle), patient related factors (biotype, medications,etc.) and smoking.

Periodontal stability is characterized by successful treatment through control of local and systemic risk factors, resulting in minimal (< 10% of sites4) BOP, no probing depths of 4 mm or greater that bleed on probing, optimal improvement in other clinical parameters and lack of progressive periodontal destruction.

<u>The treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis and accordingly must be closely monitored.</u>

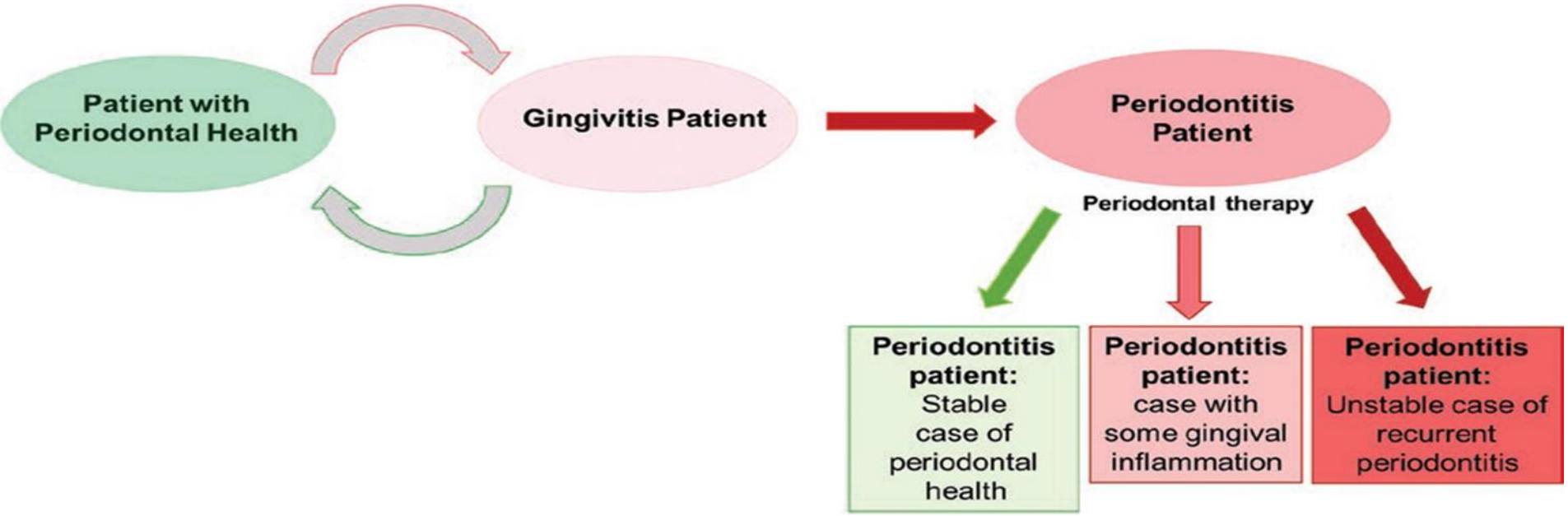


Figure summarizes the various scenarios that may arise following the transition from health,to gingivitis and ultimately periodontitis.

Categories of gingival disease:-

There are broadly two categories of gingival disease:

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

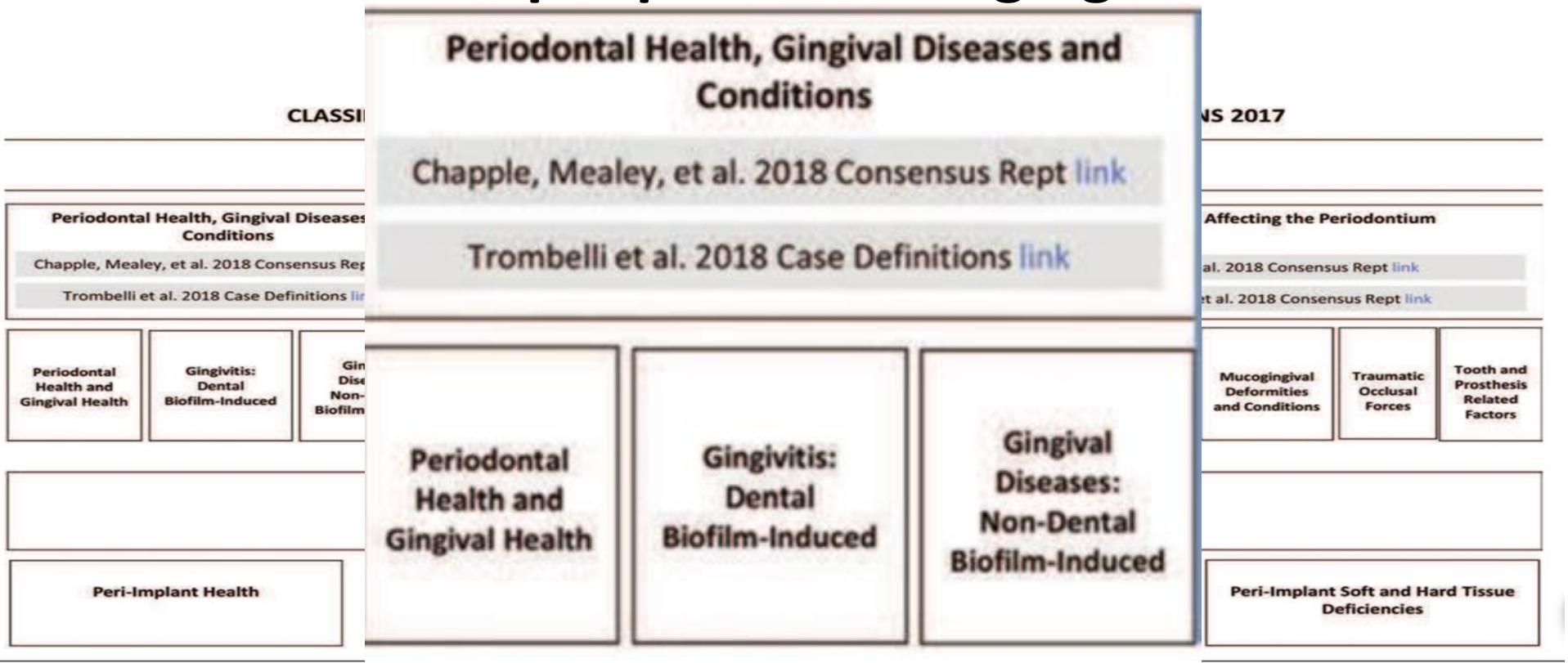


TABLE 1 Diagnostic look-up table for gingival health or dental plaque-induced gingivitis in clinical practice

Intact periodontium	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets)[a]	≤3 mm	≤3 mm
Bleeding on probing[a]	<10%	Yes (≥ 10%)
Radiological bone loss	No	No
Reduced periodontium Non-periodontitis patient	Health	Gingivitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets)[a]	≤3 mm	≤3 mm
Bleeding on probing[a]	<10%	Yes (≥ 10%)
Radiological bone loss	Possible	Possible

NB: In conditions where there is treatment but not cure, e.g. rheumatoid arthritis, periodontitis, the post-treatment parameters that define stability/health or gingivitis may differ from the parameters for health/gingivitis in a non-periodontitis patient. The threshold for "clinical health" in a treated and stable periodontitis patient is therefore set at ≤ 4 mm.

