CHRONIC PERIODONTITIS

INTRODUCTION

- Chronic periodontitis is the most prevalent(10.5% to 12%) form of periodontitis, and it generally shows the characteristics of a *slowly progressing inflammatory disease.*
- belongs to the group of *complex* inflammatory diseases in humans.

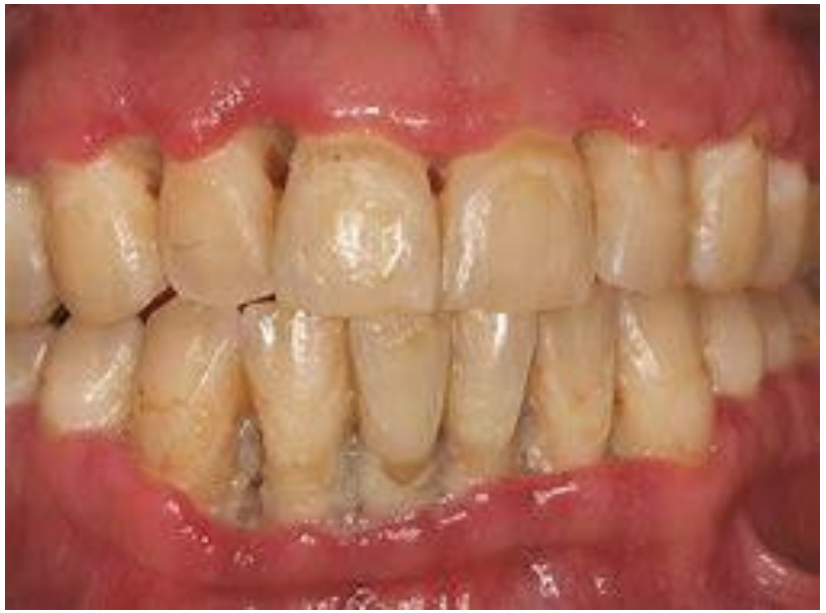
- the word complex not only describes the fact that there are *multiple clinical symptoms* that account for the disease, but also explains the *multiple factors* that lead to and influence periodontal inflammation.

DEFINITION

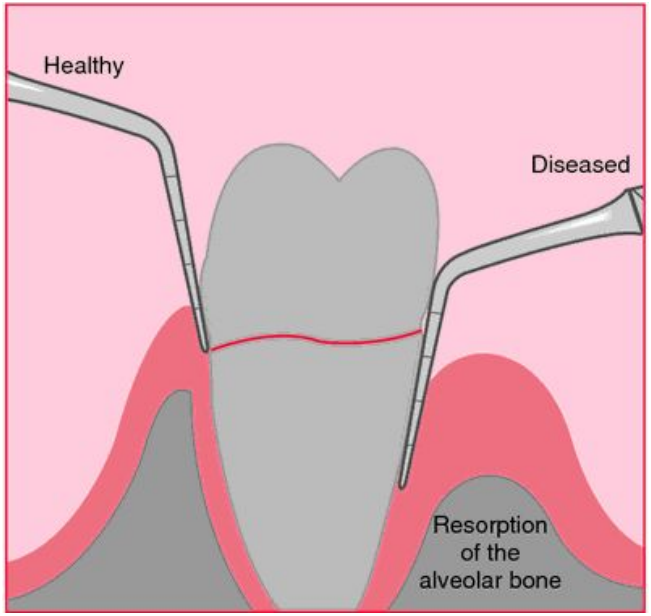
an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss, and bone loss.

Chronic periodontitis represents major clinical and etiologic characteristics such as

