Diseases of the periodontium

Dr. Madhusudhan reddy

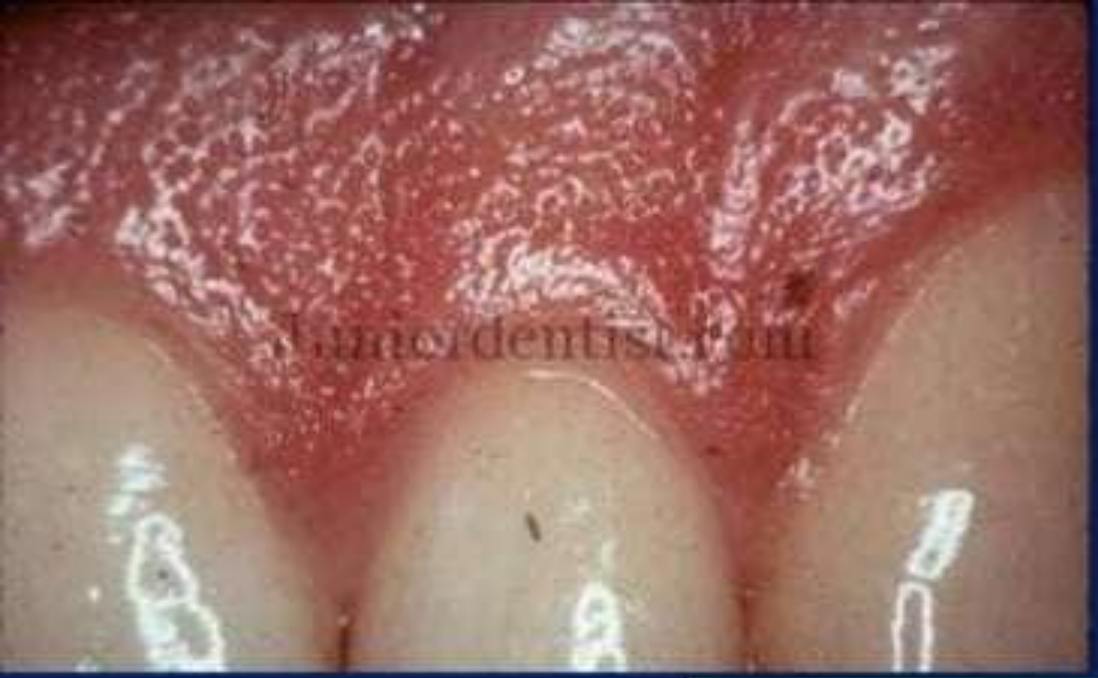
The Periodontium comprises:-

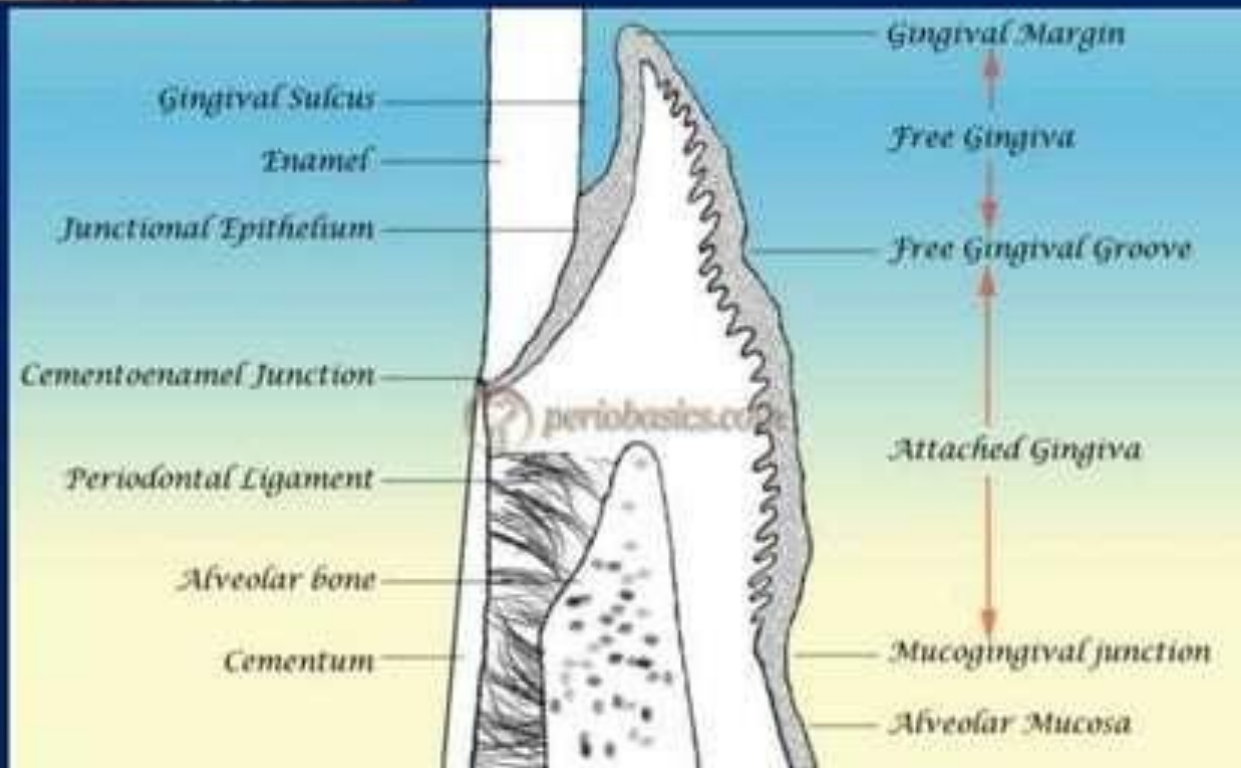
- Gingiva
- Periodontal Ligament (PDL)
- Root Cementum
- Alveolar Bone

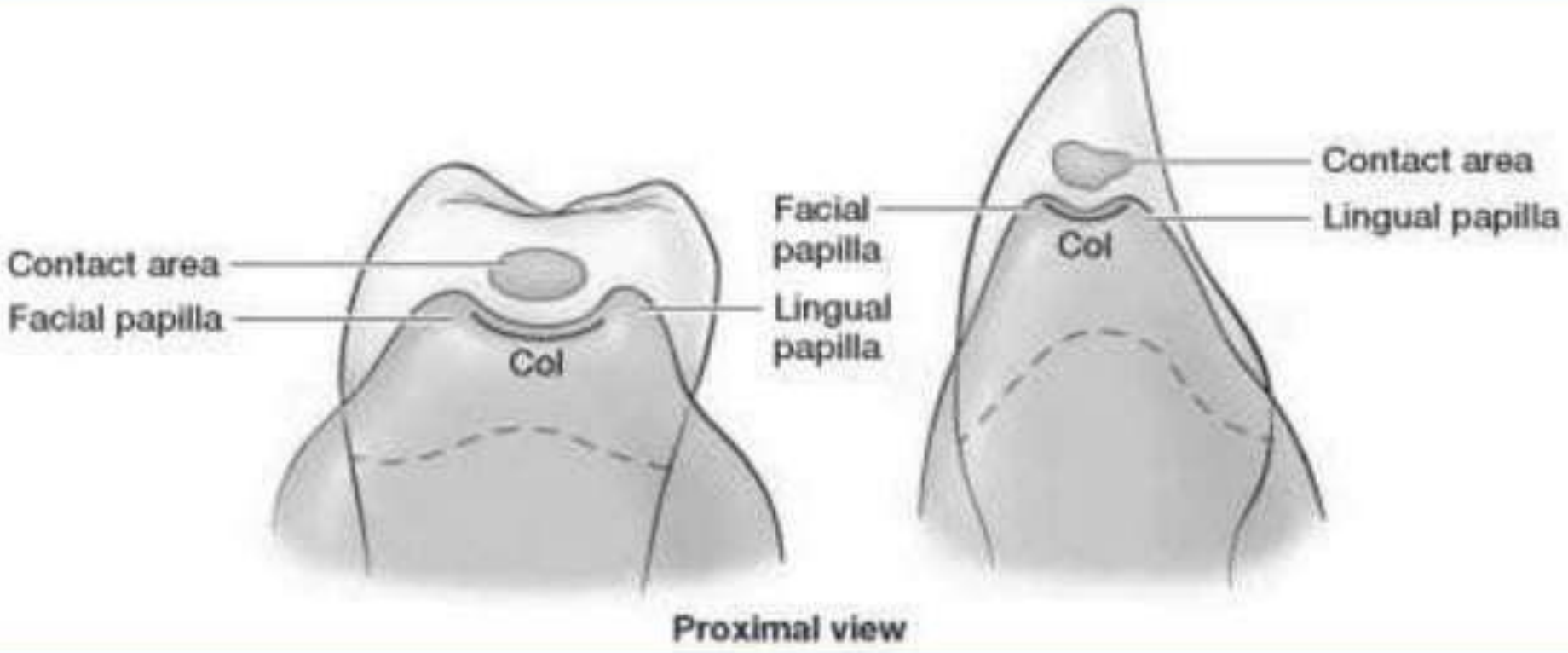
- The main function of the periodontium is to attach the tooth to the bone tissues of the jaws and to maintain integrity of the surface of the masticatory mucosa of the oral cavity