Successfully treated stable periodontitis patient	Health	Gingivitis in a patient with a history of periodontitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets)[a]	≤4 mm (no site ≥ 4 mm with BOP)[b]	≤3 mm
Bleeding on probing[a]	<10%	Yes (≥ 10%)
Radiological bone loss	Yes	Yes

NB: A successfully treated periodontitis patient in whom sites of gingival bleeding appear remains at high risk of disease recurrence at those sites and of progressive attachment loss. Therefore, gingivitis is defined as bleeding at a shallow site of ≤ 3 mm rather than ≤ 4 mm, as is the case in gingival heath. Where the probing depth is 4 mm or higher with bleeding, this is no longer a "closed pocket."[21,17]

[a]Assumes a light probing pressure of 0.2 to 0.25 N.

development of gingivitis, its severity and extent

The threshold of plaque accumulation necessary to induce gingival inflammation and impact upon its rate of progression at specific sites or at a whole mouth level varies between individuals according to both :-

1- local risk factors, known as predisposing factors,

2- systemic risk factors, referred to as modifying factors

development of gingivitis, its severity and extent

1. Local risk factors (predisposing factors)

Local risk factors for gingivitis are those that encourage plaque accumulation at a specific site by either inhibiting its removal during daily oral hygiene practices, and/or creating a biological niche that encourages increased plaque accumulation. These include:

a. **Dental plaque biofilm retention factors (including certain tooth anatomical factors) – facilitate plaque accumulation at and apical to the gingival margin, enabling biofilm adherence and maturation and increasing the difficulty of mechanical plaque removal. Several clinical studies providing a moderate level of evidence have demonstrated that subgingival restoration margins may be detrimental to gingival health.**

b. 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.

2. Systemic risk factors (modifying factors)

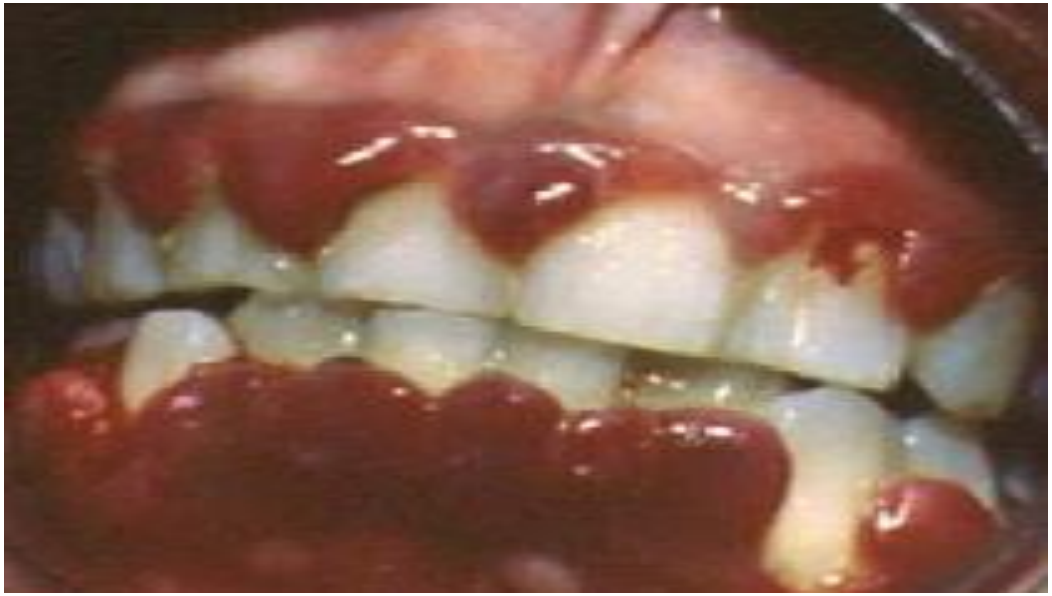
Systemic risk or modifying factors are those characteristics present in an individual, which negatively influence the immune-inflammatory response to a given dental plaque biofilm burden,resulting in exaggerated or "hyper" inflammation. Examples include:

a. 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.

b. Metabolic factors – hyperglycemia in people with or without diabetes. Excess glucose is toxic and directly induces mitochondrial stress and an enhanced respiratory burst in inflammatory cells that may activate various proinflammatory mediator cascades.

2. Systemic risk factors (modifying factors)

c. Nutritional factors – Severe Vitamin C deficiency, or scurvy,results in compromised antioxidant micronutrient defenses to oxidative stress and also negatively impacts collagen synthesis,resulting in weakened capillary blood vessel walls and a consequent propensity to enhanced gingival bleeding.



d. Pharmacological agents (prescription, non-prescription, and recreational agents) – can act via diverse mechanisms to increase susceptibility to gingivitis. This may include drugs that reduce salivary flow, drugs that impact endocrine function , and drugs that may induce gingival enlargement and pseudo-pocketing.

2. Systemic risk factors (modifying factors)

e. Elevations in sex steroid hormones – at :

a- puberty,

b- during pregnancy,or

c- following medication with first generation oral contraceptives

may modify the gingival inflammatory response.

Complex biological reactions within the gingival tissues result from such elevated sex steroid levels and generate more than expected inflammation, in response to relatively small levels of plaque.

However, modern oral contraceptive dosages have been reduced and there is little evidence for exaggerated gingival inflammatory responses to plaque with such drugs.

f. Hematological conditions – particular blood malignancies such as leukemia or pre-malignant conditions such as myelodysplasia are associated with signs of excess gingival inflammation in the absence of excessive plaque biofilm accumulation.

Signs include swollen, purple or occasionally pale gingiva due to leukemic cell infiltration, gingival bleeding that is inconsistent with levels of dental plaque biofilm accumulation, due to thrombocytopenia and/or clotting-factor deficiencies.

Classification of dental plaque biofilm-induced gingivitis

There is utility in defining the severity of gingivitis as a patient communication tool, but there are no objective clinical criteria for defining severity.Thus, in this context alone, the extent of gingivitis can be used to communicate "mild, moderate, and severe" gingivitis. Moreover,emerging evidence suggests that the contained gingivitis lesion may have systemic inflammatory consequences.