microbial biofilm formation



periodontal inflammation



attachment as well as alveolar bone loss

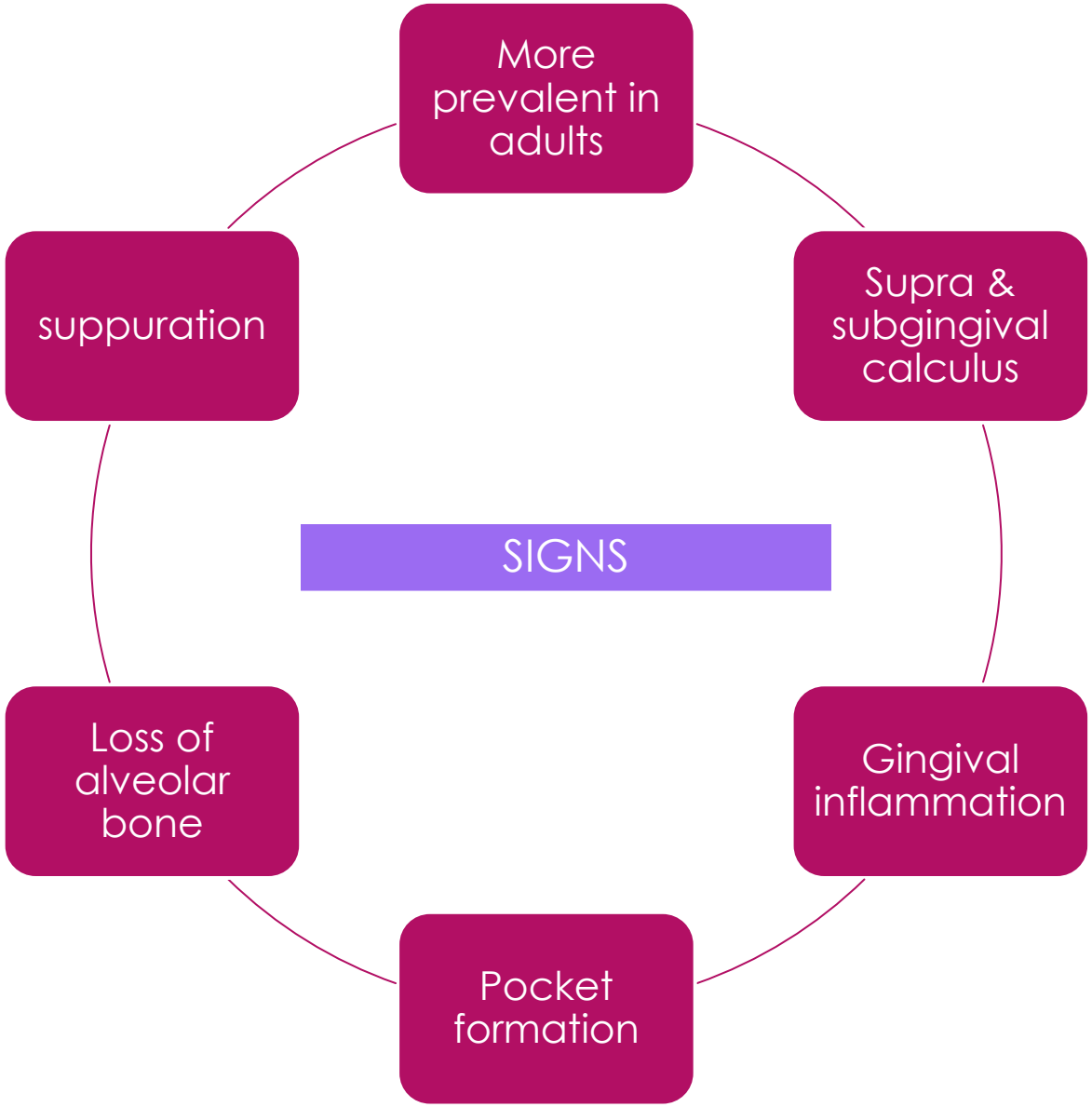
CLASSIFICATION

Classification	Forms of Periodontitis	Disease Characteristics
American Academy of Periodontology World Workshop in Clinical Periodontics, 1989[5]	Adult periodontitis	Age of onset >35 years Slow rate of disease progression No defects in host defenses
	Early-onset periodontitis (may be prepubertal, juvenile, or rapidly progressive)	Age of onset <35 years Rapid rate of disease progression Defects in host defenses Associated with specific microflora
	Periodontitis associated with systemic disease	Systemic diseases that predispose to rapid rates of periodontitis Diseases: diabetes, Down syndrome, human immunodeficiency virus infection, Papillon–Lefèvre syndrome
	Necrotizing ulcerative periodontitis	Similar to acute necrotizing ulcerative gingivitis but with associated clinical attachment loss
	Refractory periodontitis	Recurrent periodontitis that does not respond to treatment
European Workshop in Periodontology, 1993[5]	Adult periodontitis	Age of onset: fourth decade of life Slow rate of disease progression No defects in host response
	Early-onset periodontitis	Age of onset <40 years Rapid rate of disease progression Defects in host defense
	Necrotizing periodontitis	Tissue necrosis with attachment and bone loss
American Academy of Periodontology International Workshop for Classification of Periodontal Diseases, 1999[4]	Chronic periodontitis; aggressive periodontitis; periodontitis as a manifestation of systemic disease	See Box 3-3

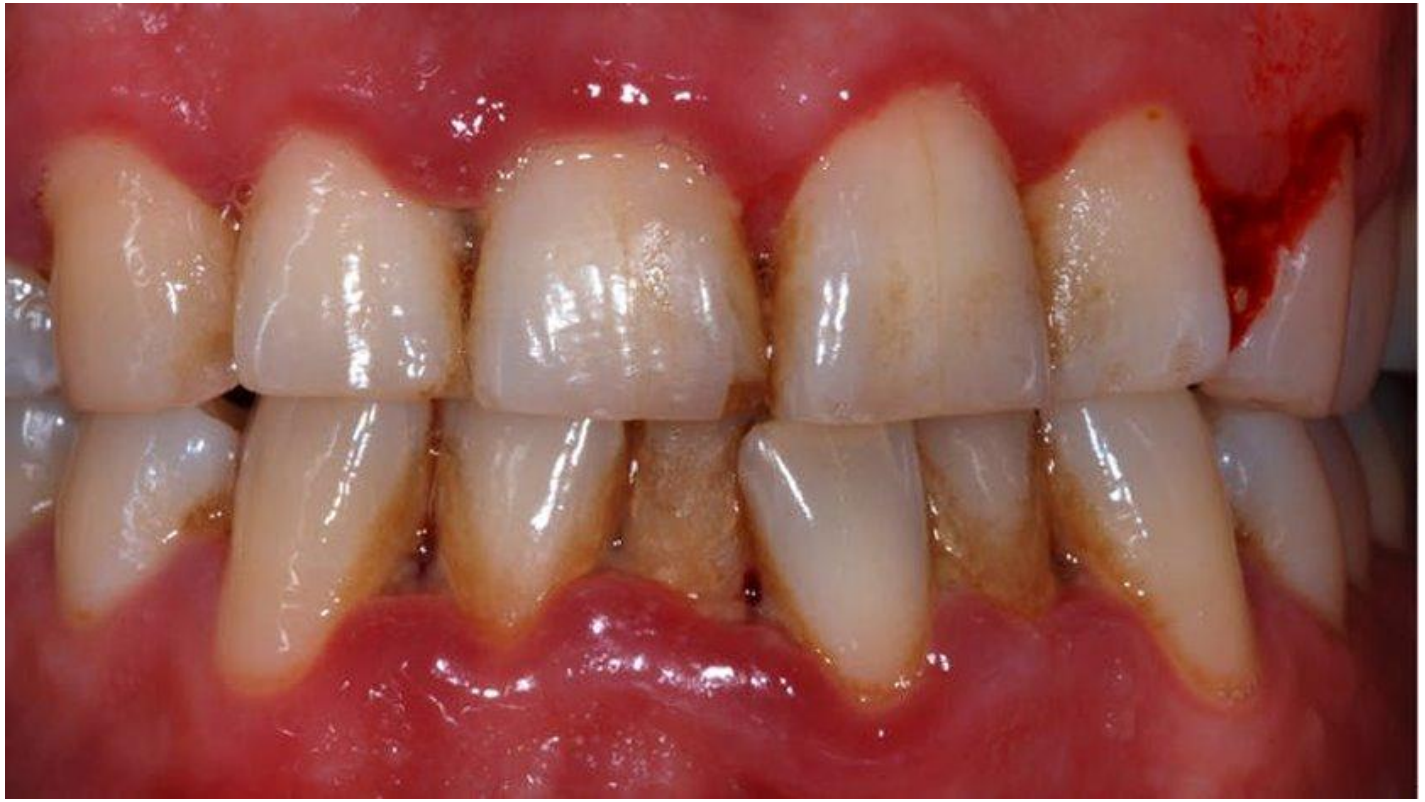
PREVALENCE

- an *age-associated* (not an age-related) disease
- increases in prevalence and severity with age
- affects both genders equally
- 50% of the human population - at least one form of periodontal disease.

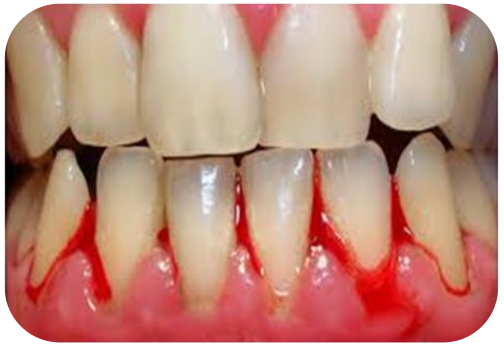
CLINICAL FEATURES


More prevalent in adults
Supra & subgingival calculus
Gingival inflammation
Pocket formation
Loss of alveolar bone
suppuration
SIGNS