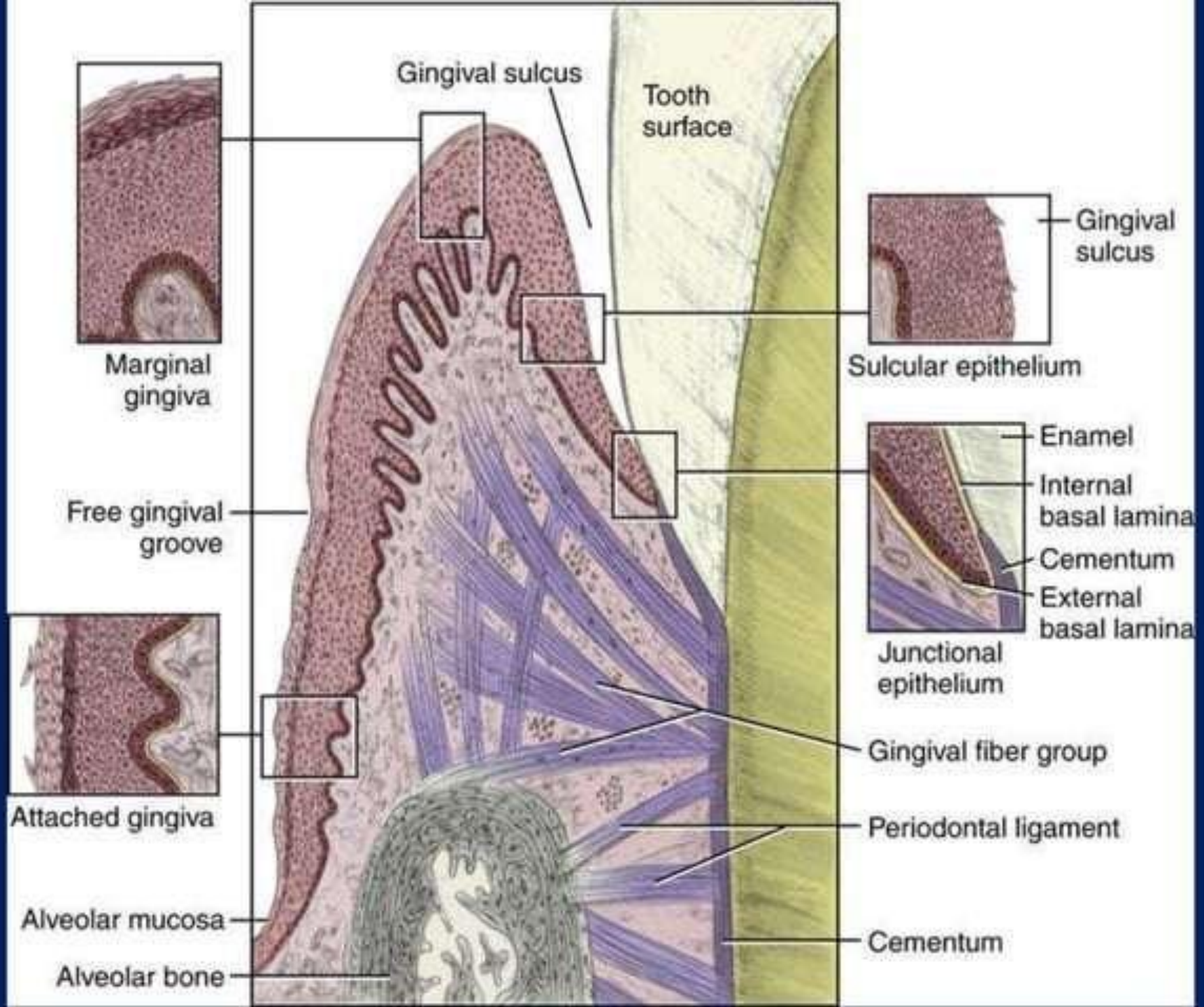
Healthy periodontium

- Healthy gingiva: Fits snugly around the teeth, filling each inter proximal space between the contact area
- Color : Coral pink
- Consistency: Firm
- Stippling (orange peel appearance)



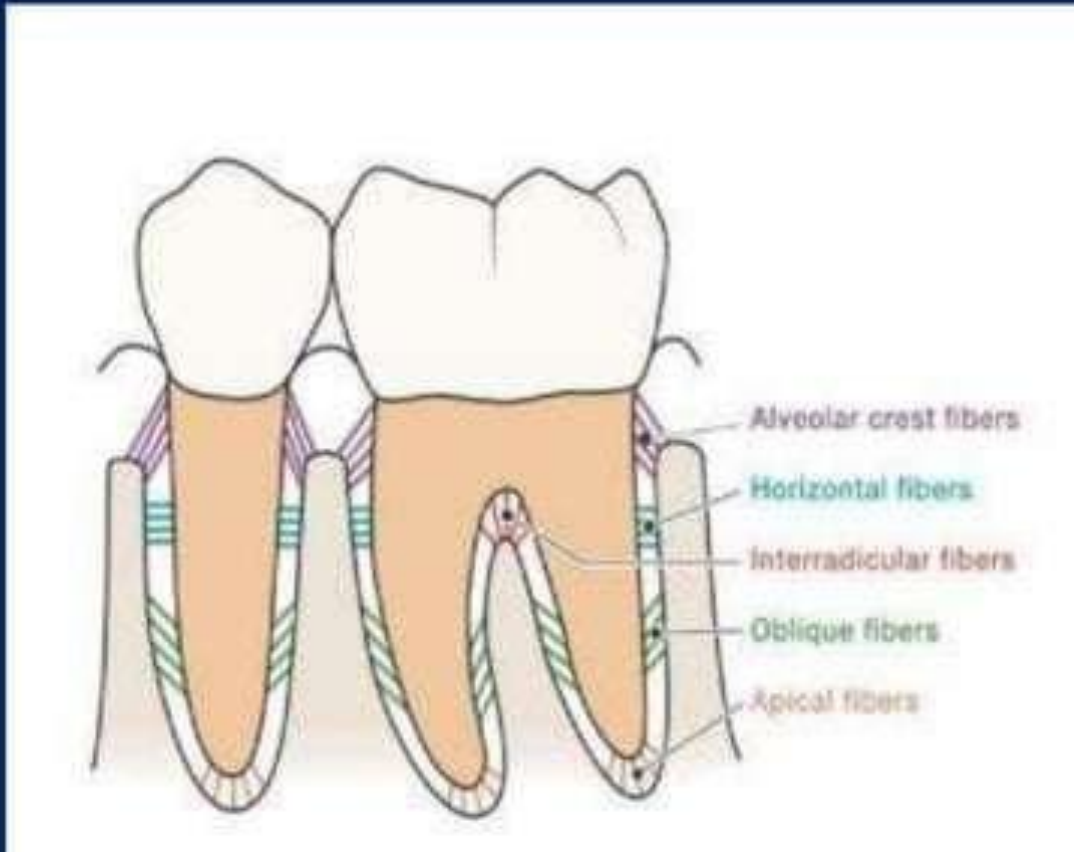

Gingival Margin
Free Gingiva
Free Gingival Groove
Attached Gingiva
Mucogingival junction
Alveolar Mucosa
Gingival Sulcus
Enamel
Junctional Epithelium
Cementoenamel Junction
Periodontal Ligament
Alveolar bone
Cementum


Contact area
Facial papilla
Col
Lingual papilla
Facial papilla
Col
Contact area
Lingual papilla
Proximal view

