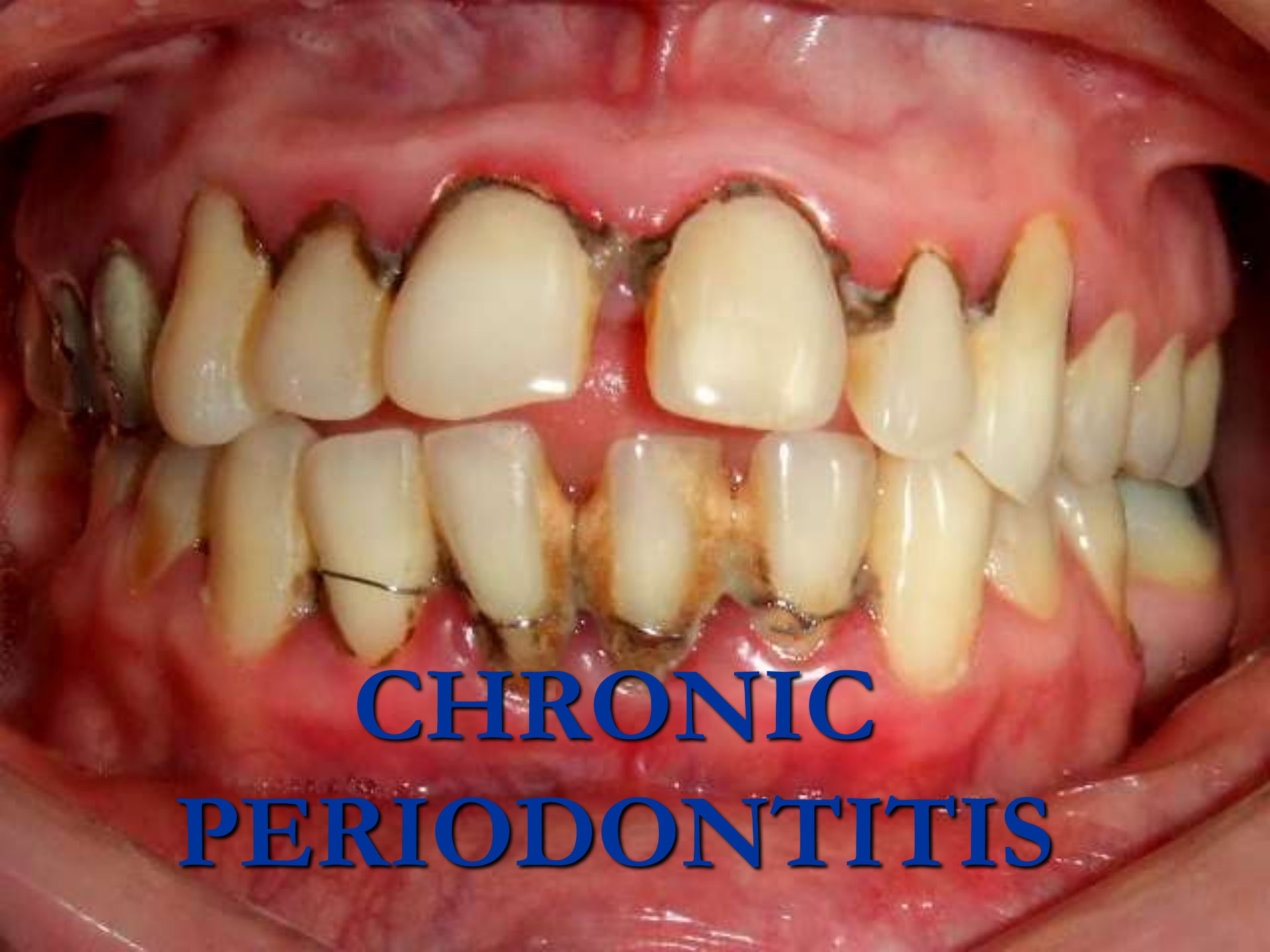

CHRONIC
PERIODONTITIS

- The ***Periodontium***, defined as those tissues supporting and investing the tooth, comprises root cementum, periodontal ligament, bone lining the tooth socket (alveolar bone), and gingiva.
- Periodontal disease comprises a variety of conditions affecting the health of the periodontium.

- The primary clinical features of periodontitis include clinical attachment loss (CAL), alveolar bone loss (BL), periodontal pockets, and gingival inflammation.
- In addition, marginal tissue recession, bleeding on probing, pathological migration, increased tooth mobility and tooth exfoliation may occur.

HISTORY

- The early English contribution to the understanding and management of periodontal diseases was made by ***John Hunter***, a physiologist and surgeon, widely known for his 1771 work on *The natural history of the human teeth*.

- *Riggs* placed the responsibility for the diseases of the gingiva and the bone on calculus deposits and other foreign bodies that made the tooth surface rough.
- Accordingly, he maintained that alveolar pyorrhea could be cured by meticulous removal of these accretions.

Classifying periodontal diseases – a long-standing dilemma

Over time, thoughts that guided the classification of periodontal diseases can be placed into three dominant paradigms :

1. primarily based on the clinical features of the diseases (1870–1920),
2. the concepts of classical pathology (1920–1970), and
3. the infectious etiology of the diseases (1970–present)

- ***Gottlieb*** is generally considered to be the first author who clearly distinguished various forms of periodontal disease.
- In the 1920s he classified periodontal disease into four types :
  - Schmutz- Pyorrhoe,
  - alveolar atrophy or diffuse atrophy,
  - Paradental-Pyorrhoe, and
  - occlusal trauma

- ***Orban & Weinmann*** adopted the nomenclature ***Periodontosis*** to designate non-inflammatory disease.
- In 1966 world workshop in periodontics, the term periodontosis was eliminated from periodontal nomenclature.

1989 World Workshop classification

I ***Adult periodontitis***

II ***Early onset periodontitis***

A Prepubertal periodontitis

1 Generalized

2 Localized

B Juvenile periodontitis

1 Generalized

2 Localized

C Rapidly progressive periodontitis

III ***Periodontitis associated with systemic diseases***

IV ***Necrotizing ulcerative periodontitis***

V ***Refractory periodontitis***

- 1999 classification is based on the Infection / Host Response paradigm that started to be the dominant paradigm in the 1970s.