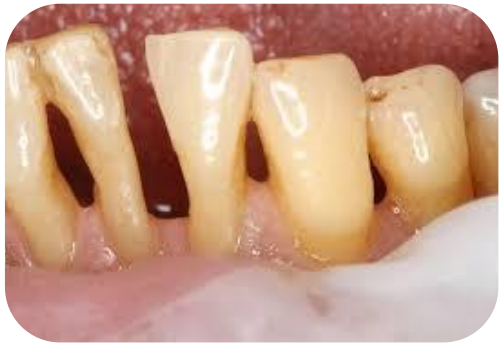
SIGNS



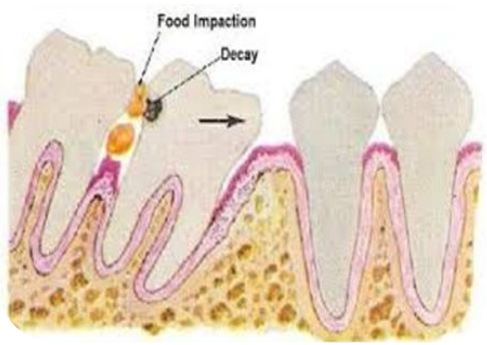
SYMPTOMS



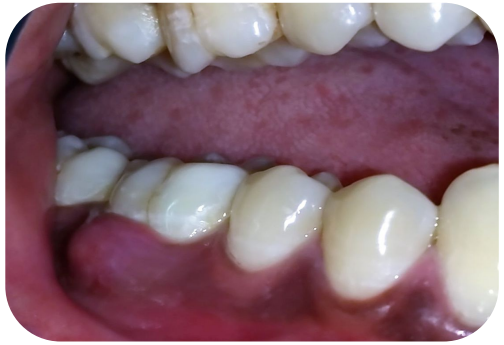
Bleeding gums



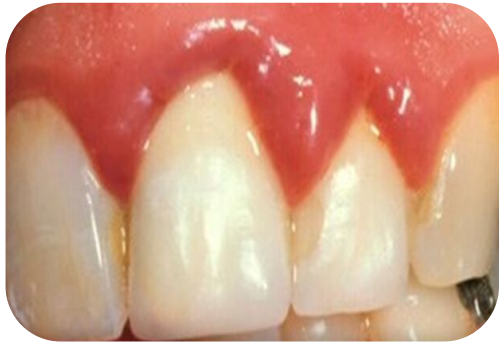
Loose teeth



food impaction

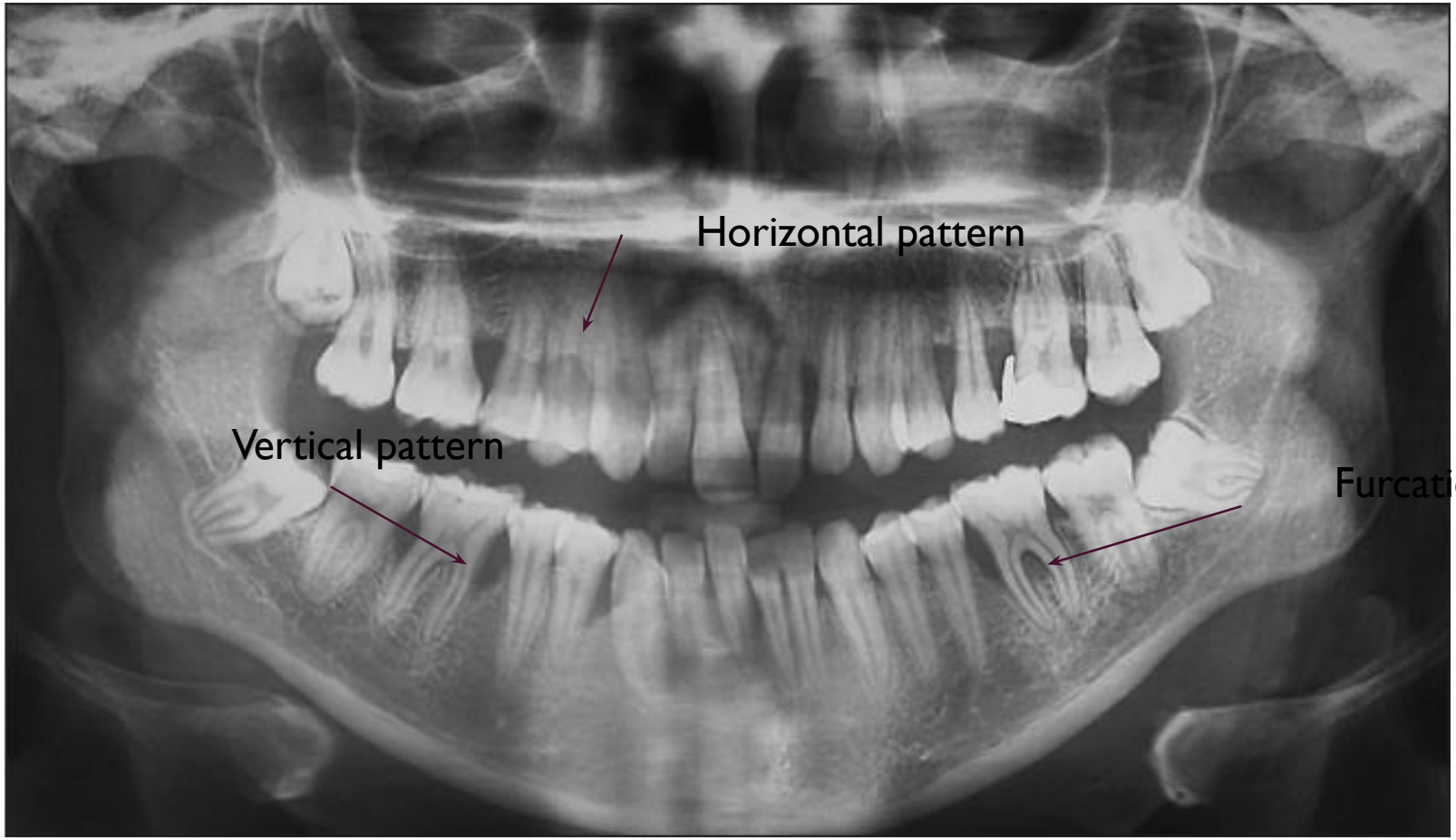


periodontal abscess



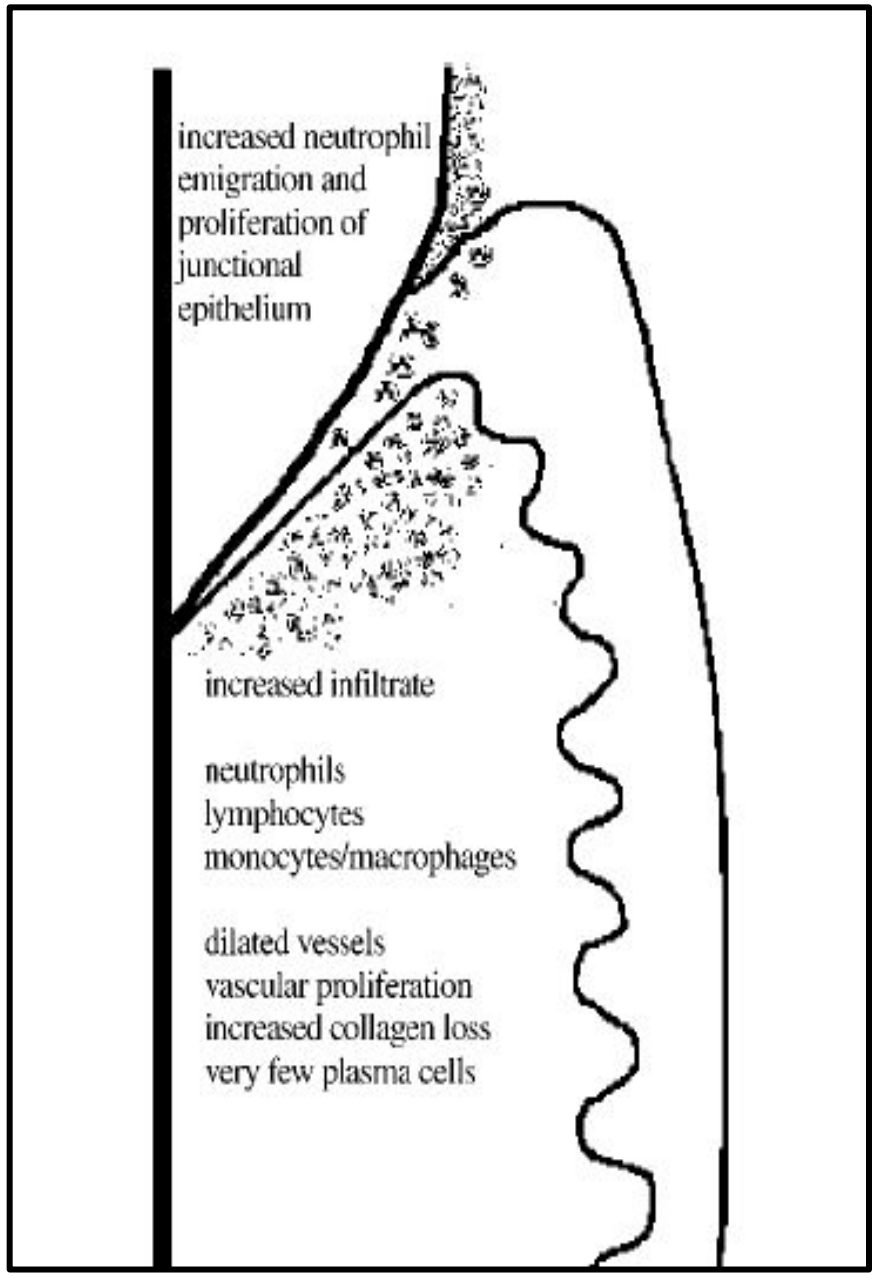
Gingival tenderness

RADIOGRAPHIC FEATURES


Horizontal pattern
Vertical pattern