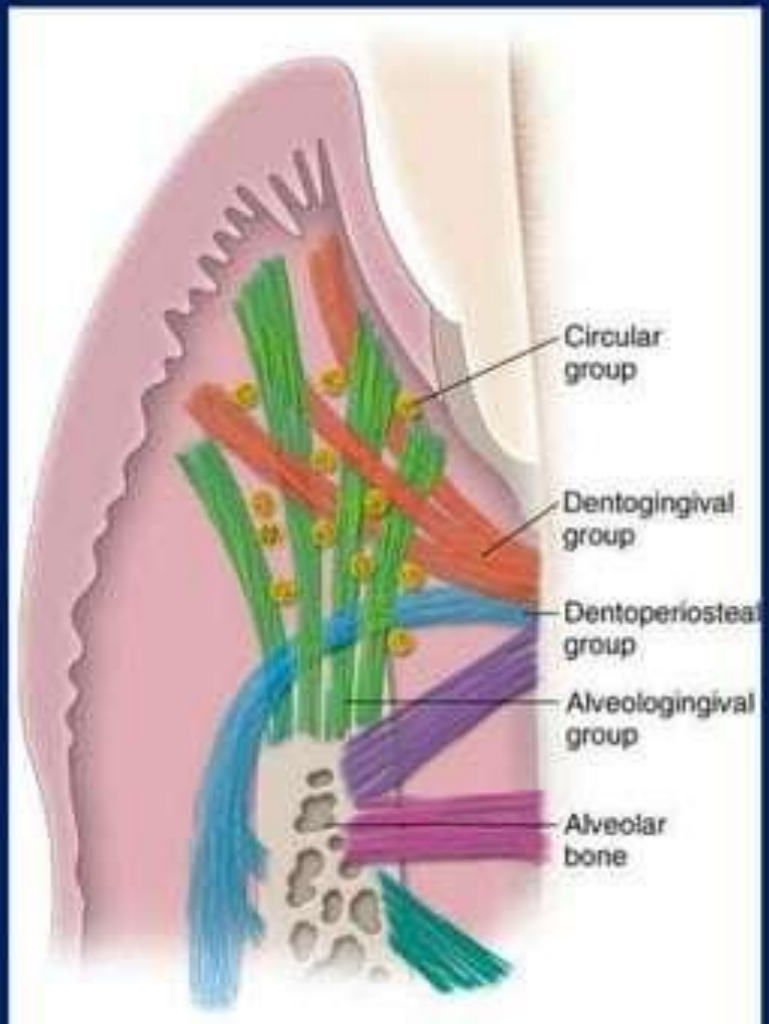

Gingival sulcus
Tooth surface
Marginal gingiva
Gingival sulcus
Sulcular epithelium
Free gingival groove
Enamel
Internal basal lamina
Cementum
External basal lamina
Junctional epithelium
Attached gingiva
Gingival fiber group
Periodontal ligament
Alveolar mucosa
Cementum
Alveolar bone

- Periodontal ligament is made up of
  - Collagen fibers
  - Oxytalan fibers
  - Fibroblasts
  - Amorphous ground substance & interstitial tissue
  - Cementoblasts
  - Osteoblasts
  - Osteoclasts
  - Epithelial rests of Malassez
  - Thin walled blood vessels
  - Lymphatic vessels
  - Sensory nerves

- PDL fibers are attached to the tooth cementum on one side and to alveolar bone on another side
- They are arranged in the groups - Principal fibers of PDL
- The grouped fibers are as follows
  - Alveolar group
  - Horizontal group
  - Oblique group
  - Apical group
  - Inter radicular group



- Gingival fibers of periodontal ligament
- These enter the lamina propria and attach the gingiva firmly to the tooth
- They are divided into 4 groups
  - Dentogingival
  - Alveologingival
  - Circular group
  - Dentoperiosteal



- Cementum is the outer covering of the root which is avascular
- Homogenous matrix comparable with bone
- Laid down on the tooth root throughout the life

Deposits on teeth

- **Organic coverings of tooth enamel are divided in to 2 types**
  - **Anatomic structures (Nasmyths membrane)**
  - **Acquired pellicle**

- *Nasmyths membrane (enamel cuticle)*
- **Anatomical covering formed during the developmental and eruptive stages of teeth**
- **Remnants of this membrane persists throughout the life**

- Pellicle
- **Thin deposit on the exposed surface of the teeth**
- **Shortly formed after eruption**
- **Usually invisible**
- **Fully formed in 30 mins and reaches its mature thickness of 0.1 – 0.8 microns within 24 hrs**
- **Free of bacteria and covers the tooth surface**
- **Its of salivary origin composed of mucoproteins or glycoproteins**

Dental stains

- Pigmented deposits on the tooth surface
- Exogenous and Endogenous stains
- Endogenous stains: incorporated into tooth structure eg: porphyria, erythroblastosis fetalis, tetracyclin
- Exogenous stains: oral chromogenic bacteria, stains from tobacco smoking,

Calculus

- Hard stone like deposit on teeth or prosthetic appliance in the oral cavity
- Odontolithiasis, tartar
- Mineralized dental plaque

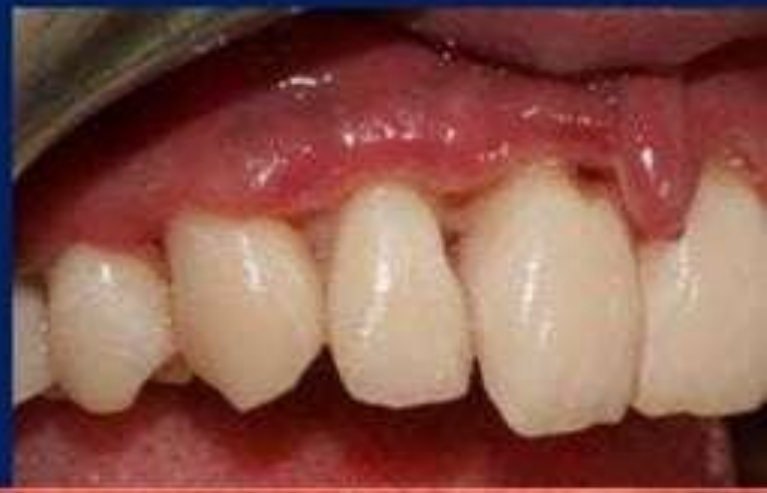
CLASSIFICATION OF PERIODONTAL DISEASE

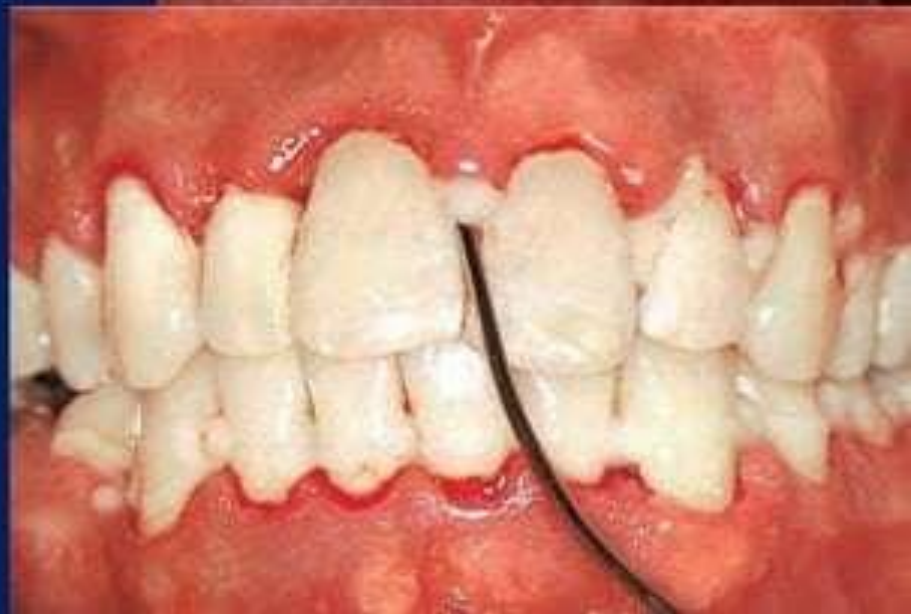
- Gingival diseases
  - –Plaque induced gingival disease
  - –Non plaque induced gingival lesions
- Chronic periodontitis
  - –Localized
  - –Generalized
- Aggressive periodontitis
  - –Localized
  - –Generalized
- Periodontitis as a manifestation of systemic disease
- Necrotizing periodontal disease
  - –Necrotizing ulcerative gingivitis (NUG)
  - –Necrotizing ulcerative periodontitis (NUP)

- Abscesses of periodontium
  - –Gingival abscess
  - –Periodontal abscess
  - –Pericoronal abscess
- Periodontitis associated with endodontic lesions
  - –Endodotic – periodontal lesion
  - –Periodontal – endodontic lesion
  - –Combined lesion
- Developmental or acquired deformities and conditions
  - –Localized tooth related factors that predispose to plaque induced gingival diseases or periodontitis
  - –Mucogingival deformities and conditions around teeth
  - –Mucogingival deformities and conditions on edentulous ridges
- Occlusal trauma

Necrotizing ulcerative gingivitis

- **Also known as**
  - **Vincent's infection**
  - **Trench mouth**
  - **Acute ulceromembranous gingivitis**
  - **Phagedenic gingivitis**
  - **Fusospirochetal gingivitis**
  - **Acute ulcerative gingivitis**





Necrotizing ulcerative gingivitis

- Specific type of gingivitis with characteristic signs and symptoms
- Disease manifests as both acute and recurrent (subacute) phases
- inflammatory condition involves primarily
  - Free gingival margin,
  - Crest of the gingiva,
  - Interdental papillae
- When it spreads to the soft palate and tonsillar areas: Vincent's angina
- Diagnostic triad: Pain, interdental ulceration, and gingival bleeding

- Epidemiology
- Frequently occurs in an epidemic pattern
- Groups of persons in close contact, especially those living under similar conditions
- Apparent during World War I, term 'trench mouth': troops in the trenches
- Similar sporadic outbreaks also occurred during World War II
- Relatively uncommon in developed countries nowadays
- Global increase associated with HIV infection.