Chronic periodontitis (Localized/Generalized)
Localized aggressive periodontitis
Generalized aggressive periodontitis
Periodontitis as a manifestation of systemic diseases Associated with hematologic disorders: Acquired neutropenia Leukemias Associated with genetic disorders Familial and cyclic neutropenia Down's syndrome Leukocyte adhesion deficiency syndromes Papillon-Lefèvre syndrome Chediak-Higashi syndrome Langerhans cell disease (histiocytosis syndromes) Glycogen storage disease Chronic granulomatous disease Infantile genetic agranulocytosis Cohen syndrome Ehlers-Danlos syndrome (Types IV and VIII) Hypophosphatasia Crohn disease (inflammatory bowel disease) Marfan syndrome
Necrotizing ulcerative periodontitis (NUP)
Abscesses of the periodontium
Combined periodontic–endodontic lesions

CHRONIC PERIODONTITIS

- Fleming in 1999 defined chronic periodontitis as "an infectious disease resulting in inflammation within the supporting tissues of the teeth , progressive attachment loss and bone loss."

Table 5. Main clinical features and characteristics of chronic periodontitis (1999 Classification) (10)

- Most prevalent in adults, but can occur in children and adolescents
- Amount of destruction is consistent with the presence of local factors
- Subgingival calculus is a frequent finding
- Associated with a variable microbial pattern
- Slow to moderate rate of progression, but may have periods of rapid progression
- Can be associated with local predisposing factors (e.g., tooth-related or iatrogenic factors)
- May be modified by and/or associated with systemic diseases (e.g., diabetes mellitus)
- Can be modified by factors other than systemic disease such as cigarette smoking and emotional stress

Table 8. Comparison of the main clinical characteristics of chronic periodontitis, localized aggressive periodontitis, and generalized aggressive periodontitis

Chronic periodontitis	Localized aggressive periodontitis	Generalized aggressive periodontitis
Most prevalent in adults, but can occur in children	Usually occurs in adolescents (circumpubertal onset)	Usually affects people under 30 years of age, but patients may be older
Slow to moderate rates of progression	Rapid rate of progression.	Rapid rate of progression (pronounced episodic periods of progression)
Amount of microbial deposits consistent with severity of destruction	Amount of microbial deposits *not* consistent with severity of destruction	Amount of microbial deposits sometimes consistent with severity of destruction
Variable distribution of periodontal destruction; no discernible pattern • No marked familial aggregation • Frequent presence of subgingival calculus	Periodontal destruction localized to permanent first molars and incisors • Marked familial aggregation. • Subgingival calculus usually absent	Periodontal destruction affects many teeth in addition to permanent first molars and incisors • Marked familial aggregation. • Subgingival calculus may or may not be present

Epidemiology

- Results from epidemiological cross-sectional surveys have demonstrated that periodontal disease seems to affect most subjects in any population.
- Severe periodontal tissue destruction, however, occurs in comparatively few subjects, i.e. 5 to 15% of any population, and not, as was previously believed, in the majority of Individuals above 40 years of age.

- Findings from longitudinal studies of different populations with untreated periodontal disease have revealed that progressive deterioration of the periodontal attachment apparatus seems to occur at an average rate of about 0.1 mm per tooth surface per year.
- The rate of progression of periodontal disease, however, varies considerably between diseased subjects.

Epidemiology
Longitudinal studies

- The **first group** established in Oslo, Norway, consisted of 565 healthy male academicians between 17 and 40 years of age. Members of the study population had maximum exposures to conventional dental care throughout their lives.

- **A second group**, established in Srilanka in 1970, consisted of 480 male tea laborers between 15 and 40 years of age. The workers had never been exposed to any programs relative to the prevention or treatment of dental diseases.

- The Norwegian group, as the members approached 40 years of age, had a mean individual loss of attachment of slightly above 1.5mm, and the mean annual rate of attachment loss was 0.08mm for interproximal surfaces and 0.1mm for buccal surfaces.
- As the Srilankans approached 40 years of age, the mean individual loss of attachment was 4.5mm, and the mean annual rate of progression of the lesion was 0.3mm for inter proximal surfaces and 0.2mm for buccal surfaces.

- This study suggests that without interference, periodontal lesions, progress at a relatively even pace, and this progress is continuous.
- Further analysis of the Sri Lankan Laborers showed that they were not all losing attachment at the same rate. Virtually, all gingival areas showed inflammation, but attachment loss varied tremendously.
- Based on interproximal loss of attachments and tooth mortality, three sub populations were identified:
  - Individuals with rapid progression (RP) of periodontal disease (8%)
  - individuals with moderate progression (MP) (81%), and
  - individuals who exhibited no progression of periodontal disease (NP) beyond gingivitis (11%).

- In short, periodontitis is an age associated and not an age related i.e. it is not the age of the individual that causes the increase in disease prevalence, but rather length of time that the periodontal tissue are challenged by chronic plaque accumulation.

Clinical features

- Supragingival and subgingival plaque accumulation (frequently associated with calculus formation)
- Gingival inflammation
- Pocket formation
- Loss of periodontal attachment
- Loss of alveolar bone
- Occasional suppuration

- Most prevalent in adults, but can occur in children and adolescents.
- Amount of destruction is consistent with the presence of local factors.
- Subgingival calculus is a frequent finding
- Associated with a variable microbial pattern

- Serum ,neutrophil or monocyte abnormalities are usually not present.
- The extent and distribution of bone loss are highly variable , both horizontal and vertical pattern may be seen.
- Slow to moderate rate of progression, but may have periods of rapid progression.

- Can be associated with local predisposing factors (e.g. tooth related or iatro genic factors).
- May be modified by an/or associated with systemic diseases (e.g. Diabetes Mellitus, HIV infection)
- Can be modified by factors other than systemic disease such as cigarette smoking and emotional stress.