There is no robust evidence to clearly differentiate mild, moderate,and severe gingivitis, and definitions remain a matter of professional opinion. Methods of defining gingivitis may include:

Defining percentages (e.g. mild = < 10%, moderate = 10%-30%, severe = > 30% sites)
Grading (e.g. grade 1 to 5 in 20% quintiles for % sites bleeding on probing).

How do we define a case of dental plaque-induced gingivitis on an intact and a reduced periodontium for epidemiological purposes?

For epidemiological purposes, gingivitis on an intact periodontium and gingivitis on a reduced periodontium in a patient without a history of periodontitis, is defined as ≥10% bleeding sites with probing depths ≤3 mm.

Localized gingivitis is defined as 10%-30% bleeding sites;

generalized gingivitis is defined as > 30% bleeding sites.

For epidemiological purposes alone, a periodontitis case cannot simultaneously be defined as a gingivitis case. Therefore, a patient with a history of periodontitis, with gingival inflammation is still a periodontitis case.

TABLE 2 Classification of gingival health and gingival diseases/ conditions

1. **Periodontal health**[2]
   - **A.** Clinical health on an intact periodontium
   - **B.** Clinical gingival health on a reduced periodontium
     - (i) Stable periodontitis patient
     - (ii) Non–periodontitis patient
2. **Gingivitis – dental plaque-induced:** ***intact periodontium; reduced periodontium in non-periodontitis patient; reduced periodontium in successfully treated periodontitis patient.***[7]
   - **A.** Associated with biofilm alone
   - **B.** Mediated by systemic or local risk factors
     - **i.** Systemic risk factors (modifying factors)
       - (a) Smoking
       - (b) Hyperglycemia
       - (c) Nutritional factors
       - (d) Pharmacological agents (prescription, non–prescription and recreational)
       - (e) Sex steroid hormones

         Puberty
         Menstrual cycle
         Pregnancy
         Oral contraceptives
       - (f) Hematological conditions
     - **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

3. Gingival diseases – non–dental plaque-induced[26]

A. Genetic/developmental disorders

i. Hereditary gingival fibromatosis[a]

B. Specific infections

i. Bacterial origin

(a) *Neisseria gonorrhoeae*[a]

(b) *Treponema pallidum*[a]

(c) *Mycobacterium tuberculosis*[a]

(d) Streptococcal gingivitis

ii. Viral origin

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

(b) Herpes simplex I & II (primary or recurrent)[a]

(c) Varicella zoster (chicken pox & shingles – V nerve)[a]

(d) Molluscum contagiosum[a]

(e) Human papilloma virus (squamous cell papilloma; condyloma acuminatum; verruca vulgaris; focal epithelial hyperplasia)

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iii. Fungal origin

(a) Candidosis

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

C. Inflammatory and immune conditions

i. Hypersensitivity reactions

(a) Contact allergy[a]

(b) Plasma cell gingivitis[a]

(c) Erythema multiforme[a]

ii. Autoimmune diseases of skin and mucous membranes

(a) Pemphigus vulgaris[a]

(b) Pemphigoid[a]

(c) Lichen planus[a]

(d) Lupus erythematosus[a]

Systemic lupus erythematosis
Discoid lupus erythematosis

iii. Granulomatous inflammatory lesions (orofacial granulomatoses)

(a) Crohn's disease[a]

(b) Sarcoidosis[a]

D. Reactive processes

i. Epulides

(a) Fibrous epulis

(b) Calcifying fibroblastic granuloma

(c) Vascular epulis (pyogenic granuloma)

(d) Peripheral giant cell granuloma[a]

E. Neoplasms

- **i.** Premalignancy
  - (a) Leukoplakia
  - (b) Erythroplakia
- **ii.** Malignancy
  - (a) Squamous cell carcinoma[a]
  - (b) Leukemic cell infiltration[a]
  - (c) Lymphoma[a]

    Hodgkin
    Non-Hodgkin

F. Endocrine, nutritional & metabolic diseases

- **i.** Vitamin deficiencies[a]
  - (a) Vitamin C deficiency (scurvy)

G. Traumatic lesions

- **i.** Physical/mechanical trauma
  - (a) Frictional keratosis
  - (b) Mechanically induced gingival ulceration
  - (c) Factitious injury (self-harm)
- **ii.** Chemical (toxic) burn
- **iii.** Thermal insults
  - (a) Burns to gingiva

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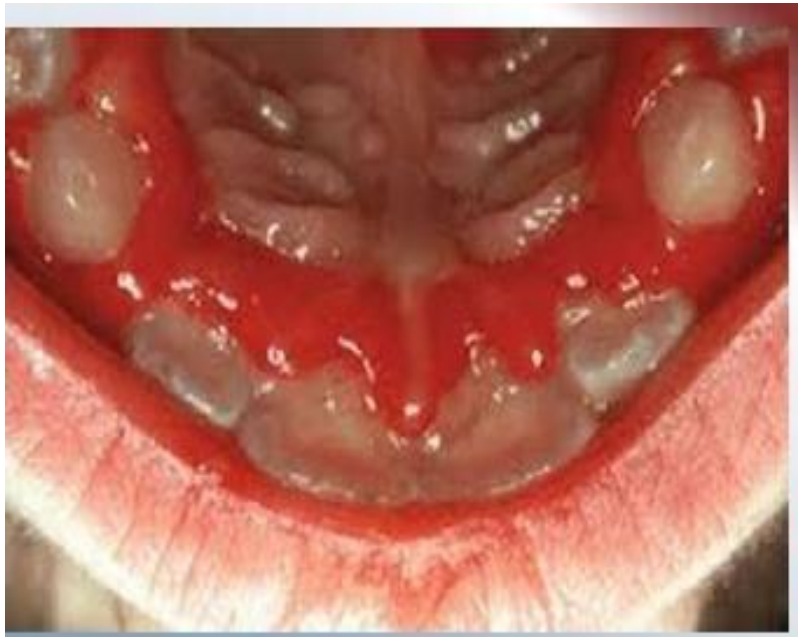
H. Gingival pigmentation