- Epidemiology
- **Not a contagious infection: similar predisposing conditions among the members of the group**
- **At any age, but is more common among young and middle-aged adults**
- **In developing countries, it is seen almost exclusively in children, related to poverty and malnutrition**

- Etiology
- **Endogenous, polymicrobial infection causing destructive inflammation due to the coexistence of several predisposing factors**
- **Fusiform bacillus and Borrelia vincentii (a spirochete): present in small numbers in the healthy gingival flora**
- **Some times vibrio and coccal forms**
- **Number of factors disturb the host-parasite relationship, facilitating overgrowth of the organisms of the fuso-spirochaetal complex**
- **Increase in IgG and IgM antibody titers to spirochetes and increased IgG titers to Bacteroides melaninogenicus**

- Predisposing Factors
- **Psychological stress** plays an important role - increased frequency of the disease in people in the **military services**
- Other predisposing factors include
  - –Immunosuppression
  - –Smoking
  - –Upper respiratory tract infection,
  - –Local trauma,
  - –Poor nutritional status
  - –Poor oral hygiene.

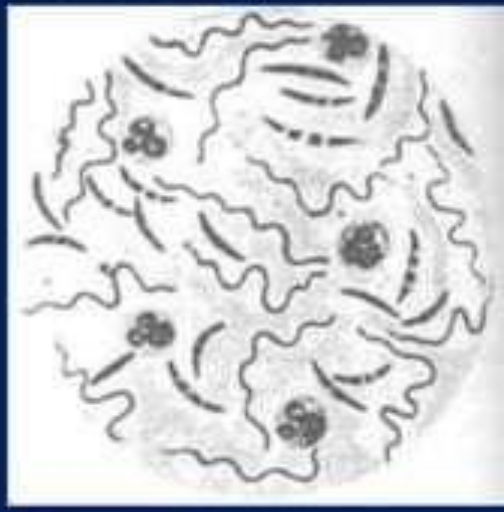
- Predisposing Factors
- **HIV-positive persons suffer from a severe form of disease ultimately HIV associated periodontitis**
- **Highest incidence occurring between October and February: respiratory infections and exanthemas are at their peak**
- **Lowest incidence occurring in July and August**
- **Deficient in vitamin C and B complex**

- Clinical Features
- **Painful, hyperemic gingiva and sharply punched-out crater like erosions of the interdental papillae of sudden onset**
- **Ulcerated remnants of the papillae and the free gingiva bleed when touched**
- **Become covered by a grayish green, necrotic pseudomembrane**
- **Ulceration tends to spread and may eventually involve all gingival margins**
- **It begins rather commonly at a single isolated focus, with a rapid onset**

- Clinical Features
- A fetid odor ultimately develops that is extremely unpleasant.
- The patient almost always complains of an inability to eat because of the severe gingival pain and the tendency for gingival bleeding
- The patient usually suffers from headache, malaise, and a low-grade fever
- Excessive salivation with the presence of a metallic taste to the saliva is often noted
- Regional lymphadenopathy is usually present.

- Clinical Features
- **In advanced and more serious cases, there may be generalized or systemic manifestations**
  - **–Leukocytosis**
  - **–gastrointestinal disturbances**
  - **–Tachycardia.**
- **After the NUG is cured, the crests of the interdental papillae, which have been destroyed leaving a hollowed-out area**
  - **–Constitute an area which retains debris and microorganisms**
  - **–Serve as an 'incubation zone.'**

- Bacteriologic Examination
- Smears of material from the gingiva
  - –Fusiform bacilli (genus Fusobacterium or Fusiformis)
  - –Oral spirochete (Borrelia vincentii),
  - –Various other spirochetes,
  - –Filamentous organisms,
  - –Vibrios,
  - –Cocci
  - –Desquamated epithelial cells
  - –Varying numbers of polymorphonuclear leukocytes



- Relative numbers of microorganisms present vary with the stage of the disease
- Secondary invaders being more prominent in the later phases as well as in the subacute form of necrotizing ulcerative gingivitis

- Diagnosis
- **Smear studies - nonspecific findings**
- **Although the presence of the disease can often be confirmed when vast numbers of the spirochete and fusiform bacteria are seen**
- **It may be also present in 'normal mouths,' acute herpetic gingivostomatitis, simple pericoronitis, marginal gingivitis and chronic gingivitis**
- **final diagnosis is made clinically**

- Histologic Features
- Microscopic examination of the gingiva: acute gingivitis with extensive necrosis
- Surface epithelium is ulcerated and replaced
  - –Thick fibrinous exudate
  - –Pseudomembrane, containing many PMN and MCO
- Lack of keratinization of the gingival tissues
- CT is infiltrated by dense numbers of PMN and shows an intense hyperemia
- Microscopic picture is an entirely nonspecific one

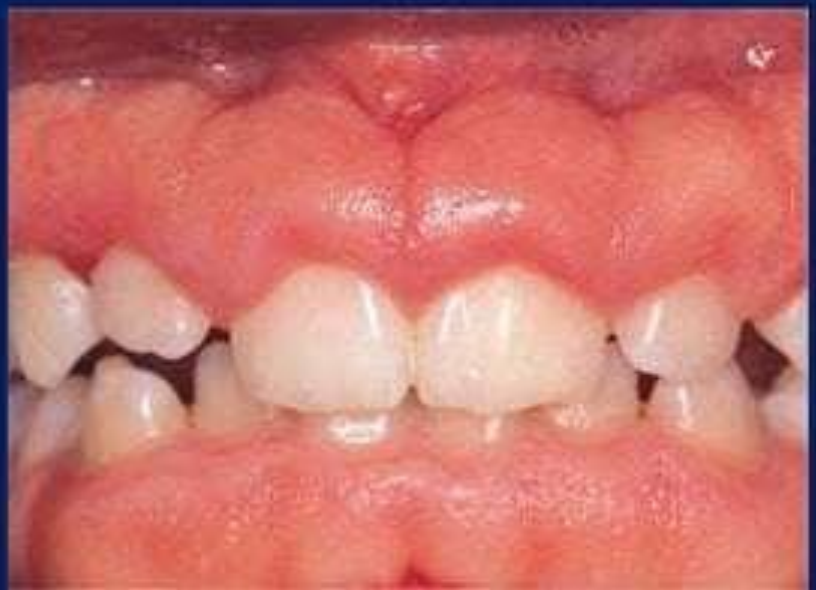
- Treatment
- **Superficial cleansing in the early acute stage with**
  - **–Chlorhexidine**
  - **–Diluted hydrogen peroxide**
  - **–or Warm Salt water**
- **This is followed by thorough scaling and polishing**
- **Topical anesthetics may be required to reduce the pain during this procedure**
- **In many such cases, prompt regression of the disease results even without medication**
- **If not use of antibiotics coupled with local treatment**

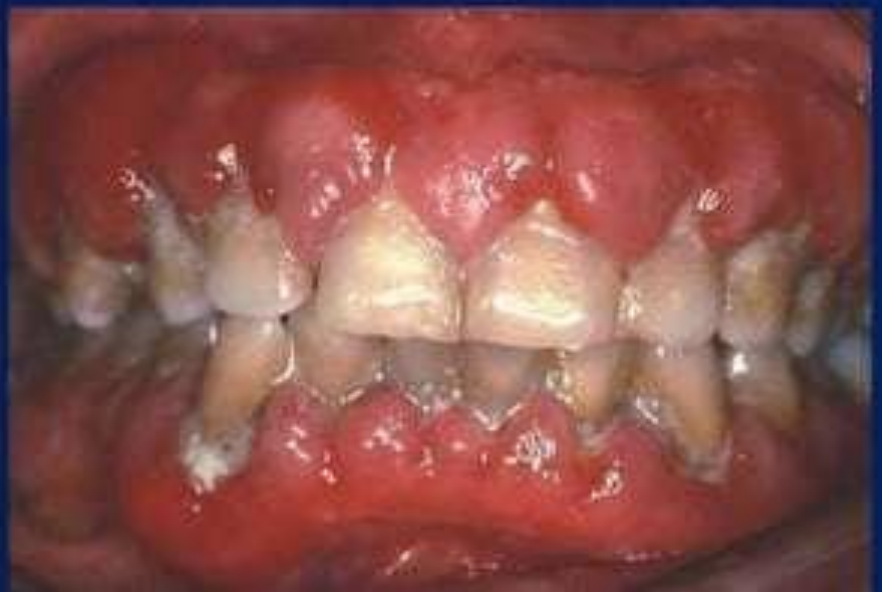
- *Treatment*
- **Usually NUG subside in 48 hours with adequate treatment**
- **Sometimes there may be**
  - **–Considerable destruction of tissue, involving the interdental papillae and marginal gingiva**
  - **–Leading to punched-out appearance of the interproximal gingiva**
  - **–And the apparent gingival recession even after the regression of the disease**
- **Recontouring of gingival papillae is usually required**
  - **–proper use of round toothpicks**
  - **–or gingivoplasty**