Signs

- In patients with poor oral hygiene, gingiva is moderately swollen and exhibits color changes ranging from pale red to magenta.
- Loss of gingival stippling
- Blunted or rolled gingival margins
- Flattened or cratered interdental papillae

- In some cases, probably as a result of long standing, low grade inflammation, thickened, fibrotic marginal tissues may obscure the underlying inflammatory changes.

Symptoms

- Bleeding gums while eating or brushing
- Loose teeth
- Occasionally pain in the absence of caries due to exposed roots that are sensitive to heat and cold.
- Areas of food impaction
- Sometimes deep radiating pain into the jaw
- Gingival tenderness or itchiness
- Acute pain may occur due to periodontal abscess

Radiographic Features

- Radiographically, deposits of calculus may be seen in patients with gross neglect.
- On periapical and bitewing radiographs, early lesions of the bone include development of cuplike lesions interproximally, with bone loss seen at the crest of the interproximal alveolar process, not yet involving the lamina dura.
- In more advanced cases reduced crestal height and loss of lamina dura occur.

- The extent and distribution of bone loss are highly variable; both vertical and horizontal patterns may be seen.
- Bone loss can also be seen between roots in furcations.

Disease distribution

- Site specific disease.
- Clinical signs i.e. inflammation, pocket formation ,attachment loss, and bone loss are due to the direct site specific effects of subgingival plaque.
- Can be described as

  localized: ≤ 30% of the sites are affected

  generalized: >30% of the sites are affected.

Disease severity

Mild Periodontitis:

- Characterized by probing attachment loss no more than 1-2 mm, minimal furcation invasions, and little tooth mobility.
- Supra and subgingival plaque are present, along with various amounts of calculus.
- Bleeding on gentle probing is commonly seen. Radiographic evidence of bone loss is minimal (usually less than 20% of the total attachment)
- This stage of involvement can be localized to several teeth or generalized to many areas throughout the mouth.

Moderate periodontitis:

- Patient exhibit 3-4mm of probing attachment loss early to moderate furcation invasions, and slight to moderate tooth mobility.
- Radiographically evident bone loss is usually horizontal and may consist of up to 40% of the total possible periodontal attachment on the tooth.
- Furcation radiolucencies may be evident.
- Bleeding on probing is a frequent finding and purulence may be seen.

Severe Periodontitis:

- Probing attachment loss of 5mm or more with significant furcation invasions, often through and through is found.
- Excessive tooth mobility is commonly found.
- Radiographic bone loss exceeds 40% and angular bony defects are seen.
- Purulent exudate can be present, along with bleeding on probing.

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.
- More rapidly progressive lesions occur most frequently in interproximal areas and are usually associated with areas of greater plaque accumulation and inaccessibility to plaque control measures.

Models of change of periodontal status

- Several models of disease progression have been proposed to explain temporal patterns of tissue destruction.
- Historically, it was believed that periodontitis resulted in a slow, continuous and progressive destruction of the periodontium.
- Subsequent clinical studies suggested that disease progression occurs as episodic bursts of activity with periods of remission – **EPISODIC BURST THEORY**.

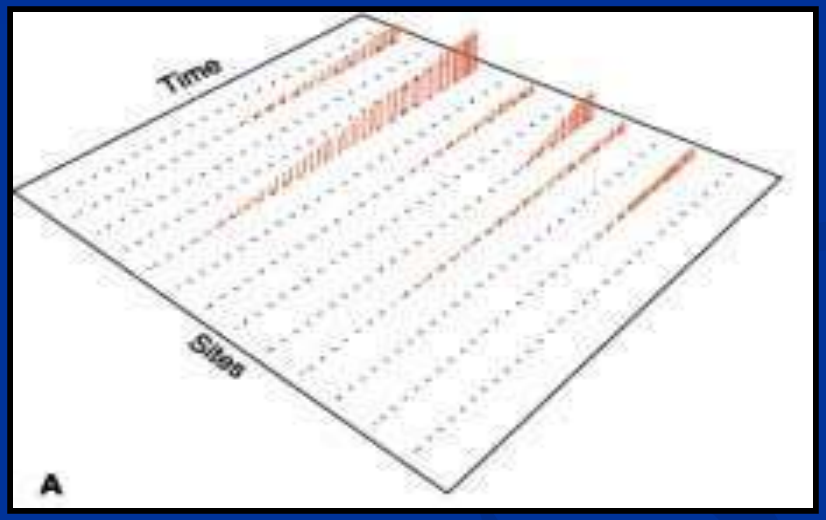
The various models of disease progression are:

Continuous model (Socransky et al 1984):

- Slow, steady, progressive disease process.

Episodic burst theory (Goodson et al; 1982; Zimmerman 1986):

- Irregular periods of exacerbation and remisson.

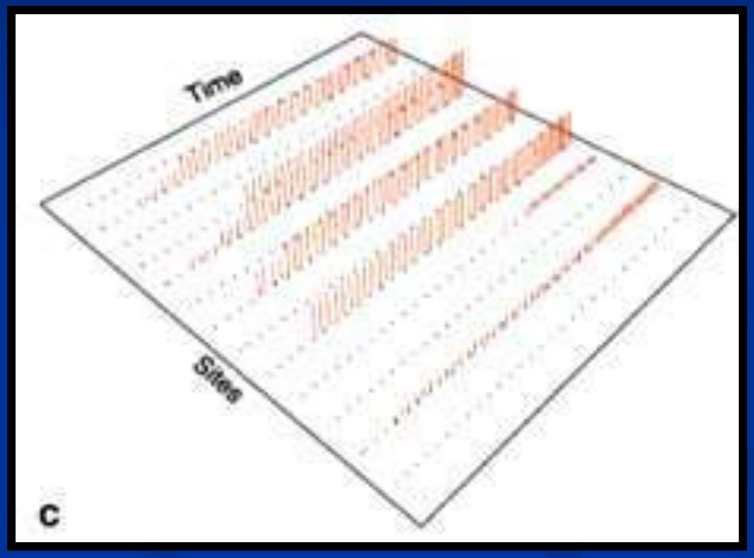


Asynchronous burst theory (Socransky et al 1984):

- Periods of exacerbation and remission during defined period.