Furcation involvement

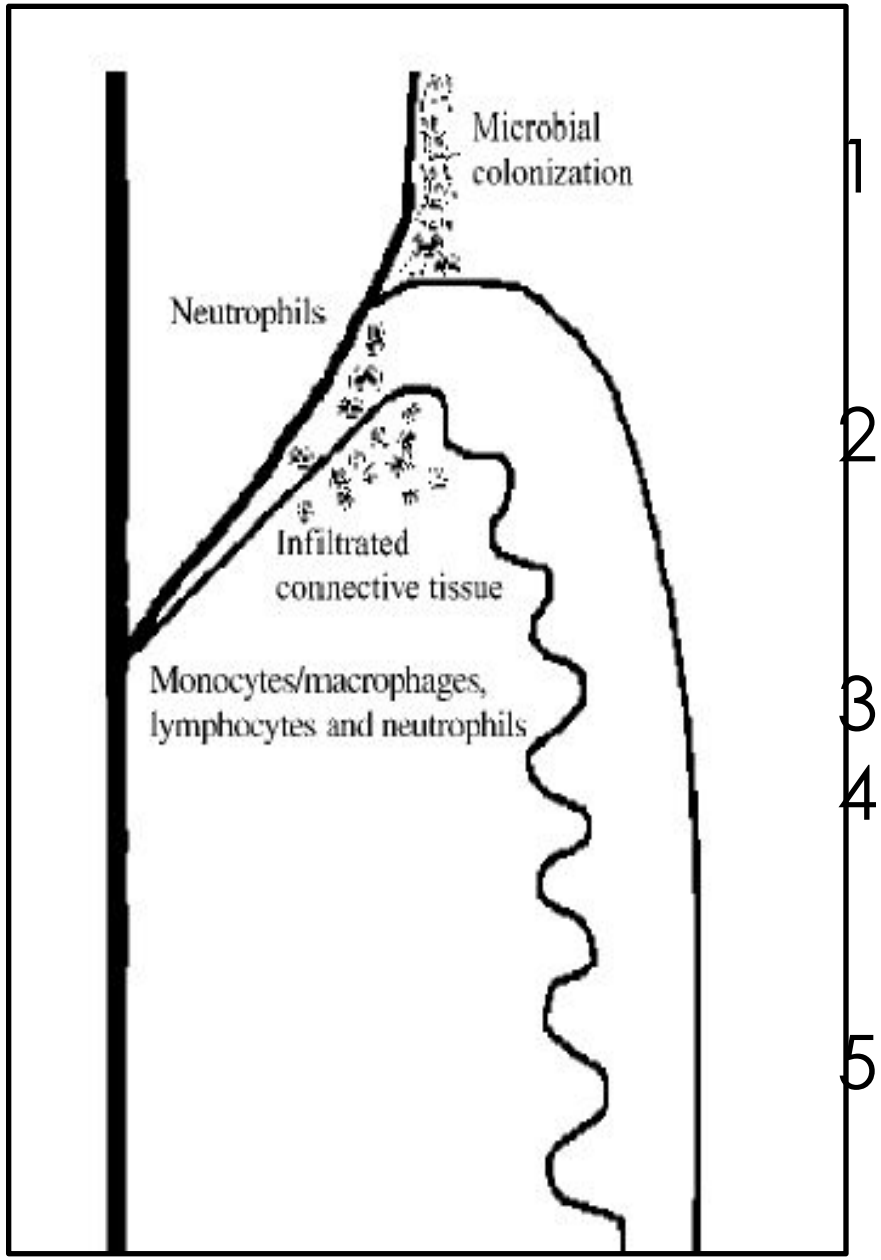
HISTOPATHOLOGICAL CHANGES

INITIAL LESION



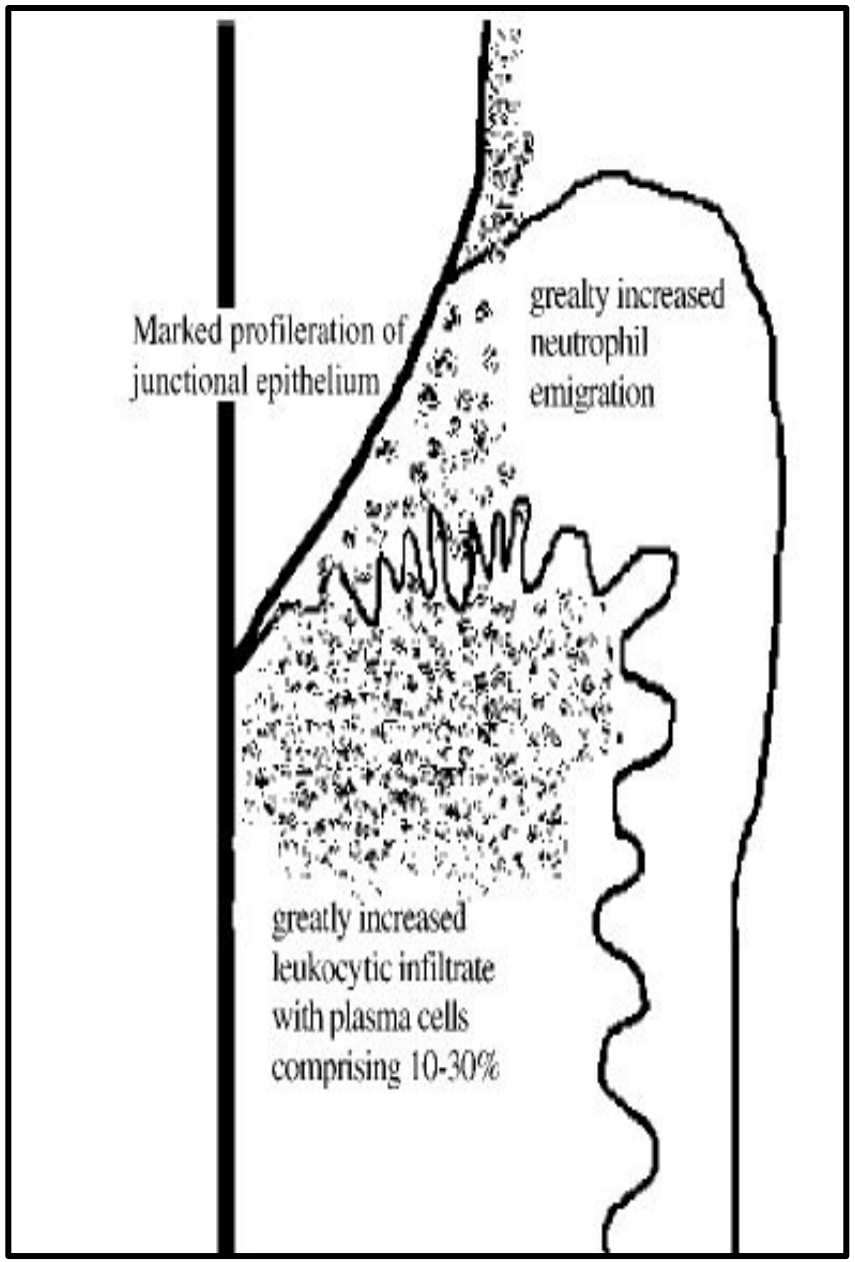
1. Slightly elevated vascular permeability and vasodilation
2. Gingival crevicular fluid flows out of the sulcus
3. Migration of leukocytes, primarily neutrophils, in relatively small numbers through the gingival connective tissue, across the junctional epithelium, and into the sulcus