- Prognosis
- **Treatment cannot be considered complete until the gingival tissue contours almost normal.**
- **High recurrence rate**
- **Occasional serious sequelae such as gangrenous stomatitis or noma, septicemia and toxemia**
- **Even death have also been reported following this disease**

Gingival Enlargement

- Increase in the size of the gingiva so that soft tissue overfills the interproximal spaces
- Balloons out over the teeth and protrudes into the oral cavity
- May be localized to one papilla
- Or may involve several or all of the gingival papillae throughout the mouth





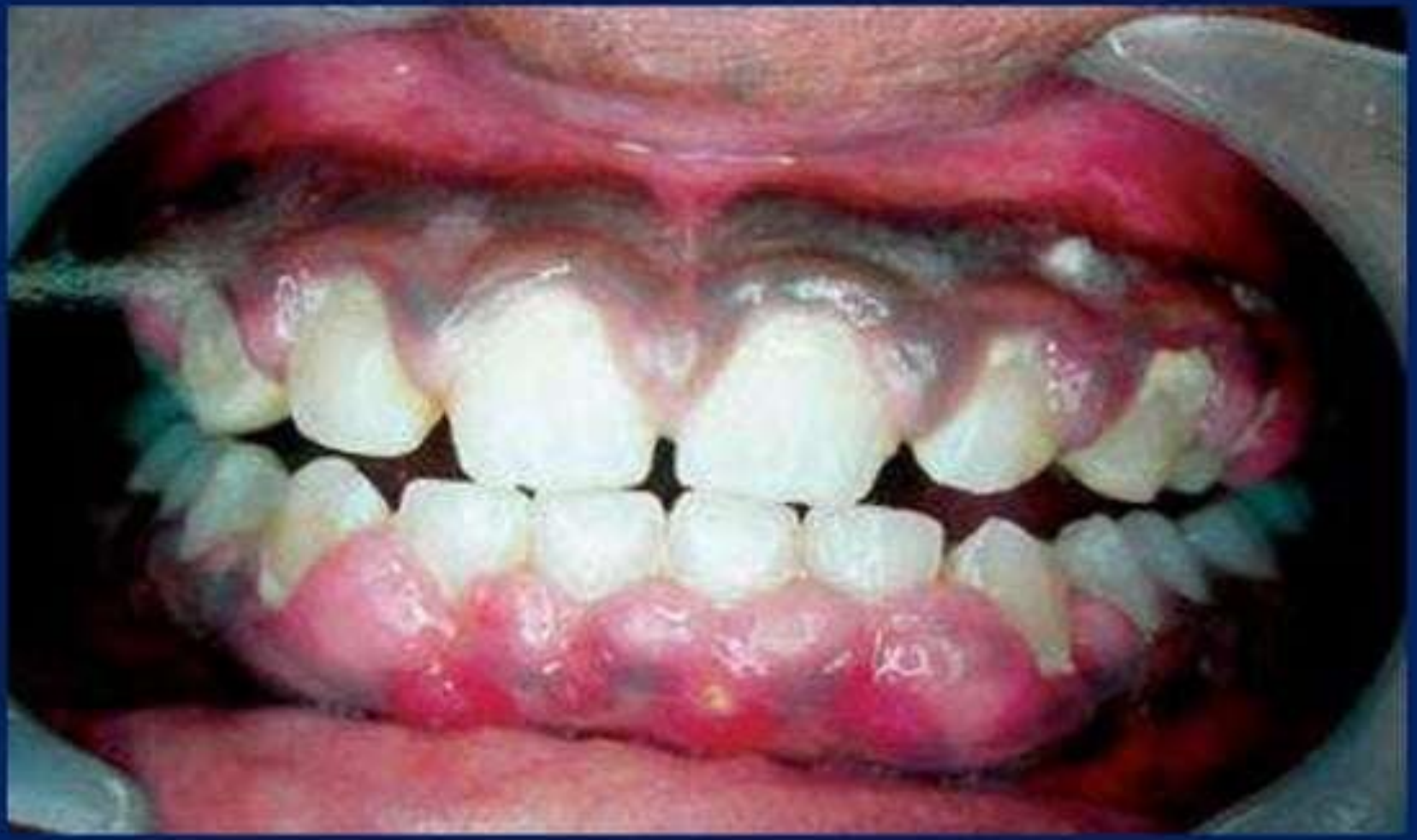
- The enlargement is usually more prominent on the labial and buccal surfaces
- Although it does occasionally develop in the lingual gingiva.
- It does not involve the vestibular mucosa
- Not to be confused with overgrowths of bone, or exostoses, noted occasionally on the alveolar bone,

- Etiology
- **It can be classified based on etiologic factors and pathologic changes as follows:**
  - **–Inflammatory gingival enlargement**
  - **–Drug induced gingival enlargement**
  - **–Enlargement associated with systemic factors**
  - **–Conditioned enlargement**
  - **–Enlargements due to systemic diseases**
  - **–Idiopathic gingival enlargement**
  - **–Neoplastic enlargement**
  - **–False enlargement**

Inflammatory Enlargement

- Local irritation: poor oral hygiene, accumulation of dental calculus or mouth breathing
- Host tissue response to dental plaque accumulation.
- Clinical Features
  - –Gingivae are soft edematous,
  - –Hyperemic or erythematosus
  - –Sensitive to touch - bleed easily
  - –Glossy, non stippled surface
- Histological Features
  - –Hyperemia edema
  - –Lymphocytic infiltration
  - –Proliferation of the fibrous CT of the gingiva

Inflammatory gingival enlargement due to local collection of plaque



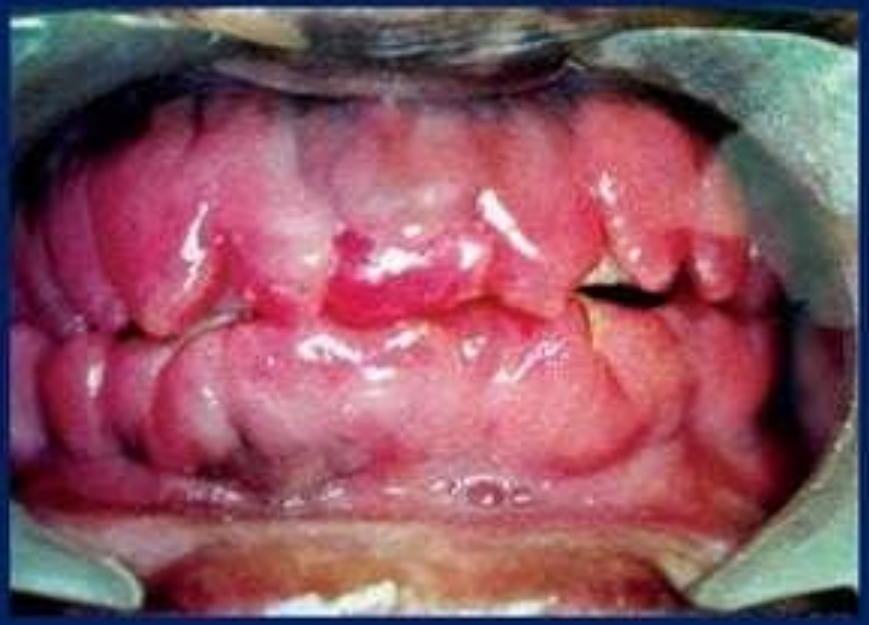
Drug-induced Gingival Enlargement

- **Anticonvulsants:** Diphenylhydantoin (dilantin sodium), valproic acid, methsuximide, and succinimides
- **Immune suppressants:** Cyclosporine
- **Calcium channel blockers:** nifedipine, nitrendipine and verapamil
- **Hydantoins:** ethotoin and mephenytoin
- **Phenytoin**