Epidemiologic model (Cohen et al 1988)

- Consistent with continuous disease aging process that depends only on the duration of the process



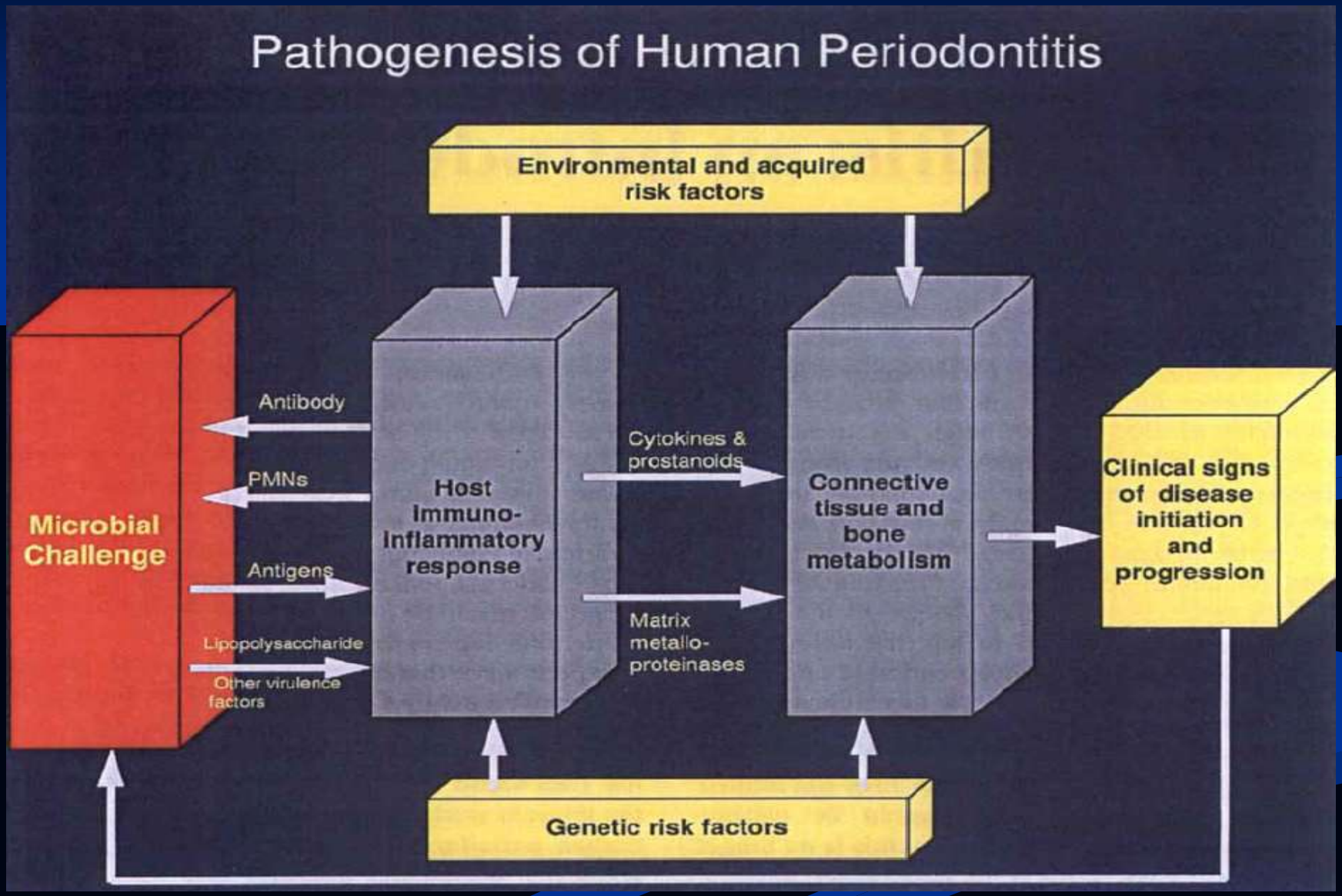
Factors that may cause the conversion of gingivitis to periodontitis

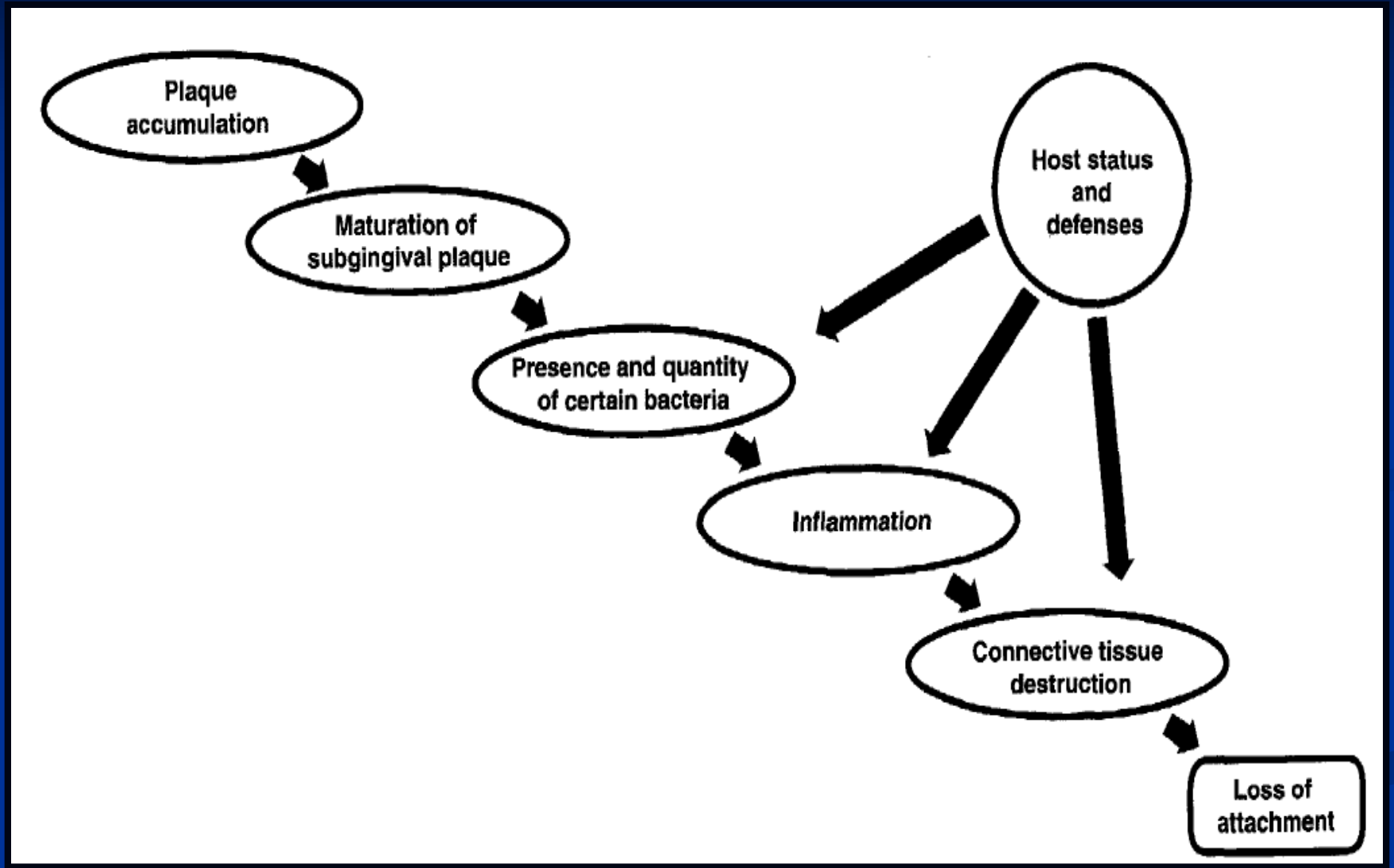
- There are two main theories on the role of plaque bacteria in periodontal disease: the specific and the nonspecific plaque hypothesis.
- The ***specific plaque hypothesis*** is that a very limited number of organisms are actively involved in causing disease.
- The ***nonspecific plaque hypothesis*** states that many of the heterogeneous mixture of organisms in plaque play a role in disease.