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

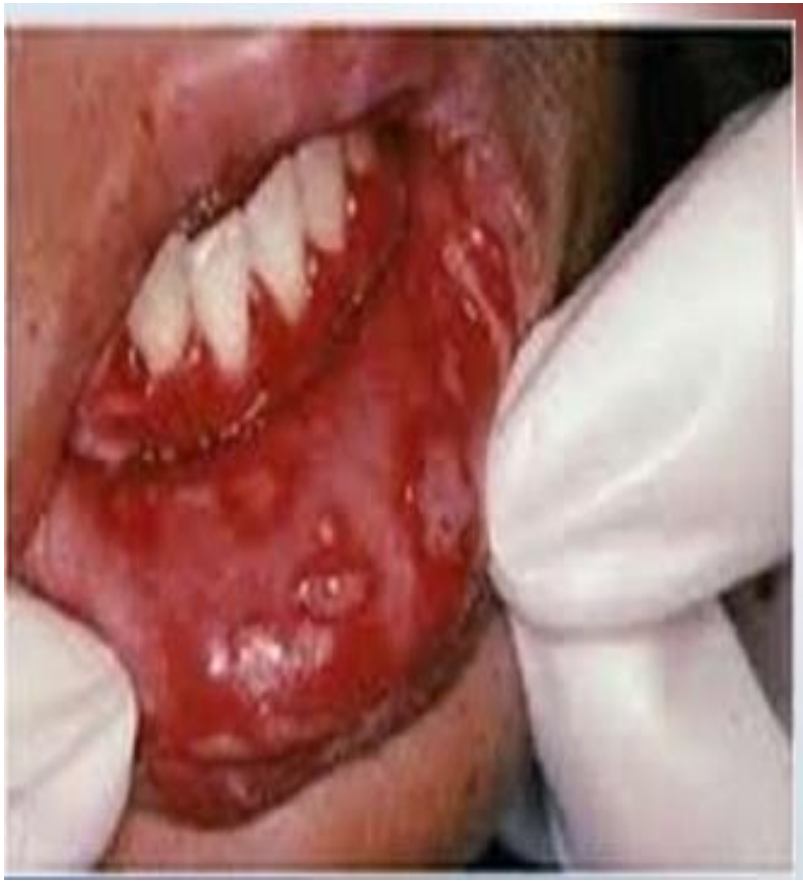
Non–Plaque-Induced Gingival Lesions

Gingival Diseases of Specific Bacterial Origin



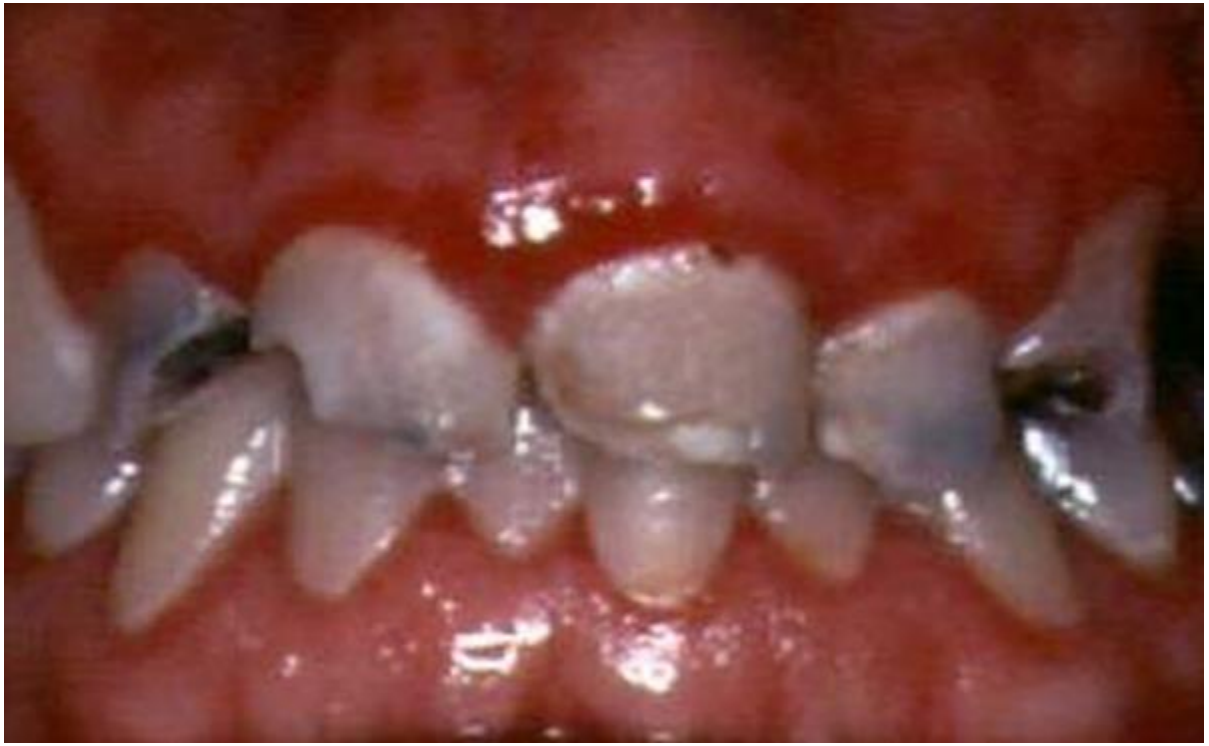
Streptococcal gingivitis or gingivostomatitis is a rare condition that may present as an acute condition with fever, malaise, and pain associated with acutely inflamed, diffuse, red, and swollen gingiva with increased bleeding and occasional gingival abscess formation.