EARLY LESION



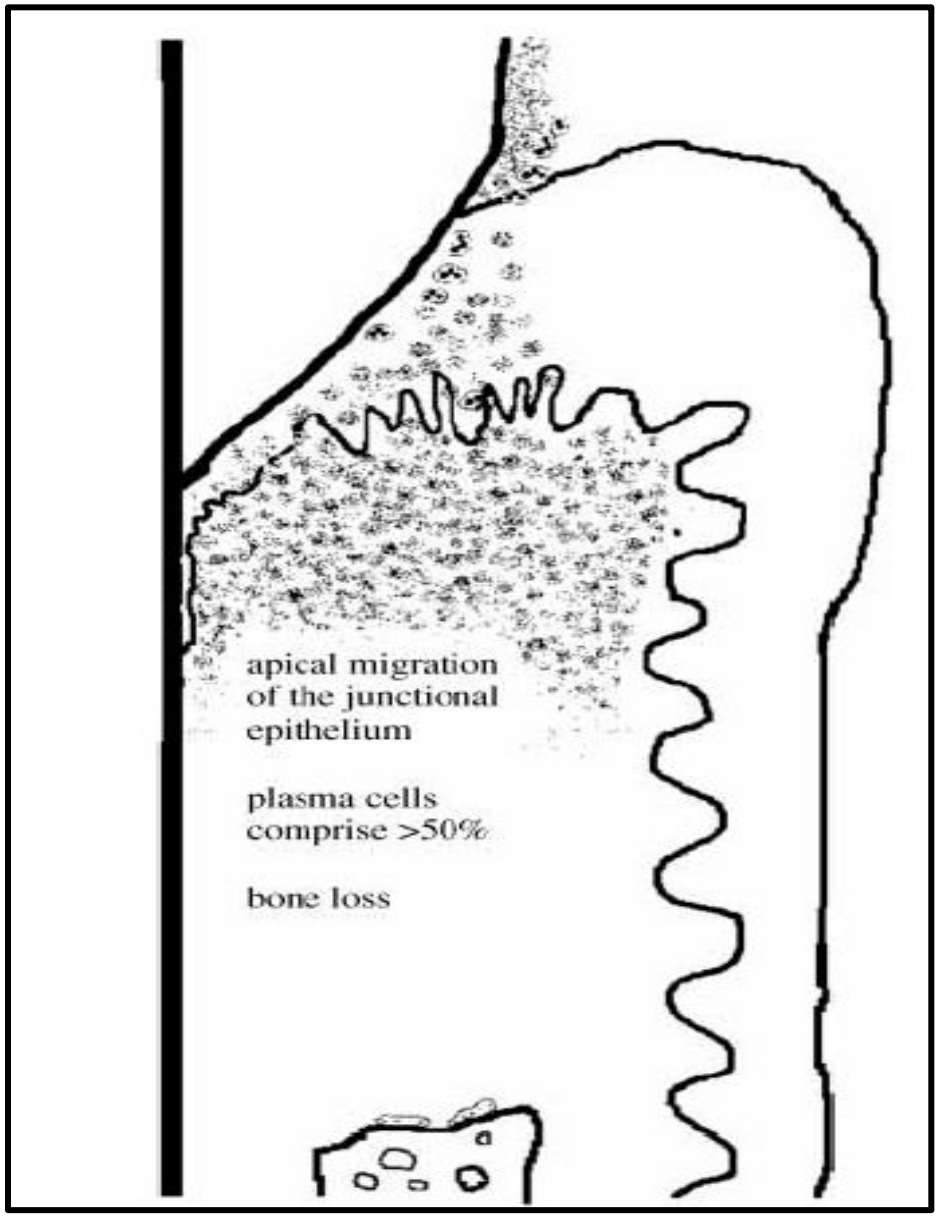
1. Increased vascular permeability, vasodilation, and gingival crevicular fluid flow
2. Large numbers of infiltrating leukocytes (mainly neutrophils and lymphocytes)
3. Degeneration of fibroblasts
4. Collagen destruction that results in collagen-depleted areas of the connective tissue
5. Proliferation of the junctional and sulcular epithelium into collagen-depleted areas

ESTABLISHED LESION



1. Dense inflammatory cell infiltrate (i.e., plasma cells,lymphocytes, and neutrophils).
2. Accumulation of inflammatory cells in the connective tissues
3. Elevated release of matrix metalloproteinases and lysosomal contents from neutrophils
4. Significant collagen depletion and proliferation of epithelium
5. Formation of pocket epithelium that contains large numbers of neutrophils

ADVANCED LESION



1. Predominance of neutrophils in the pocket epithelium and in the pocket
2. Dense inflammatory cell infiltrate in the connective tissues (primarily plasma cells)
3. Apical migration of junctional epithelium to preserve an intact epithelial barrier
4. Continued collagen breakdown that results in large areas of collagen-depleted connective tissue
5. Osteoclastic resorption of alveolar bone

DISEASE DISTRIBUTION

- ***site-specific*** clinical picture
- attachment and bone loss are not equally distributed throughout the dentition as well as around teeth
- Due to the site-specific nature and based on the number of teeth with clinical attachment loss, chronic periodontitis can be classified into the following categories:

Localized

less than 30% of the teeth show attachment and bone loss

Generalized

30% or more of the teeth show attachment and bone loss

- the local inflammatory response may lead to different patterns of bone loss, including vertical(angular) and horizontal bone destruction.
- Although vertical bone loss is associated with intrabony pocket formation, horizontal bone loss is usually associated with suprabony (supraalveolar) pockets.