- ***Marsh*** challenged the dichotomy between the theories " ... Since plaque-mediated diseases, while not necessarily having a totally specific etiology, do show evidence of specificity". He proposed a unifying "ecological plaque hypothesis".
- This hypothesis proposes that a change in a key environmental factor triggers a shift in the balance of the resident plaque microflora. Potentially pathogenic organisms are weakly competitive and may be suppressed by intermicrobial antagonism when there is gingival health.

Pathogenesis of Periodontitis

- Periodontitis has a multifactorial etiology.
- Nevertheless, a common underlying chain of events in the pathogenesis is shared by all forms of the disease.
- This common chain of events is influenced by other factors including genetic and other risk factors that may differ from one form of the disease to another.


Pathogenesis of Human Periodontitis
Environmental and acquired risk factors
Microbial Challenge
Antibody
PMNs
Antigens
Lipopolysaccharide
Other virulence factors
Host immuno-inflammatory response
Cytokines & prostanoids
Matrix metallo-proteinases
Connective tissue and bone metabolism
Clinical signs of disease initiation and progression
Genetic risk factors


Plaque accumulation
Maturation of subgingival plaque
Presence and quantity of certain bacteria
Inflammation
Connective tissue destruction
Loss of attachment
Host status and defenses

Risk factors for periodontitis

- The term risk factor refers to an aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic, which on the basis of epidemiological evidence is known to be associated with a health-related condition.

Age

- Both the prevalence and

  severity of periodontal

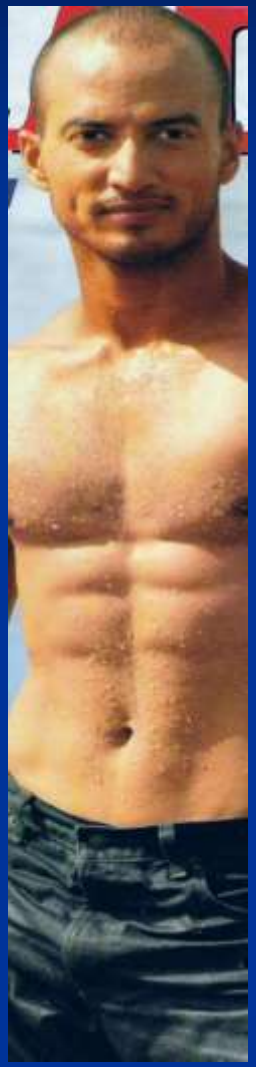
  disease increases

  with age.

CP is age associated and not age related.

Gender

Gender plays a role in periodontal disease



- Periodontal disease …..more prevalent or more severe in men than in women at comparable ages as researched by
- United States national survey have shown that males have poorer oral hygiene


RACE

- Beck and coworkers showed that blacks more susceptible to periodontitis than whites.
- ***P. intermedia*** was a risk factor for blacks but not for whites.
- When blacks and whites belonged to the same socioeconomic group, differences in periodontal disease often disappeared.

Socioeconomic Status

- ***Ramfjord et al*** in a nutritional survey of 700 men in Alaska

Lower socioeconomic status:

- Gingivitis
- Poor oral hygiene
- Decreased dental awareness
- Decreased frequency of dental visits

Lower SES alone does not result in increased risk for periodontitis

Stress

- Necrotizing ulcerative gingivitis and stress,
- Emotional stress may interfere with normal immune function and may result in increased levels of circulating hormones that can have an impact on the periodontium.

- Individuals with financial strain, distress, depression, or inadequate coping mechanisms have more severe loss of attachment.
- Adult patients with periodontitis who are resistant to therapy are more stressed than those who respond to therapy.

Genetic Factors

- Studies by ***Michalowicz*** in twins have shown that genetic factors influence clinical measures of:

1. Gingivitis,
2. Probing pocket depth,
3. Attachment loss, and
4. Interproximal bone height.

- The familial aggregation seen in localized and generalized aggressive periodontitis also is indicative of genetic involvement in these diseases.
- A specific interleukin- 1 (IL-1) genotype has been associated with severe chronic periodontitis as shown by.