Gingival Diseases of Viral Origin

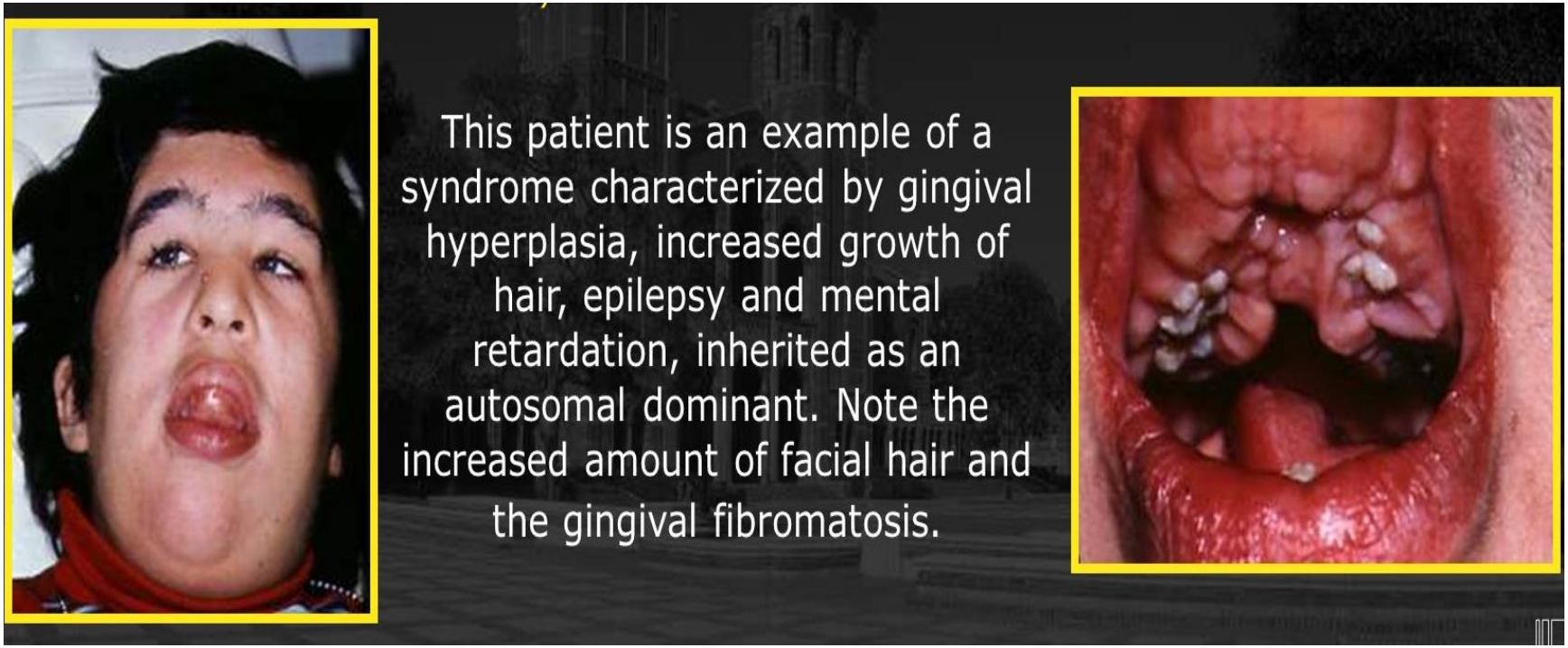


the most common being the herpes viruses-Primary herpetic gingivostomatitis Clinically appear as: Multiple tiny vesicles that progress to form painful ulcers. Painful erythematous swollen gingival. Fever, malaise, cervical lymphadenopathy.