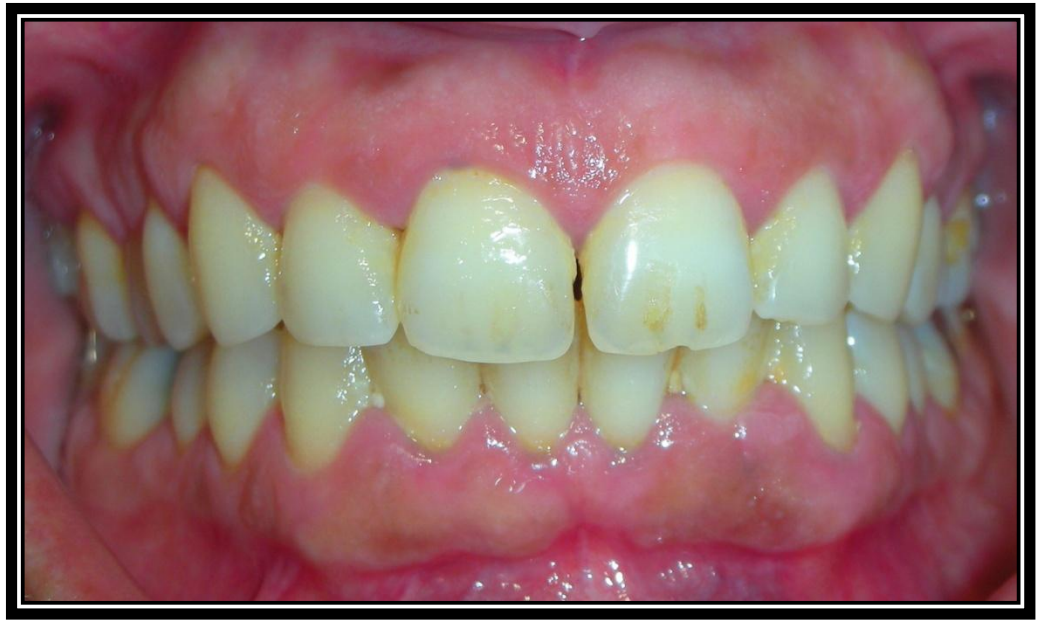
Key Differences Between Gingivitis and Chronic Periodontitis

Plaque-Induced Gingivitis	Chronic Periodontitis
Inflammation of the gingiva without attachment/bone loss.	Inflammation of periodontal apparatus with attachment/bone loss.
With optimal oral hygiene, this condition can be resolved completely (reversible).	The attachment loss is irreversible, in spite of successfully controlling the inflammation.
Not all sites with gingivitis progress to periodontitis	All patients with chronic periodontitis must have experienced prior gingivitis
The dental implant counterpart of gingivitis is peri-implant mucositis.	The dental implant counterpart of periodontitis is peri-implantitis.

DISEASE SEVERITY

Mild Periodontitis

clinical attachment loss of 1 to 2 mm

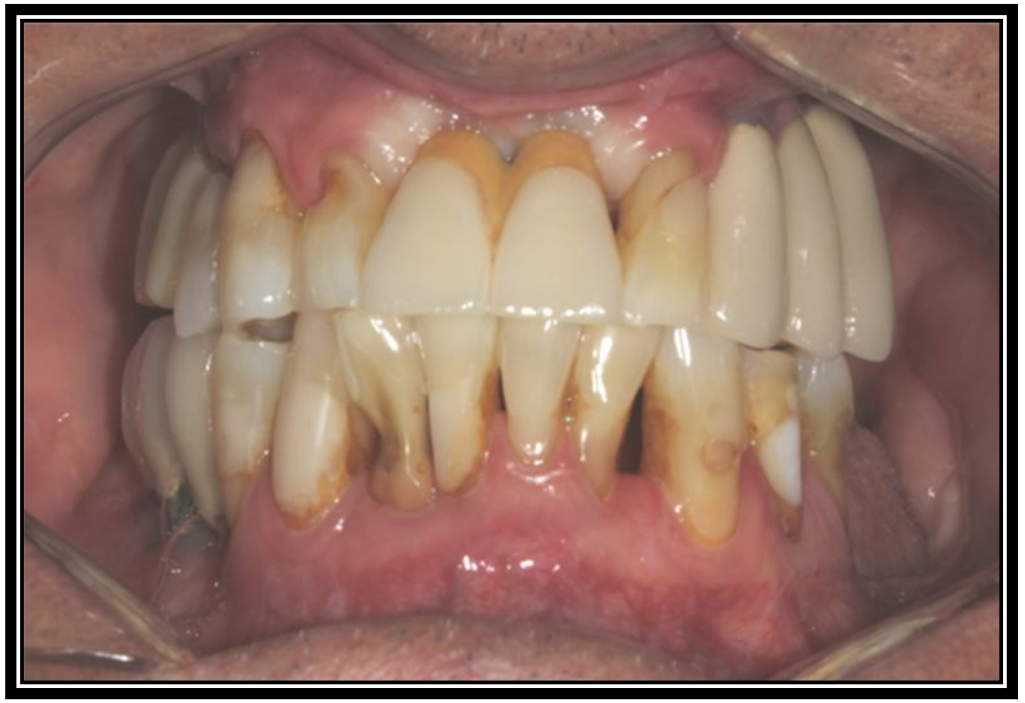


Moderate Periodontitis

clinical attachment loss of 3 to 4 mm

Severe Periodontitis

clinical attachment loss of 5 mm or more



Site Specificity of Chronic Periodontitis

- **Not all sites in the mouth are equally prone to chronic periodontitis, and it exhibits site specificity.**
- **The progression of the disease occurs in certain sites but not uniformly.**
- **Interproximal sites, in general, are more prone to periodontal destruction, compared to buccal/facial sites.**

DISEASE PROGRESSION

- Patients appear to have similar susceptibility to plaque induced chronic periodontitis throughout their lives.
- The rate of disease progression is usually slow but may be modified by environmental, systemic or behavioral factors.
- Because of its slow progression it usually becomes clinically significant in the mid 30s or later.

Models of change of periodontal status

- ***Continuous model*** (Socransky et al 1984): disease progression is slow and continuous, with affected sites showing a constantly progressive rate of destruction throughout the duration of the disease.

- ***Random or Episodic burst theory*** (Goodson et al; 1982; Zimmerman 1986): proposes that periodontal disease progresses by short bursts of destruction followed by periods of no destruction. This pattern of disease is random with respect to the tooth sites affected and the chronology of the disease process.

- ***Asynchronous,multiple-burst theory*** (Socransky et al 1984): suggests that periodontal destruction occurs around affected teeth during defined periods of life and that these bursts of activity are interspersed with periods of inactivity or remission. The chronology of these bursts of disease is asynchronous for individual teeth or groups of teeth.