- Immunologic alterations, such as:
- Neutrophil abnormalities,
- Monocytic hyperresponsiveness to LPS stimulation in patients with LAP and
- Alterations in the monocyte / macrophage FcγRII receptor for IgG2 also appear to be under genetic control.
- HLA-DR4- …EOP

HLA-DRB1*1501; HLA-DQB1*0602… accelerated T cell response to *P.g* and susceptibility to EOP in Japanese .

- Negative associations of periodontitis have been reported for HLA-A2.
- HLA- A9 and B15 have a strong association .
- Reports suggest an increase of HLA-A9, as well as HLA-28 and HLA-WI5, in LJP patients.

Tobacco Smoking

- Cigarette smoke is a very complex mixture of substances with over 4000 known constituents.
- These include:
  - CO, HCN,
  - Reactive oxidizing radicals,
  - Carcinogens
  - Main psychoactive and addictive molecule - **nicotine**
- Many of these components could modify the host response in periodontitis.

NECROTISING ULCERATIVE INFECTIONS

- Necrotizing ulcerative gingivitis
- Acute herpetic gingivostomatitis

NECROTIZING ULCERATIVE GINGIVITIS (NUG)

- Introduction
- Prevalence and epidemioloogy
- Clinical features
- Histopathology
- Etiology
- Diagnosis

SYNONYMS

- Acute ulceromembraneous gingivitis
- Trench mouth
- Ulcerative gingivitis
- Vincent's stomatitis
- Plant-Vincent's stomatitis
- Stomatitis ulcerosa
- Fusospirillary gingivitis
- Fetid stomatitis
- Putrid stomalitis
- Acute septic gingivitis
- Pseudomembranous angina
- Spirochetal stomatitis

NECROTIZING ULCERATIVE GINGIVITIS (NUG)

INTRODUCTION :

NUG has been described in the world workshop in clinical periodontics as "rapidly destructive non-communicable gingival infection of complex etiology."

In 1890's Plant and Vincent attributed its origin to fusiform bacilli and sprochetes

PREVALENCE :

It is primarily a disease of young adults in the age group of 18-30 years.

EPIDEMOLOGY :

Frequently occurs in epidemic pattern seeping through groups or persons in close contact.

It was especially apparent during World War I, among so the term "trench mouth" was coined.

CLINICAL FEATURES

1. HISTORY :

- Characterized by Sudden onset, sometimes following an episode of debilitating disease/acute respiratory tract infection.
- Changes in living habits, psychological stress.
- Patient also gives history of soreness and bleeding resulting from pressure in involved area during eating and tooth brushing.

2. ORAL SIGNS AND SYMPTOMS

SIGNS :

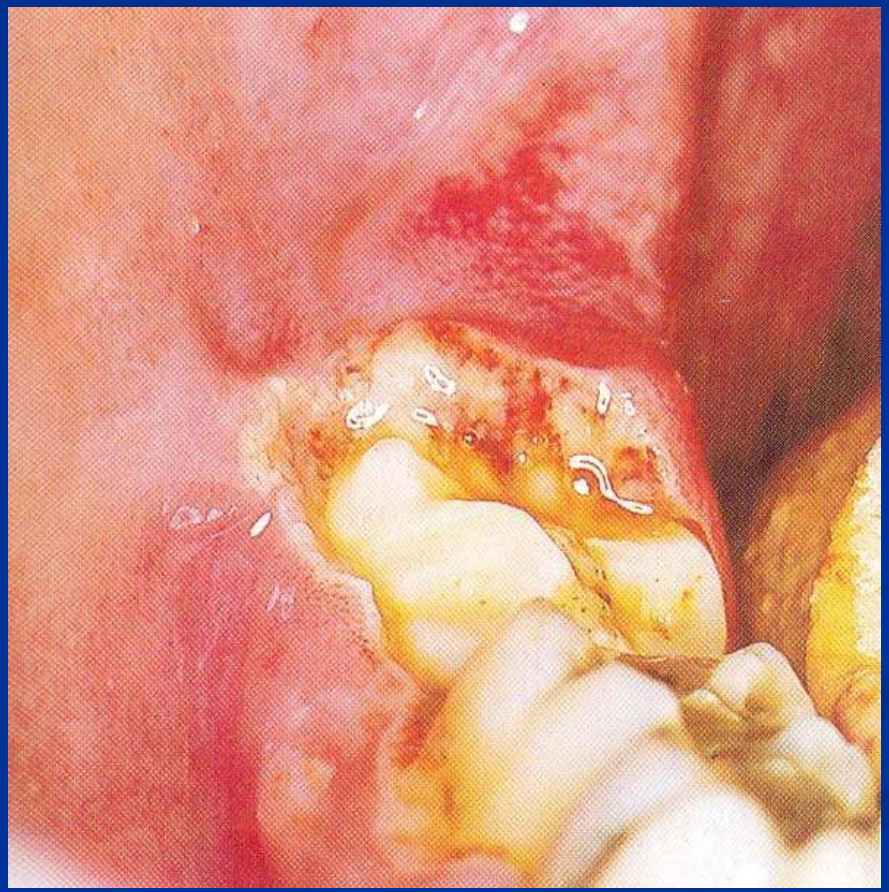
- Punched out crater like depressions, at the crest of interdental papillae, - extending to marginal gingival.
- Surface of gingival crater – covered by gray, pseudomembranous slough.
- Crater is demarcated from the remainder by a linear erythema.
- Cases devoid of pseudomembrane, appears as red, shiny and hemorrhagic.
- Progressively destroy gingival and periodontal tissue (Recurrent NUG-NUP)
- Gingival hemorrhage, pronounced bleeding on slightest stimulation.
- Fetid odour and increased salivation.
- Rare in edentulous mouth.