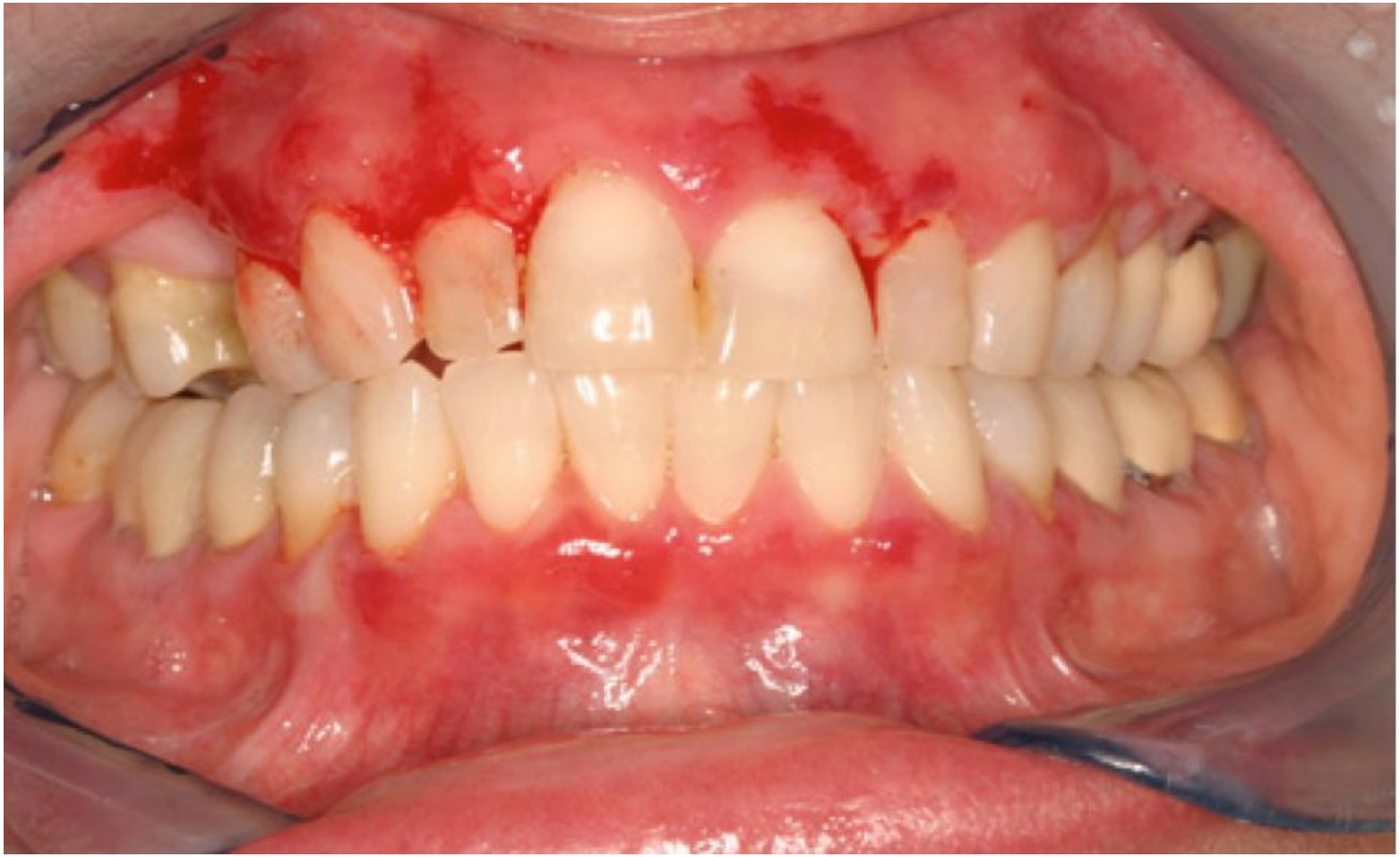
Gingival Diseases of Fungal Origin



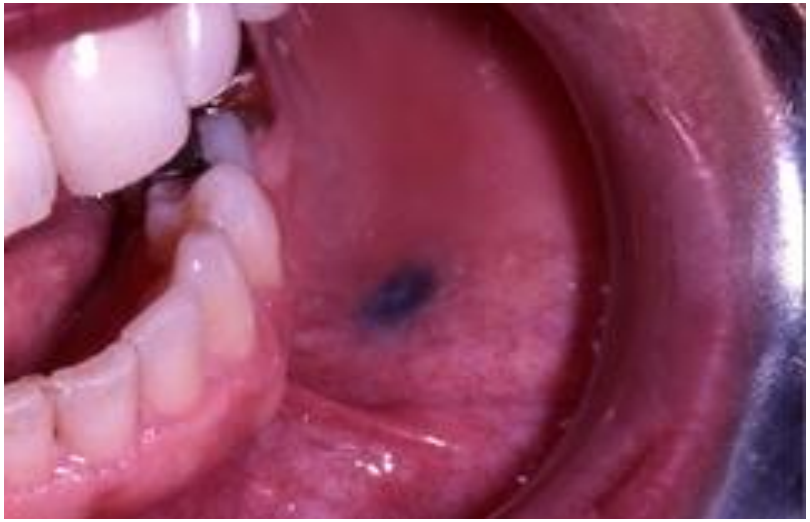
Gingival Diseases of Genetic Origin

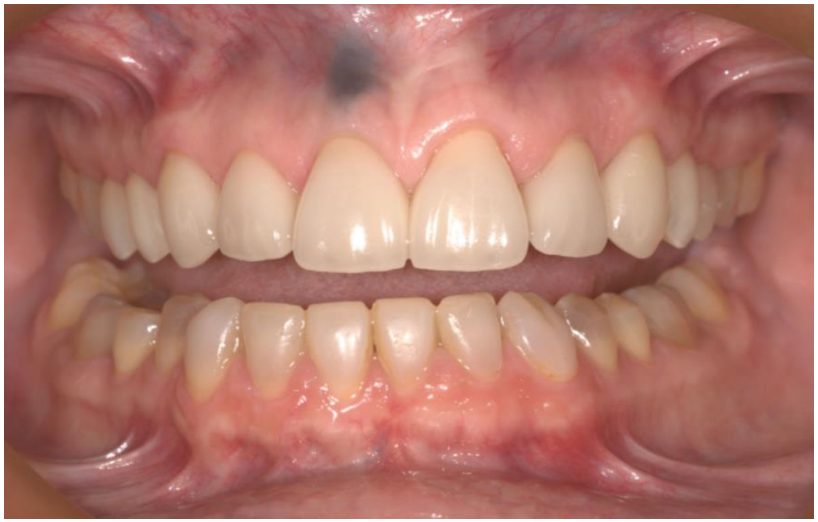


Traumatic Lesions

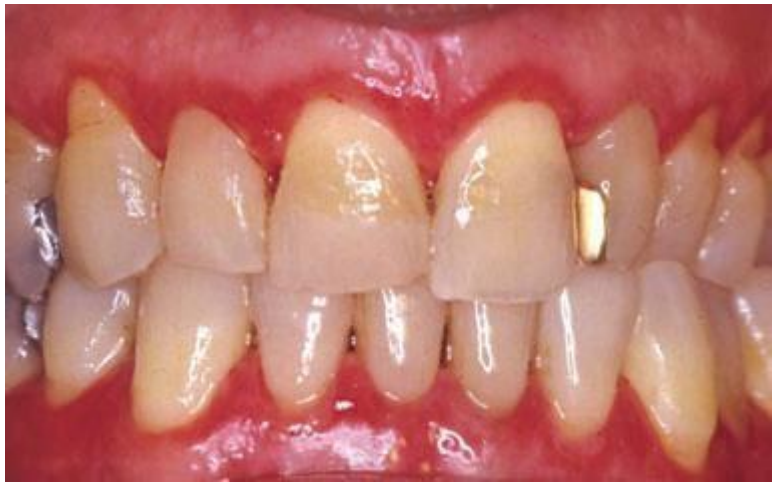


Foreign-Body Reactions

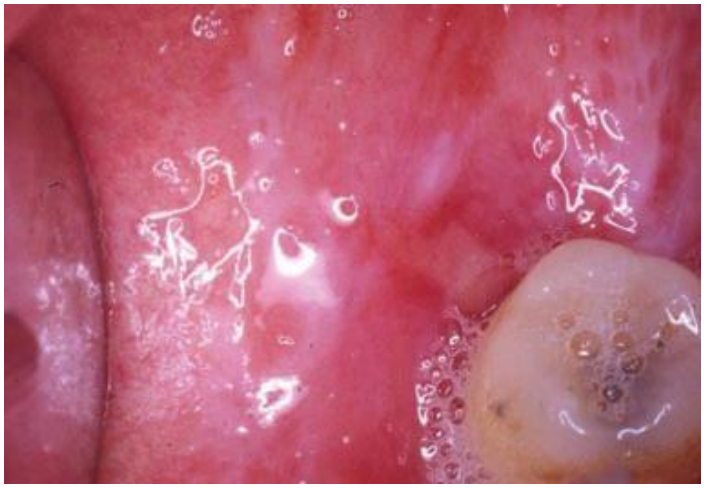




Oral lichen planus

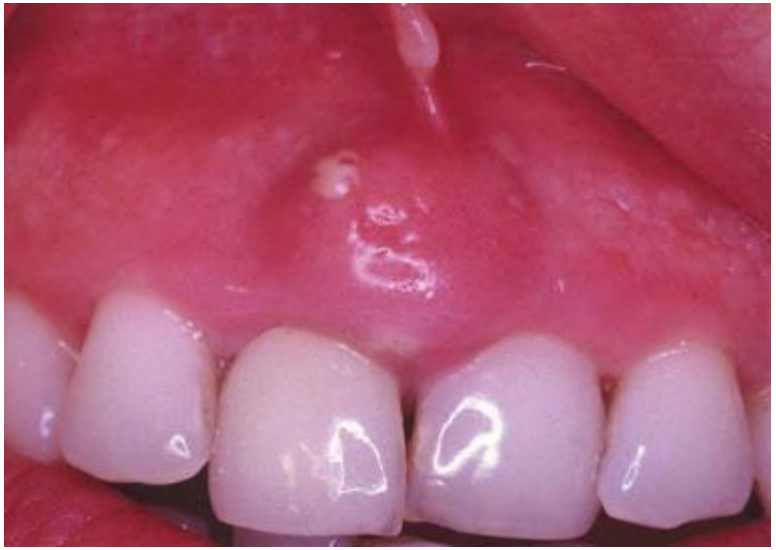


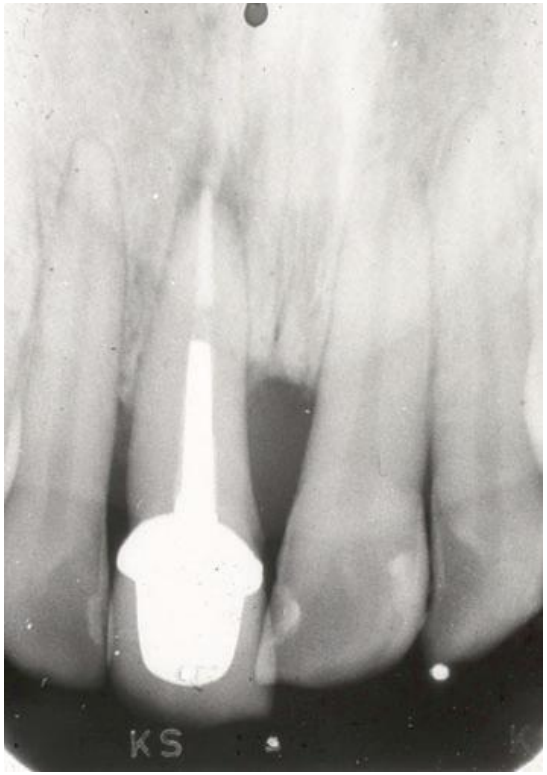
Intense erythema of the marginal and adjacent attached gingiva that has not responded to plaque control measures