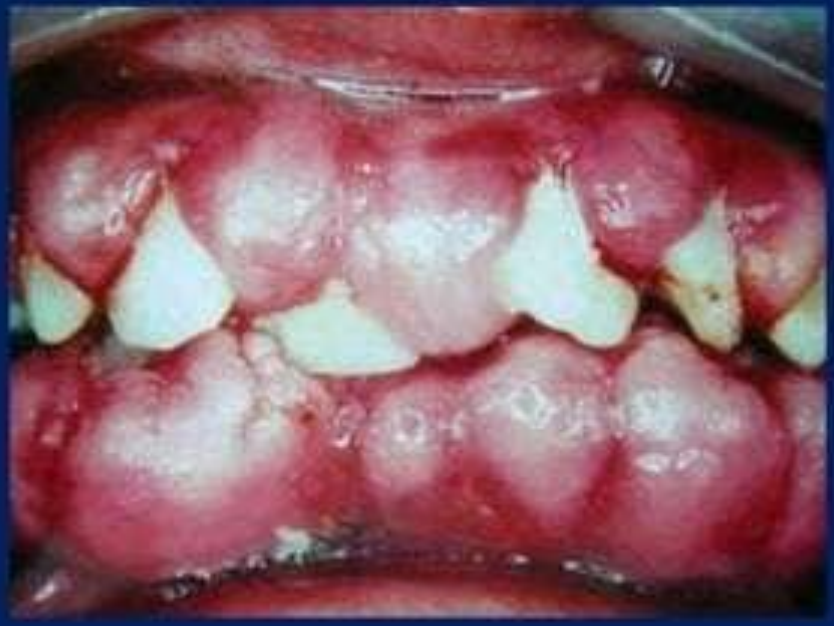
  –Stimulates proliferation of fibroblast-like cell in tissue culture

  –Decreases the collagen degradation

Enlargement of the gingiva in an epileptic patient receiving dilantin sodium



Gingival enlargement in a patient receiving nifedipine



- Clinical Features
  - –Begins painless
  - –Involving one or two interdental papillae
  - –Increased stippling and ultimately a roughened or pebbled surface with lobulations
  - –Gingival tissues are dense, resilient, and insensitive
  - –Show little or no tendency to bleed

ENLARGEMENT ASSOCIATED WITH SYSTEMIC FACTORS

- **Conditioned Enlargement**
  - **–Hormonal Enlargement: Pregnancy and Puberty**
  - **–Allergic Enlargement**
  - **–Nutritional Enlargement**

- Hormonal Enlargement: Puberty
- **Inflammatory gingival enlargement often occurs at puberty, both in men and women**
- **Endocrine imbalance at this particular stage of the patient's development.**
- **Or because of poor oral care at this age: local irritation associated with eruption of teeth, and/or nutrition may be inadequate**
- **Thus, the enlargement may be only indirectly associated with an endocrine disturbance**

- Hormonal Enlargement: Pregnancy
- **Gingival enlargement of the inflammatory type during pregnancy**
- **This proliferation may be due to**
  –Disturbed nutrition,
  –Poor oral hygiene,
- **Increased levels of estrogen and progesterone in pregnancy**
- **Vascular permeability: leads to gingival edema**
- **Altered inflammatory response to dental plaque**

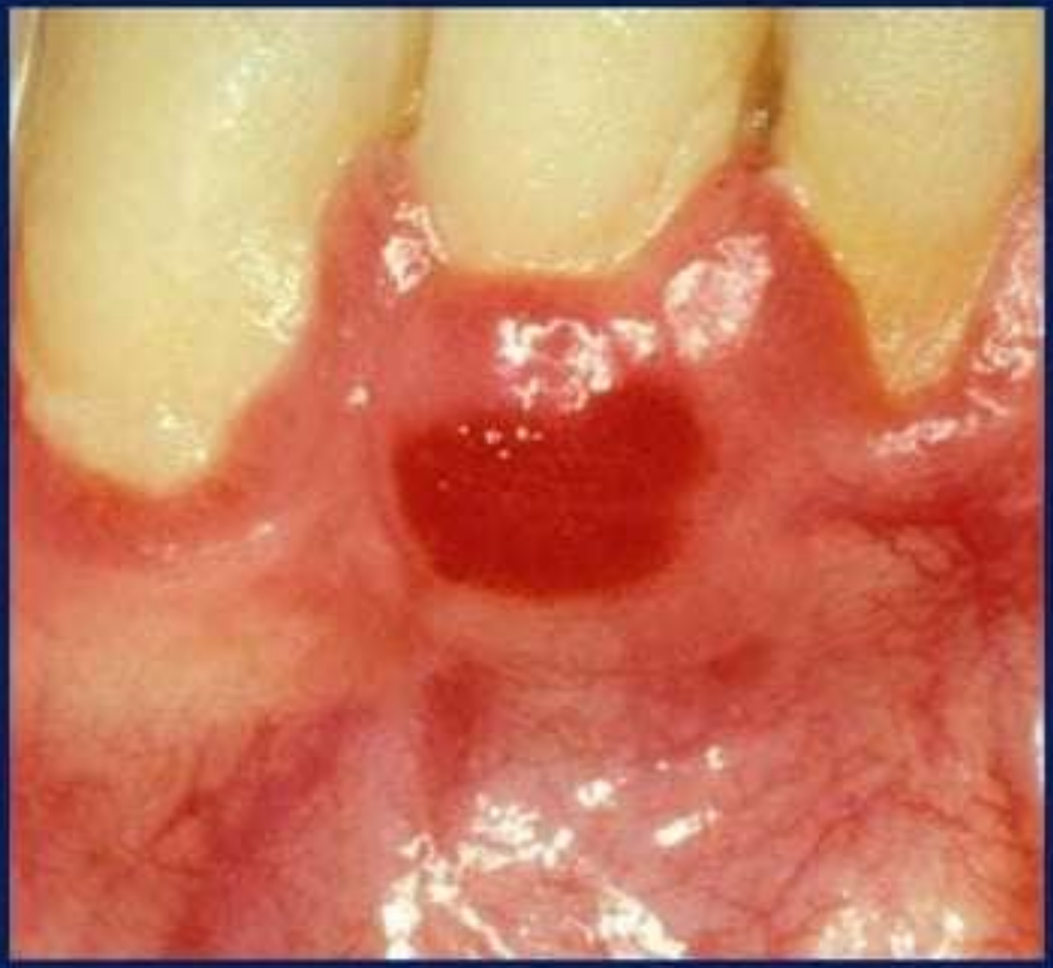
- **Microscopic studies resemble Pyogenic granuloma**
  - **–Increased vascularity,**
  - **–Multiplication of fibroblasts,**
  - **–Edema**
  - **–Infiltration of leukocytes into the gingiva**

- Allergic Enlargement: Plasma Cell Gingivitis
- Also called as Atypical gingivitis, plasma cell gingivostomatitis
- Arises as a hypersensitive reaction to a component of chewing gum, dentifrices, or some of the dietary components
- Commonly presents

  –Mild marginal gingival enlargement, sometimes extending to involve the attached gingiva

  –More prevalent in young women

  –Initial symptom is soreness of the mouth, which is intensified by hot or spicy food



- Allergic Enlargement: Plasma Cell Gingivitis
- **In severe cases, extends to buccal and vestibular mucosa.**
  - **–Gingiva appears swollen,**
  - **–Erythematous,**
  - **–Friable with loss of stippling**
- **The involvement of other oral tissues like the tongue and lips is common**
- **They appear atrophic, dry, and exhibit cracks or fissures**

- Plasma Cell Gingivitis - Histologic Features
- Surface epithelium is hyperplastic, shows intracellular edema, and micro abscesses
- Connective tissue

  –densely infiltrated with chronic inflammatory cells

  –predominantly a polyclonal mixture of plasma cells
- Marked vascular dilatation with severe thinning of epithelium over the connective tissue pegs

- *Treatment and Prognosis*
- **Possible allergens should be identified**
- **Careful study of the patient's history and eliminated**
- **Topical and systemic steroids give good results**

- Nutritional Enlargement: Vitamin C Deficiency
- **Spongy, bleeding gums of scurvy, tender, swollen, and edematous**
- **Gingival sulci are often filled with partially clotted blood**
- **Crests of the interdental papillae are red or purple**
- **Sometimes ulceration and necrosis of the papillae as infection becomes superimposed upon the susceptible tissues**
- **Hemorrhages following slight trauma to other parts of the body**

- **Enlargement is essentially a conditioned response to bacterial plaque.**
- **Combined effect of vitamin C deficiency and inflammation produces this enlargement.**
- **Treatment: improvement of oral hygiene and administration of vitamin C**

ENLARGEMENT DUE TO SYSTEMIC DISEASES

- Leukemia
- Granulomatous Diseases
- Regional Enteritis (Crohn's Disease)
- Idiopathic Gingival Enlargement
- Neoplastic enlargements
- False enlargements

- Leukemia
- **Early finding in acute monocytic, lymphocytic or myelocytic leukemia**
- **Leukemic enlargement may be diffused or marginal, localized or generalized**
- **Gingiva appears shiny, bluish red, soft, edematous, easily compressible, tender, and frequently ulcerated**
- **No signs of stippling**
- **Gngivae are usually inflamed, owing to local infection**
- **Occasionally a necrotizing ulcerative gingivitis develops**

- Histology

  –gingival tissues are packed with immature leukocytes

  –specific type depending on the nature of the leukemia

  –These abnormal white blood cells are unable to perform their defensive function

  –Capillaries are engorged

  –Connective tissue is edematous.