CLINICAL PICTURES OF NUG

Picture showing early stage of nug

Note the necrosis and desquamation of papilla in nug

NUG AFFECTING GINGIVA IN 3RD MOLAR REGION



SYMPTOMS

- Lesions extremely sensitive to touch
- Constant radiating gnawing pain – intensified by spicy or hot foods and chewing
- Metallic foul taste
- Increased amount of pasty saliva

SYSTEMIC SIGNS &SYMPTOMS

- Local Lymphadenopathy and slight increase in temperature – mild and moderate type
- In severe cases – high fever, increased pulse rate, leukocytosis, loss of appetite, generalized lassitude.
- In children – insomnia, constipation, GIT disorders, headache and mental depressions.
- In rare cases –
  - Noma
  - Fusospirochetal meningitis
  - Fatal brain abscesses
  - Pulmonary infections
- Toxemia

CLINICAL COURSE :

- Indefinite, - progressive destruction of periodontium and denudation of roots ..
- Spontaneous remission / repeated remissions and exacerbations.

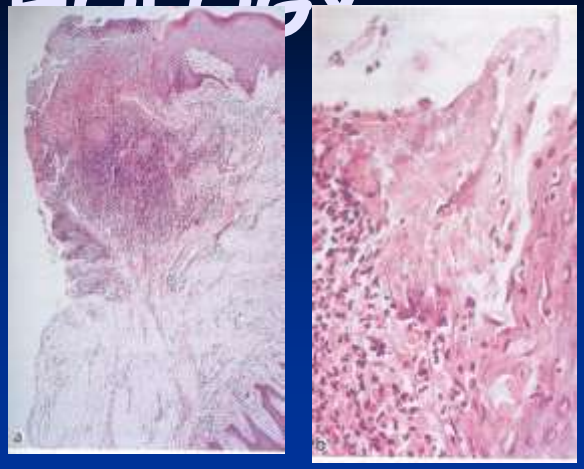
HISTOPATHOLOGY

Microscopically the lesion appears as nonspecific acute necrotizing inflammation at gingival margin involving stratified squamous epithelium and the underlying connective tissue.

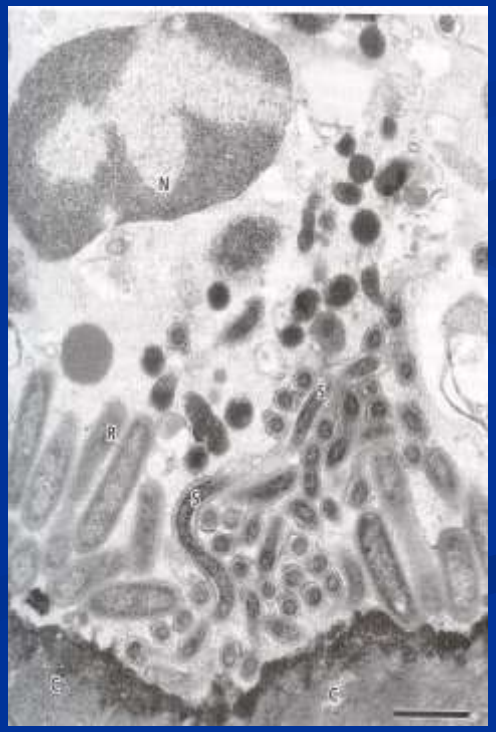
EPITHELIUM :

Surface epithelium is destroyed → replaced by pseudomenmbranous mesh work of fibrin; necrotic epithelial cells polymorph nuclear neutrophils and various types of microorganisms.

This zone appears as surface pseudomembrane.

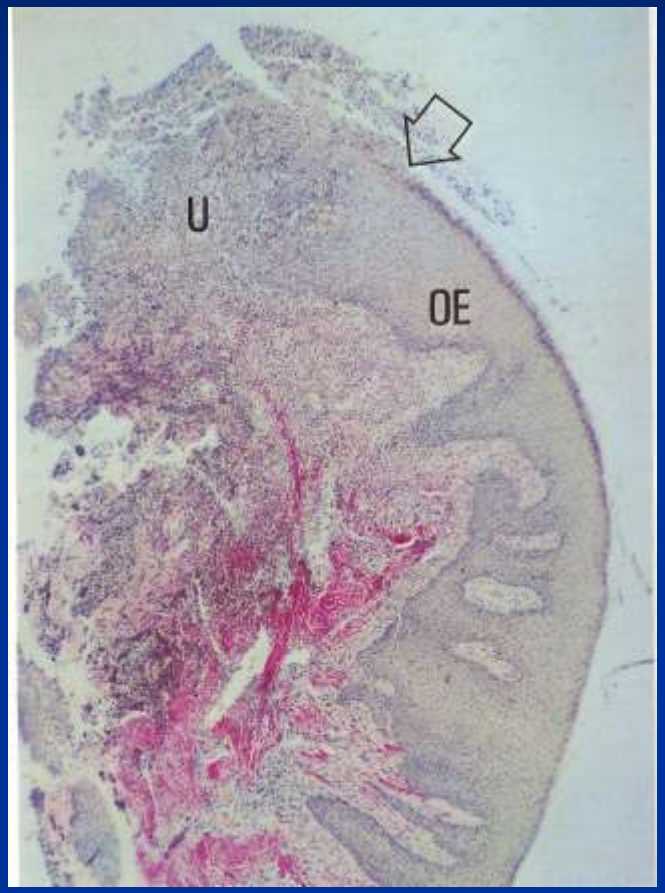


Photomicrograph of gingival tissue affected by ANUG. Upper left ulcerated surface .Underneath is connective tissue infiltrated with inflammatory cells



Electron micrograph demonstrating phagocytosing neutrophil (N) close to the surface of a seqesterum (C) covered by numerous microorganisms ,including spirochetes (S) and rods (R)