Typical cheek lesions with straie and white plaques with an ulcerative lesion adjacent to the second molar, typical of oral lichen planus

Acute periodontal abscess





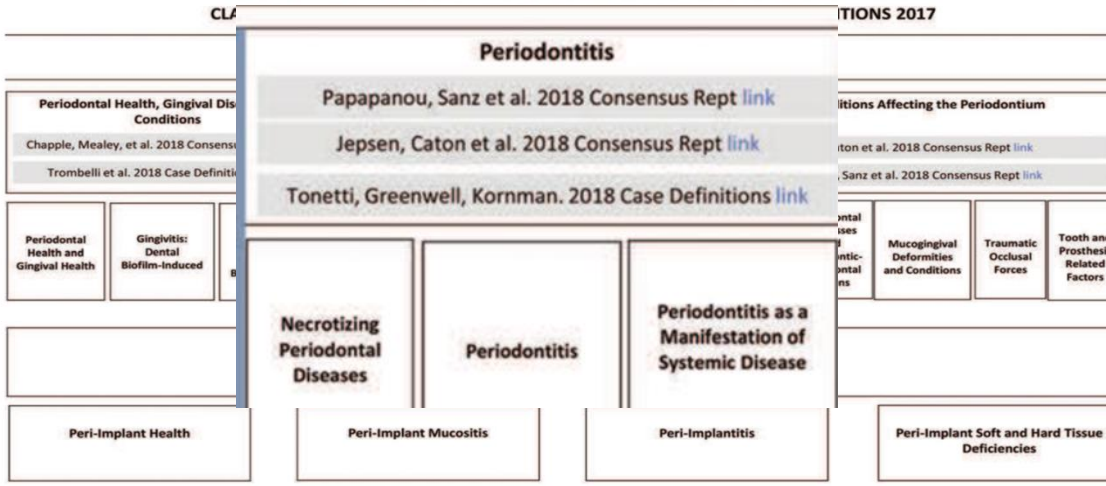
A NEW CLASSIFICATION OF PERIODONTITIS

The workshop agreed that, consistent with current knowledge on pathophysiology, three forms of periodontitis can be identified:

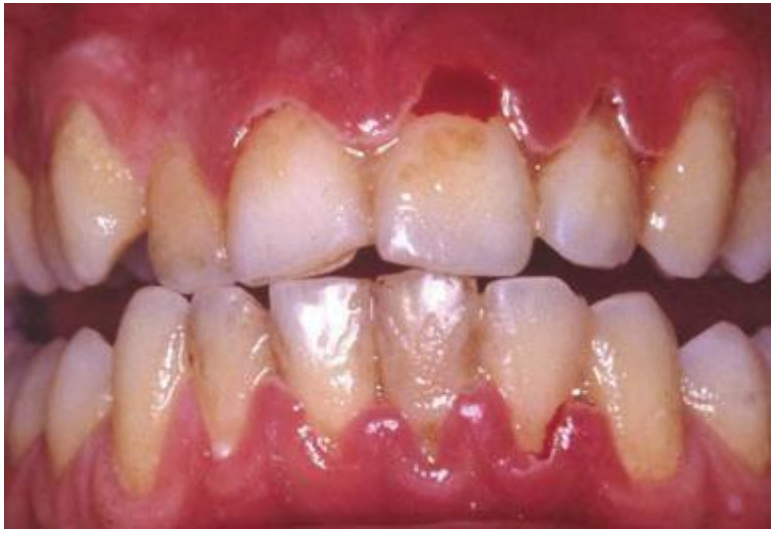
1-necrotizing periodontitis

2-periodontitis as a manifestation of systemic disease.

3-Periodontitis (the forms of the disease previously recognized as “chronic” or “aggressive”, now grouped under a single category, “periodontitis”.

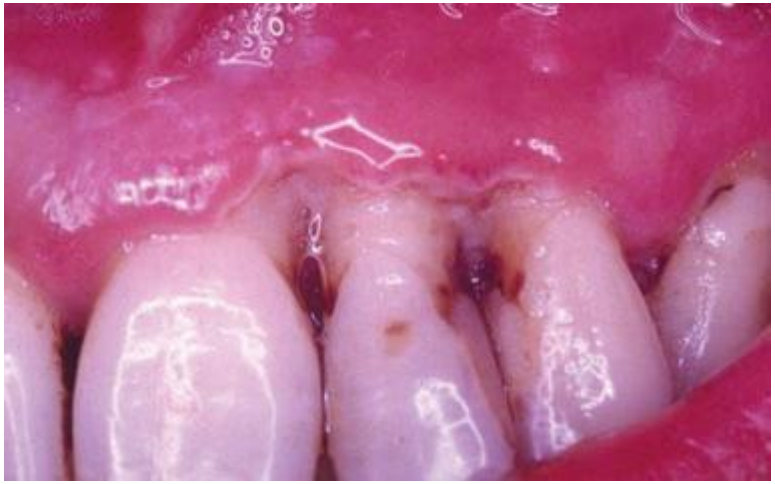


Necrotizing periodontal disease



A 21-year-old male, under stress and a smoker. Complains of pain of sudden onset, fetor oris and gingival bleeding
Note the papillary and gingival ulcers, marked bleeding and presence of a pseudomembrane at the margin, the result of necrosis.

Necrotizing periodontitis



Note the tissue destruction that has occurred following recurrent episodes of infection. The disease is not self limiting and will continue if untreated with periods of remission and exacerbation.

FORMS OF PERIODONTITIS

1. **Necrotizing Periodontal Diseases**

   Herrera et al. 2018 link

   a. Necrotizing Gingivitis
   b. Necrotizing Periodontitis
   c. Necrotizing Stomatitis

2. **Periodontitis as Manifestation of Systemic Diseases**

   Jepsen, Caton et al. 2018 Consensus Rept link | Albandar et al. 2018 link

   Classification of these conditions should be based on the primary systemic disease according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes

3. **Periodontitis**

   Fine et al. 2018 link | Needleman et al. 2018 link | Billings et al. 2018 link

   a. **Stages:** Based on Severity[1] and Complexity of Management[2]
      - Stage I: Initial Periodontitis
      - Stage II: Moderate Periodontitis
      - Stage III: Severe Periodontitis with potential for additional tooth loss
      - Stage IV: Severe Periodontitis with potential for loss of the dentition

   b. Extent and distribution[3]: localized; generalized; molar-incisor distribution
   c. **Grades:** Evidence or risk of rapid progression[4], anticipated treatment response[5]
      i. Grade A: Slow rate of progression
      ii. Grade B: Moderate rate of progression
      iii. Grade C: Rapid rate of progression