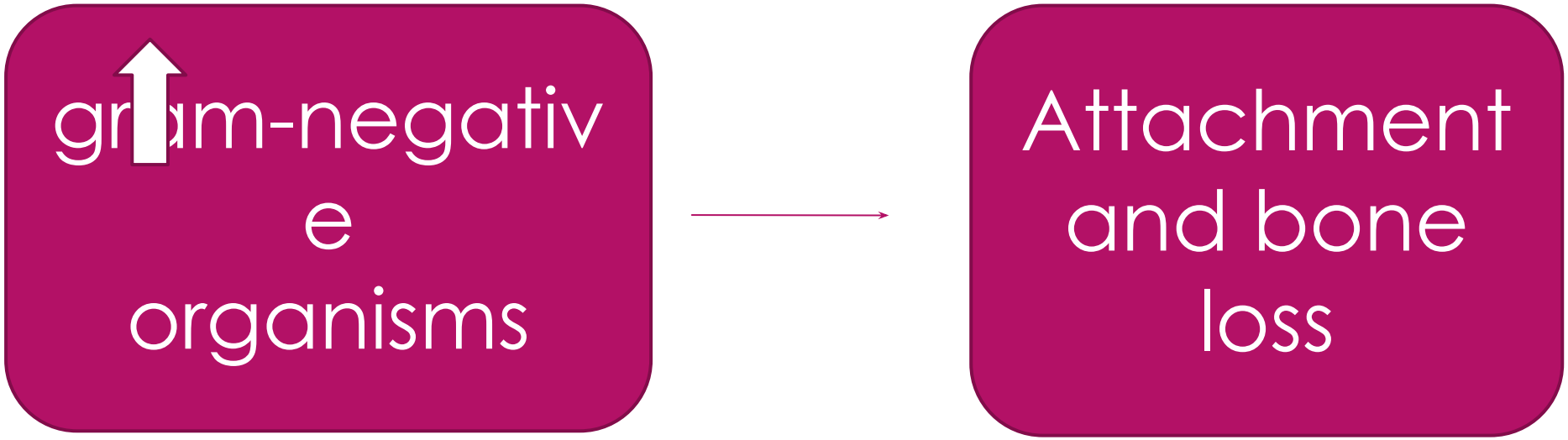
RISK FACTORS FOR PERIODONTITIS

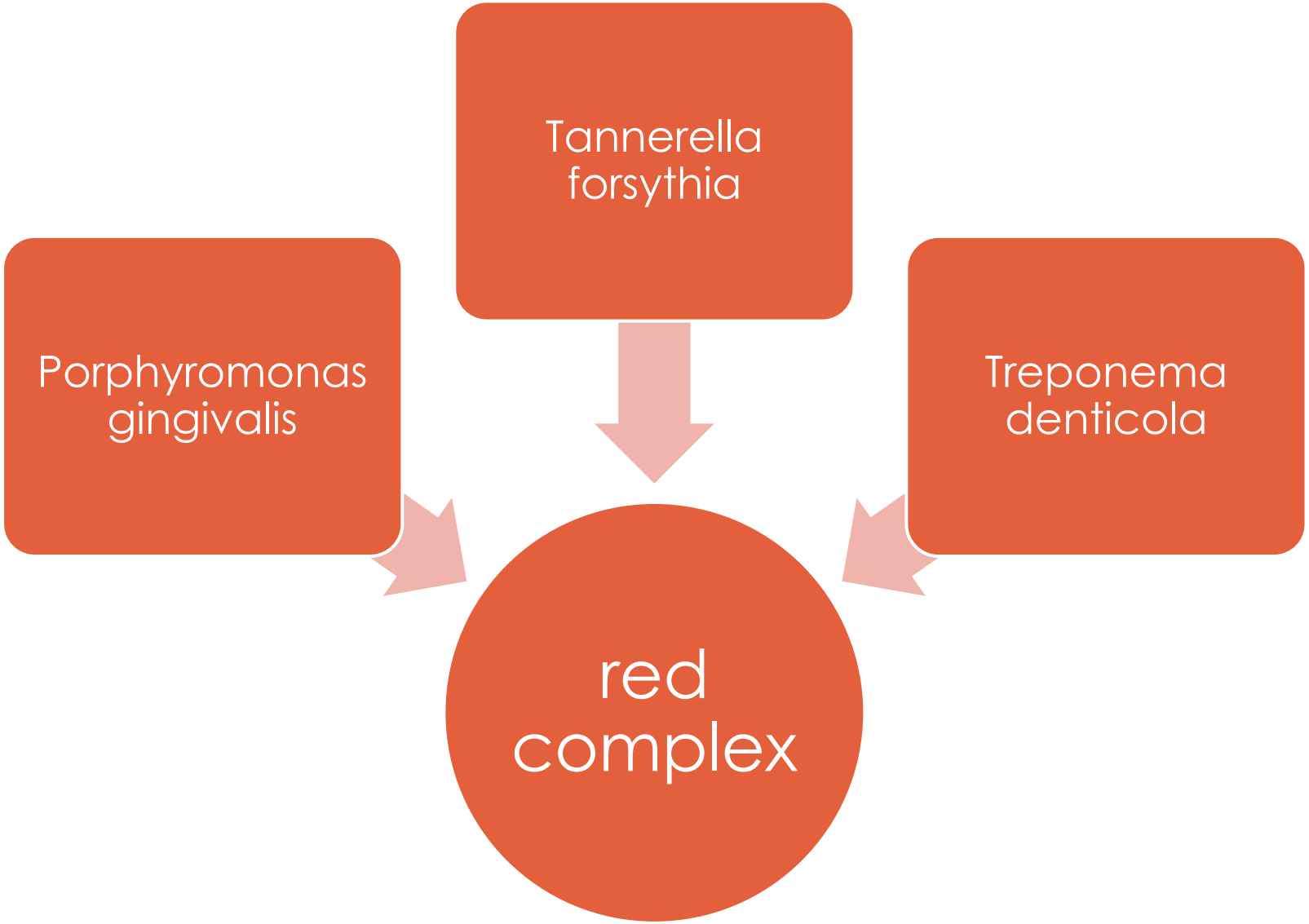
- A number of factors influence the etiopathogenesis of chronic periodontitis.
- composition of the oral microflora and the amount of dental biofilm (plaque) are major etiologic factors
- risk factors may occur simultaneously, or a selection of factors is present in patients with chronic periodontitis

- Degree of the individual risk factor contribution differs among patients, so it is worthwhile to not only identify the risk factors but also to specify each risk factor's degree of contribution
- prior history of gingivitis and periodontitis should be considered as general predictors for the development or progression of chronic periodontitis