- Granulomatous Diseases
- **Some local and systemic granulomatous diseases may involve the gingiva and present as gingival enlargement**
  - **–Crohn's disease**
  - **–Sarcoidosis**
  - **–Wegner's granulomatosis**
  - **–Foreign body gingivitis**

- Idiopathic Gingival Enlargement
- Gingival tissues are so diffusely enlarged that the teeth are completely covered
- If the enlargement is present before tooth eruption, the dense fibrous tissue may even interfere with or prevent eruption
- Also called as 'fibromatosis', 'fibromatosis gingivae,' 'elephantiasis gingivae,' and 'congenital macrogingiva'
- Cause of this developmental enlargement of gingival tissue is not known
- It is probably hereditary, being transmitted as an autosomal dominant trait in some instances

- Idiopathic Gingival Enlargement
- **Clinical Features**

  –large masses of firm,

  – dense, resilient,

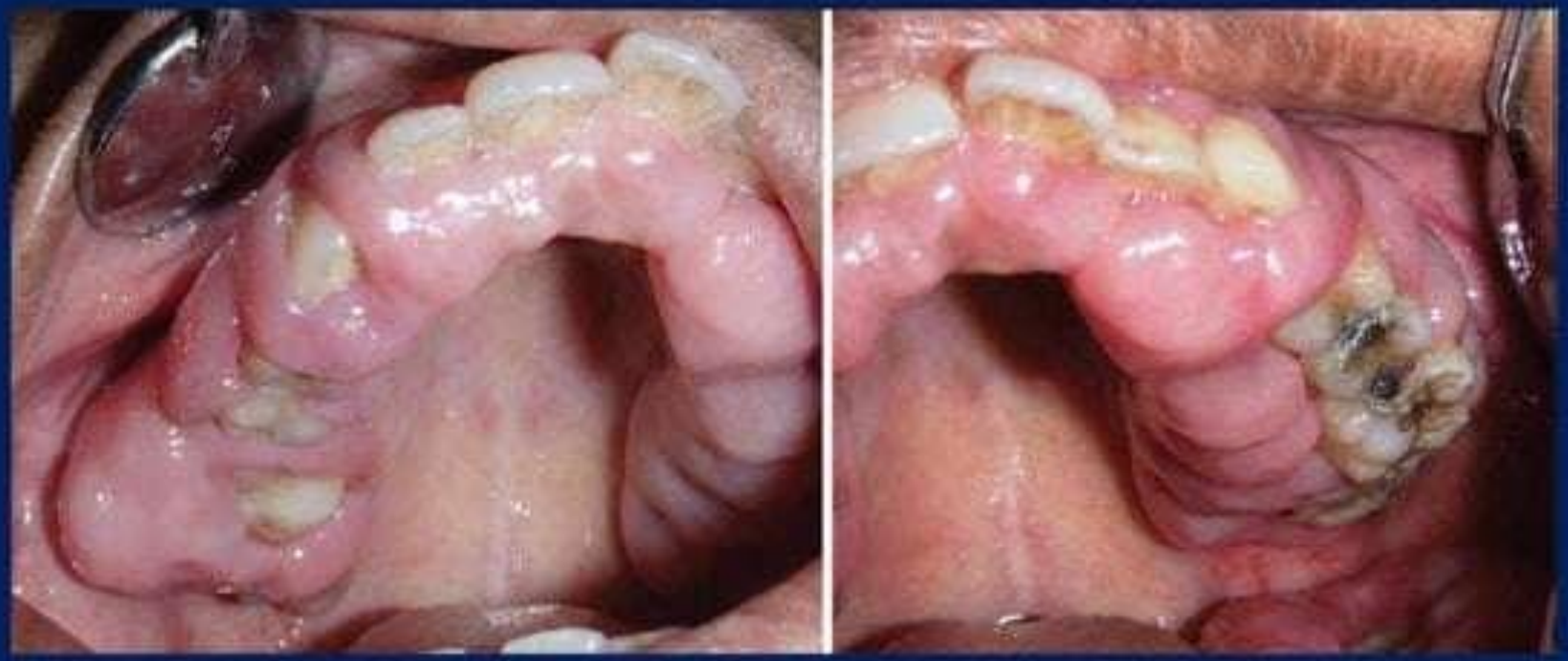
  –insensitive growth that covers the alveolar ridges and extends over the teeth

  –normal in color

  –patient complains only of the deformity

  –gingivae are so enlarged that the lips protrude
- **This enlargement may be detected at an early age and in a few cases even at birth**
- **Teeth do not erupt normally because of the dense fibrous tissue.**

Idiopathic Gingival Enlargement



- Histological Features

  –hyperplastic epithelium with elongation of rete ridges

  –mild hyperkeratosis

  –underlying stroma is made up almost entirely of dense bundles of mature fibrous tissue with few young fibroblasts present

- Management

  –Surgical removal of the excess fibrous tissue is the only feasible treatment

  –condition may recur afterwards

- **Neoplastic enlargements:** benign and malignantneoplasms involving the gingiva
- **False enlargements**
  - –Due to underlying dental or osseous anomalies
  - –Not an abnormality of the gingiva as such.

PERIODONTITIS

- An inflammatory disease of the supporting tissues of the tooth caused by specific MCO or group of specific MCOs, resulting in progressive destruction of the PDL and alveolar bone with pocket formation, recession, or both.
- It is classified as