[1] Severity: Interdental clinical attachment level (CAL) at ~~site with greatest loss: Radiographic~~ bone loss & tooth loss
[2] Complexity of management: Probing depths, pattern of bone loss, furcation lesions, number of remaining teeth, tooth mobility, ridge defects masticatory dysfunction
[3] Add to Stage as descriptor: localized <30% teeth, generalized ≥ 30% teeth
[4] Risk of progression: direct evidence by PA radiographs or CAL loss, or indirect (bone loss/age ratio)
[5] Anticipated treatment response: case phenotype, smoking, hyperglycemia

Staging of periodontitis

Periodontitis stage		Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15% to 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 teeth	Tooth loss due to periodontitis of ≥5 teeth
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			

Staging of periodontitis

Staging is largely dependent upon the severity of disease at presentation as well as on the complexity of disease management,

while

grading provides supplemental information about biological features of the disease, including:-

a history based analysis of 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.

- general health status, and other exposures such as smoking or level of metabolic control in diabetes.

Thus, grading allows the clinician to incorporate individual patient factors into the diagnosis

Mucogingival conditions

The new case definitions related to treatment of gingival recession are based on interproximal loss of clinical attachment and also incorporate the assessment of the exposed root and cemento-enamel junction.

The consensus report presents a new classification of gingival recession that combines clinical parameters including the gingival phenotype as well as characteristics of the exposed root surface.

<u>In the consensus report the term periodontal biotype was replaced by periodontal phenotype</u>

3. Mucogingival deformities and conditions around teeth

Cortellini & Bissada 2018 link

a. Gingival phenotype
b. Gingival/soft tissue recession
c. Lack of gingiva
d. Decreased vestibular depth
e. Aberrant frenum/muscle position
f. Gingival excess
g. Abnormal color
h. Condition of the exposed root surface

Occlusal trauma and traumatic occlusal forces

Traumatic occlusal force, replacing the term excessive occlusal force, is the force that exceeds the adaptive capacity of the periodontium and/or the teeth. Traumatic occlusal forces can result in occlusal trauma (the lesion) and excessive wear or fracture of the teeth.

There is lack of evidence from human studies implicating occlusal trauma in the progression of attachment loss in periodontitis.

4. **Traumatic occlusal forces**

Fan & Caton 2018 link

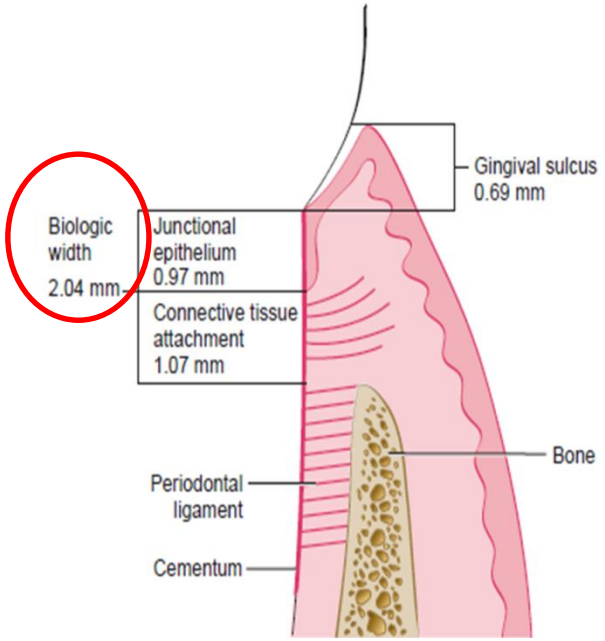
a. Primary occlusal trauma
b. Secondary occlusal trauma
c. Orthodontic forces

Prosthesis- and tooth-related factors

The section on prostheses-related factors was expanded in the new classification.
The term biologic width was replaced by supracrestal attached tissues.

Clinical procedures involved in the fabrication of indirect restorations was added because of new data indicating that these procedures may cause recession and loss of clinical attachment.

supracrestal attached tissues



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

Ercoli & Caton 2018 link

a. Localized tooth-related factors
b. Localized dental prostheses-related factors

Peri-implant Diseases and Conditions Consensus Report
Berglundh, Armitage et al. 2018
Active link to consensus report

PERI-IMPLANT DISEASES AND CONDITIONS

1. **Peri-implant health**

   Araujo & Lindhe 2018 link

2. **Peri-implant mucositis**

   Heitz-Mayfield & Salvi 2018 link

3. **Peri-implantitis**

   Schwarz et al. 2018 link

4. **Peri-implant soft and hard tissue deficiencies**

   Hammerle & Tarnow 2018 link

Renvert et al. 2018 Case Definitions link

A NEW CLASSIFIC ATION FOR PERI-IMPLANT DISEASES AND CONDITIONS

A new classification for peri-implant health,peri-implant mucositis and peri-implantitis was developed by the workshop. An effort was made to review all aspects of peri-implant health, diseases, and relevant aspects of implant site conditions and deformities to achieve a consensus for this classification that could be accepted worldwide. Case definitions were developed for use by clinicians for individual case management and also for population studies.

Peri-implant health

Peri-implant health was defined both clinically and histologically.Clinically, peri-implant health is characterized by an absence of visual signs of inflammation and bleeding on probing. Peri-implant health can exist around implants with normal or reduced bone support. It is not possible to define a range of probing depths compatible with peri-implant health.

Peri-implant mucositis

Peri-implant mucositis is characterized by bleeding on probing and visual signs of inflammation. While there is strong evidence that peri-implant mucositis is caused by plaque, there is very limited evidence for non-plaque induced peri-implant mucositis. Peri-implant mucositis can be reversed with measures aimed at eliminating the plaque.

Peri-implantitis

Peri-implantitis was defined as a plaque-associated pathologic condition occurring in the tissue around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone. Peri-implant mucositis is assumed to precede peri-implantitis. Peri-implantitis is associated with poor plaque control and with patients with a history of severe periodontitis.

The onset of peri-implantitis may occur early following implant placement as indicated by radiographic data. Peri-implantitis, in the absence of treatment, seems to progress in a non-linear and accelerating pattern.

Hard and soft tissue implant site deficiencies

Normal healing following tooth loss leads to diminished dimensions of the alveolar process/ridge that result in both hard and soft tissue deficiencies.

Larger ridge deficiencies can occur at sites associated with severe loss of periodontal support, extraction trauma, endodontic infections,root fractures, thin buccal bone plates, poor tooth position,injury and pneumatization of the maxillary sinuses.

Other factors affecting the ridge can be associated with medications and systemic diseases reducing the amount of naturally formed bone, tooth agenesis,

and pressure from prostheses

References

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