Microbiological Aspects

- Plaque accumulation - primary initiating agent

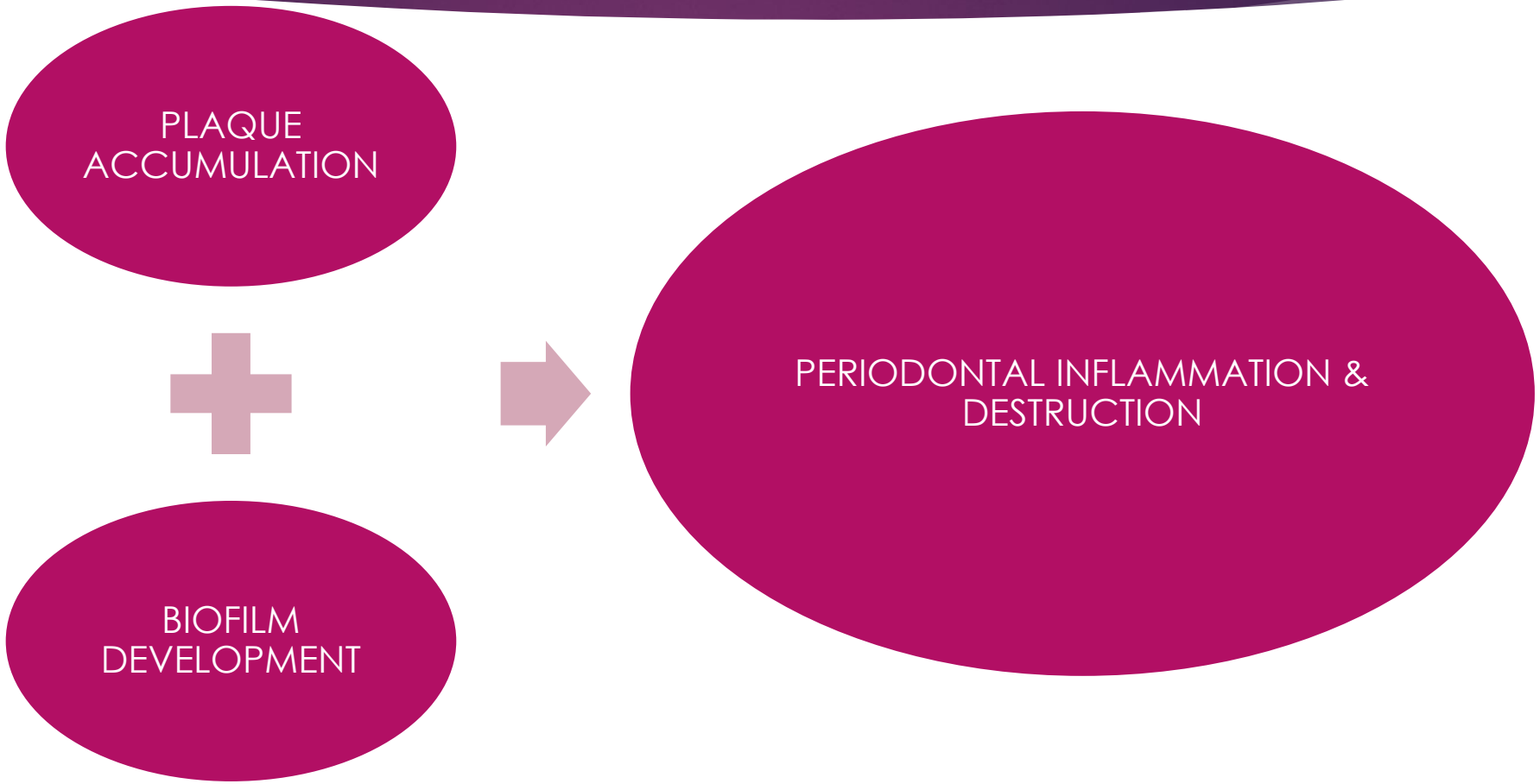



Tannerella forsythia
Porphyromonas gingivalis
Treponema denticola
red complex

- Periodontal pathogens may invade the periodontal tissue and thus induce an immune response with increasing concentrations of proinflammatory mediators that may enhance periodontal breakdown.
- In addition, a number of periodontal pathogens are capable of producing proteases that directly influence tissue stability and host immune responses.

- As the dental biofilm develops, early signs of an inflammatory reaction occur in the gingival margin (i.e., gingivitis), without actual attachment loss
- Generally, optimal plaque control leads to the complete resolution of this early gingival inflammation.
- Alternatively, with neglected oral hygiene, inflammation will progress and eventually result in the loss of attachment around teeth.

Local Factors



PLAQUE RETENTIVE FACTORS

- CALCULUS
- ROOT IRREGULARITIES
- OVERHANGING RESTORATIONS
- FURCATION
- SUBGINGIVAL CARIES

Systemic Factors

- Periodontitis is also associated with other systemic disorders, such as Haim– Munk syndrome, Papillon–Lefèvre syndrome, Ehlers–Danlos syndrome, Kindlers syndrome, and Cohen syndrome.
- Diseases that impair the host immune response (e.g., human immunodeficiency virus, acquired immunodeficiency syndrome) may also show periodontal destruction.

- It is also known that osteoporosis, a severely unbalanced diet, and stress as well as dermatologic, hematologic, and neoplastic factors interfere with periodontal inflammatory responses.
- In addition to being associated with defined syndromes, periodontitis also occurs with severe systemic diseases, such as diabetes mellitus, cardiovascular disorders, stroke, and lung disorders.

Immunologic Factors

- Chronic periodontitis is a disease that is induced by bacteria organized in the dental biofilm. However, the onset, progression, and severity of the disease depend on the individual host's immune response.
- Patients may show alterations in their peripherial monocytes, which are related to the reduced reactivity of lymphocytes or an enhanced B-cell response.

Genetic Factors

- Genetic variations such as single nucleotide polymorphisms (SNPs) and genetic copy number variations may directly influence innate and adaptive immune responses as well as the structure of periodontal tissues.
- Periodontal destruction has been found among family members and across different generations within a family, thereby suggesting a genetic basis for the susceptibility to periodontal disease.

Environmental and Behavioral Factors

- In addition to microbial, immunologic, and genetic factors, the development and progression of chronic periodontitis is further influenced by environmental and behavioral factors, such as smoking and psychological stress.

Smoking

- major risk factor
- Periodontitis is influenced by smoking in a dose dependent manner.
- The intake of more than 10 cigarettes per day tremendously increases the risk of disease progression as compared with non-smokers and former smokers.

the following features are found in smokers.

- Increased periodontal pocket depth of more than 3 mm
- Increased attachment loss
- More recessions
- Increased loss of alveolar bone
- Increased tooth loss
- Fewer signs of gingivitis (e.g., less bleeding with probing)
- Greater incidence of furcation involvement

- As a result of the consumption of tobacco, reactive oxygen (i.e., radicals) is released that chemically irritates periodontal tissues via DNA damage, the lipid peroxidation of cell membranes, the damage of endothelial cells, and the induction of smooth muscle cell growth.

Stress

- Emotional stress may interfere with normal immune function (Ballieux 1991 Haffajee AD, Socransky SS 1994); and may result in increased levels of circulating hormones that can have an impact on the periodontium (Rose 1980).
- Individuals with financial strain, distress, depression, or inadequate coping mechanisms have more severe loss of attachment. (Genco 1999).

- Adult patients with periodontitis who are resistant to therapy are more stressed than those who respond to therapy. (Axetelius 98)

DIAGNOSIS

TREATMENT

- Chronic periodontitis can be treated effectively by a systematic periodontal therapy that includes **optimal long-term plaque control, debridement of soft and hard deposits, or surgical pocket reduction**
- Depending on the individual periodontal risk, each patient should be remotivated, reinstructed, and retreated (if necessary) during a systematic supportive periodontal therapy regimen

- Scaling and root planing, curettage
- Reevaluation at regular intervals (2 weeks to 9 months)
- Surgical therapy which includes
  - Periodontal flap surgery
  - Resective osseous surgery
  - Regenerative periodontal therapy

CONCLUSION

- A timely diagnosis of chronic periodontitis at its earliest stage is essential to avoid more challenging severe stages of the disease.
- Most cases of slight and moderate chronic periodontitis can be successfully managed by mechanical removal and/or reduction of subgingival bacterial biofilms and calculus

- However, any factor that affects either the local environment or the host response may contribute to progression of the disease and a poor treatment response.
- Thus, it is essential that clinicians are aware of etiologic and risk factors associated with disease development and progression in order to plan and execute a successful treatment.

FREQUENTLY ASKED QUESTIONS

- CLINICAL FEATURES OF CHRONIC PERIODONTITIS – 2M,5M (DEC 2016, JAN 2018,JULY 2008)
- DIFFERENCE BETWEEN CHRONIC AND AGGRESSIVE EPRIODONTITIS-2M


THANK
YOU!