  –Chronic,

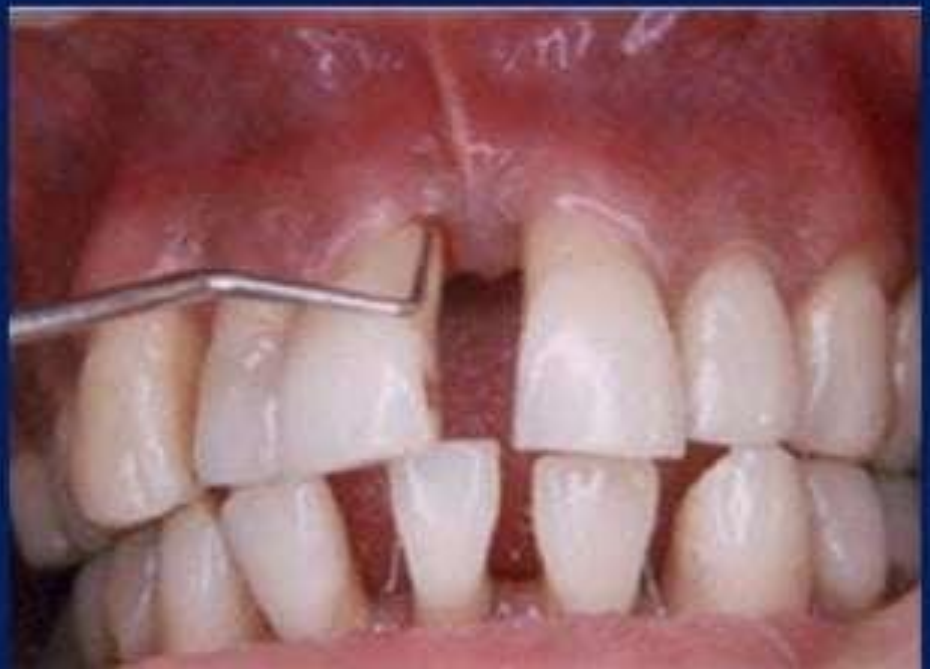
  –Aggressive,

  –Manifestation of systemic diseases

  –Occlusal trauma: Acute periodontitis that results from acute trauma

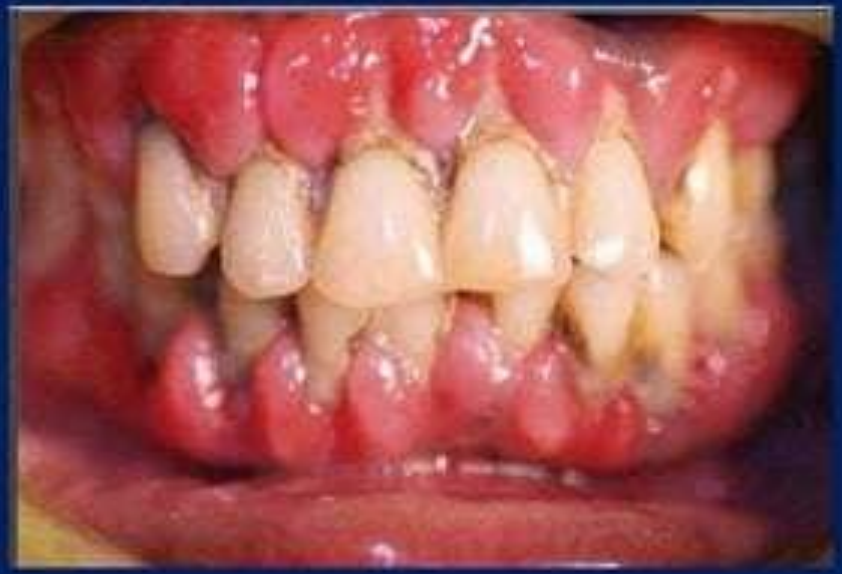
AGGRESSIVE PERIODONTITIS

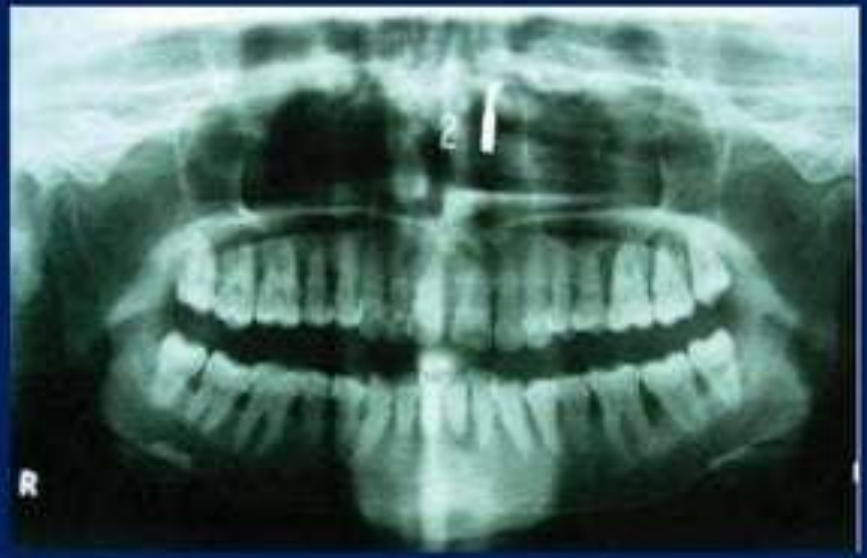
- Rapidly progressing type of periodontitis that occurs in patients who do not have large accumulations of plaque and calculus
- This may be either localized or generalized
- Earlier it was called Juvenile periodontitis and Rapidly progressive periodontitis



Presence of a large amount of plaque, calculus and gingival infection

Loss of bone support involving more than three teeth other than first molars and incisors





- Etiology
- **Familial tendency suggesting a genetic predisposition**
- **Defect in the immune response rather than plaque and calculus deposition.**
- **Disease is specific**
- **Microbiology**

  –Actinobacillus actinomycetemcomitans

  –Porphyromonas gingivalis

  –P. gingivalis

  –Bacteriodes forsythus

  –Other species related are Capnocytophaga sputigena, Mycoplasma subspecies and Spirochetes

- Histological Features
- **Functional defects of polymorphonuclear leukocyte, monocytes, or both**
- **But without any systemic manifestations**
- **Because of this, their defensive ability against some of the periodontal pathogens is defective**
- **The bactericidal activity against Actinobacillus actinomycetemcomitans is particularly faulty in localized form.**

- Clinical Features: Localized form
- Usually occurs around puberty
- Strong familial tendency
- Localized to the first molars and incisors
- Attachment loss in at least two permanent teeth, one of which is the first molar
- Striking feature: absence of clinical inflammation with minimal local factors despite the presence of a deep periodontal pocket
- Rate of alveolar bone loss is considerably higher than in chronic periodontitis
- Associated primarily with A.actinomycetemcomitans.

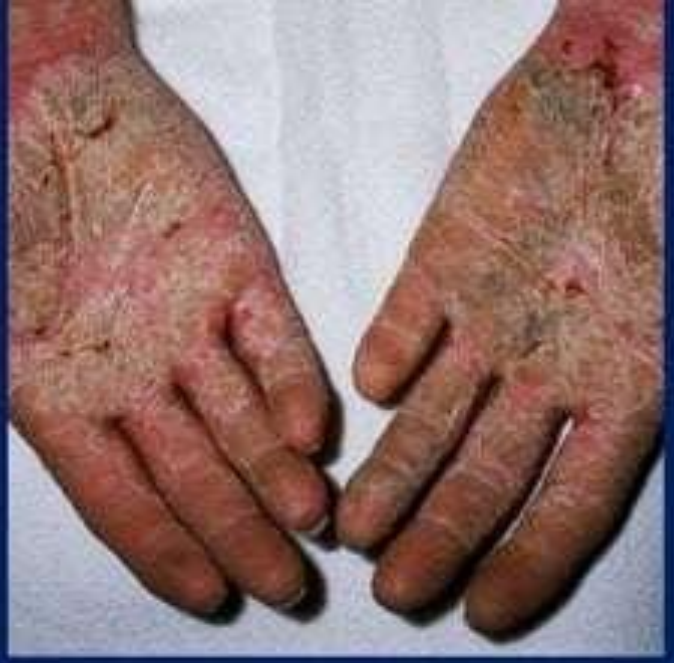
- Clinical Features: Generalized form
- **Usually affects patients under 30 years of age**
- **Involves at least three teeth other than the first molar and incisor**
- **Exhibits poor serum antibody response to infecting agents**
- **Many cases represent the localized form which becomes generalized with time**
- **Organism associated with the generalized form is more complex and closely resembles chronic periodontitis**

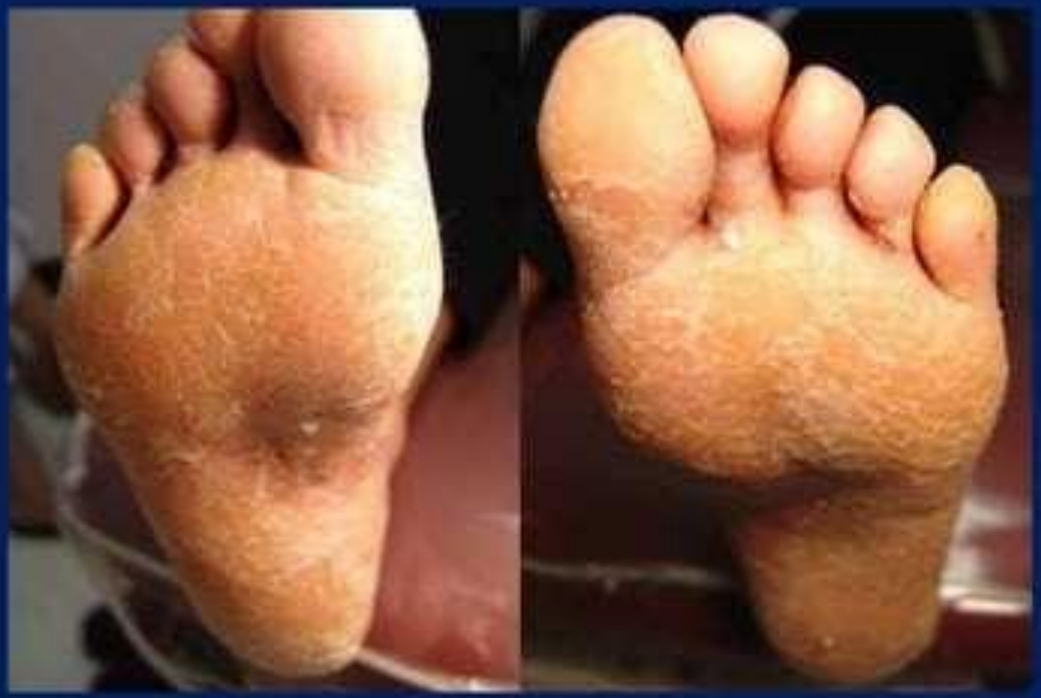
- Diagnosis
- **Definitive diagnosis**
  - **–Family history,**
  - **–Clinical,**
  - **–Radiographical,**
  - **–Microbiological,**
  - **–Histological examination with leukocyte function tests**

- Papillon-Lefèvre syndrome
- **Autosomal recessive disorder: oral and skin lesion**
- **Oral manifestations**

  –aggressive periodontitis

  –severe destruction of the alveolar bone involving both the deciduous and permanent dentitions
- **Skin lesions: keratotic lesions of palmar and plantar surfaces**
- **Rapid bone loss, mobility and pathological migration occurs**
- **Loss of the entire dentition at a much younger age**





- Radiographic Features
- **Localized form**

  –vertical loss of alveolar bone is seen around the first molar and incisor

  –arc-shaped alveolar bone loss extends from the distal surface of the second premolar to the mesial surface of the second molar

  –widening of the PDL space

  –'vertical' pocket formation, differs from the 'horizontal' type of bone loss seen in chronic periodontitis appears to be 'horizontal.'
- **Generalized form**

  –bone loss may range from the involvement of one or two teeth to a maximum number of teeth

- Management
- Antibiotics should be administered in combination with mechanical removal of plaque and inflamed periodontal tissues.
- Periodontal surgery should be performed with prophylactic antibiotic cover
- Postoperative usage of chlorhexidine mouthrinse
- Periodic follow-up is necessary since there is possibility of reinfection