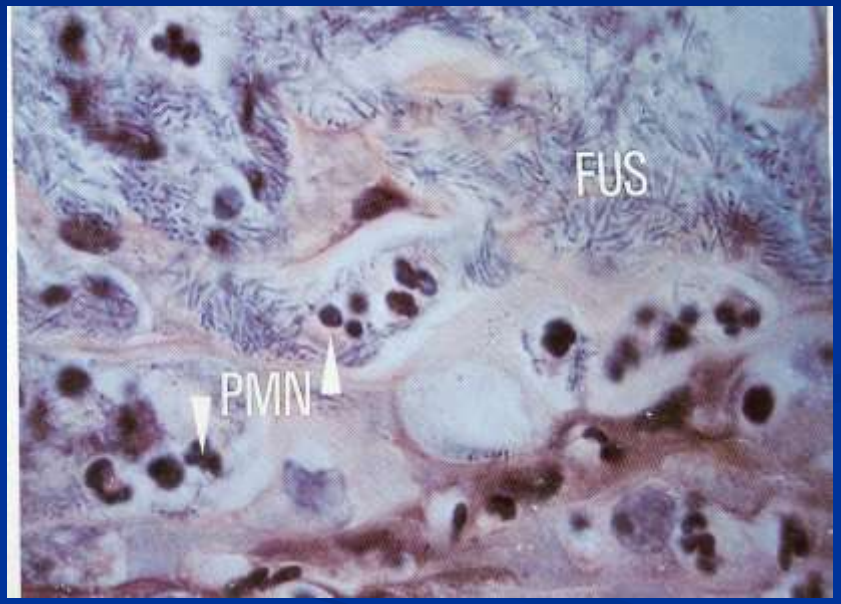
PAPPILA BIOPSY



Papilla biopsy from a patient with ANUG. The tip of the papilla and the tissue approaching the col have been destroyed by ulceration (U).The oral epithelium (OE) is relatively intact.In deeper layers of biopsy , observe the red stained intact collagen ,while beneath the decimated papilla tip collagen is largely obliterated

CONNECTIVE TISSUE :

- Marked by hyperemia with numerous engorged capillaries and a dense infiltration of PMN's.
- The acutely inflamed hyperaemic zone appears clinically as linear erythema, beneath the surface pseudomembrane.
- There is a gradual blend of the epithelium and connective tissue from the involved gingiva to necrotic lesion.



Surface of disintegrated tissue , note the PMN'S and Fusiformbacilli

ROLE OF BACTERIA

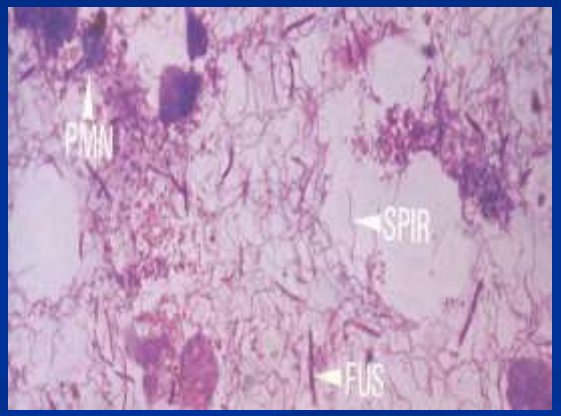
Light and electron microscope studies done to study the relationship between ANUG lesion and bacteria.

LIGHT MICROSCOPE STUDIES :

Exudate on the surface of the necrotic lesion – cocci, fusiform bacilli and spirochetes.

Layer between necrotic and living tissue – enormous number of fusiform bacilli and spirochetes + leukocytes and fibrin.

Spirochetes and other bacteria invade underlying tissue (living).



Bacteriologic smear of psuedomembr-ane

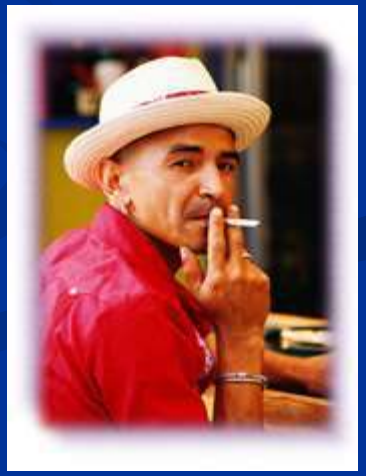
ELECTRON MICROSCOPY :

Listgarten in his study described 4 zones, observed in gingiva.

- Zone I: Bacterial zone :
- Most superficial, consists of varied small bacteria and small, medium and large spirochetes.
- Zone II : Neutrophil-rich zone :
- Contains leukocyte especially neutrophils with bacteria including many spirochetes of various types between the leukocytes.
- Zone 3 : Necrotic zone
- Disintegrated tissue cells, fibrillar material, remmants of collagen fibers, intermediate and large spirochetes and other bacteria
- Zone 4 : Zone of spirochetal infiltration
- Well preserved tissue infiltrated with intermediate and large spirochetes without other organisms.

CIGARETTE SMOKING

- Local and systemic catecholamines released in response to nicotine from smoking cause a reduction in gingival papillary blood flow → contributes to ischaemia in periodontal tissue.
- Decreased blood flow results in deficient host defense and papillary necrosis.



ETIOLOGY :

The specific etiology of the ANUG has not been established -

Fusobacterium necrophrom

ROLE OF BACTERIA :

Plant and Vincent produced the concept that ANUG is caused by specific bacteria namely fusiform bacillus and spirochetes organisms.

Porphyromonas gingivalis

Others-bacteroides melaninogenicus subsp intermedius. Fusobacterium, treponema macrodentium, actinomyces odontolyticus and selemonas species.

Prevotella intermedia

SYSTEMIC PREDISPOSING FACTORS :

- Systemic disease
- Nutritional deficiency
- Emotional stress

SYSTEMIC DISEASES

- Systemic disease involving depression in function and or number of leukocytes and immune suppression e.g. malignancy, systemic lupus erythematosus, agranulocylosis, AIDS.
- Viral infections → cytomegalovirus

NUTRIONAL DEFICIENCY

- Has a conditioning effect on bacterial pathogenecity.
- Malnutrition especially vitiman A, ascorbic acid → implicated in etiology of NUG

EMOTIONAL STRESS :

Stress → increased adrenocortical activity → inability to control indigenous bacteria.

Stress → increased epinephrine levels → localized ischemia of gingiva → NUG.

Psychiatric disturbance + stress

↓

activation of HPA axis

↓

increase serum and urine cortisol levels

↓

depression of lymphocyte and PMN function NUG (predisposition)

DIAGNOSIS

- Interproximal necrosis and ulceration
- Painful gingival
- Bleeding (spontaneous or on slight provocation)
- Pseudomembrane (fibrin, debris)
- Fever, malaise, lymphodenopathy
- Foeter ories

- CONCLUSION …


THANK YOU