Diseases of periodontium.

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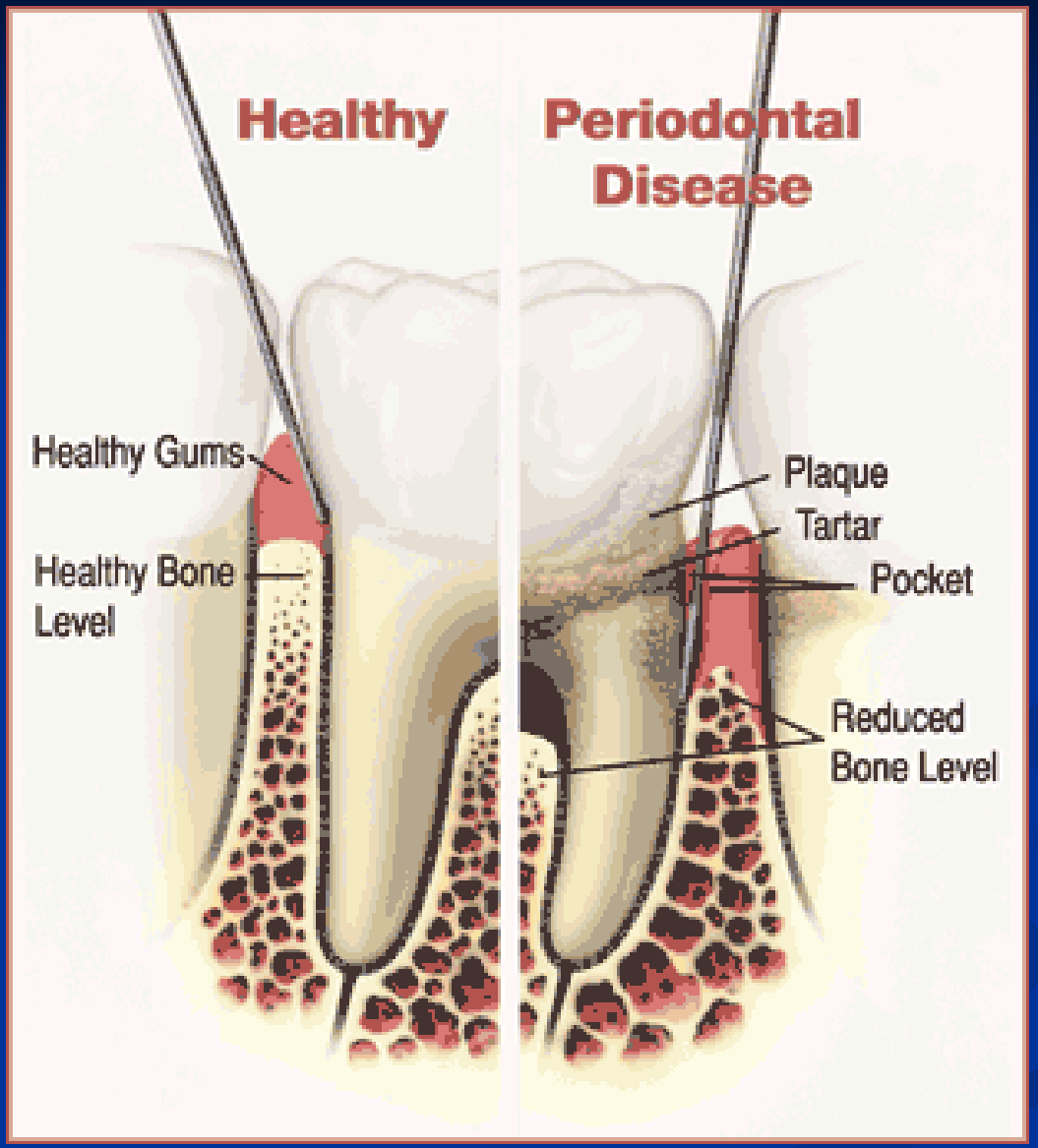
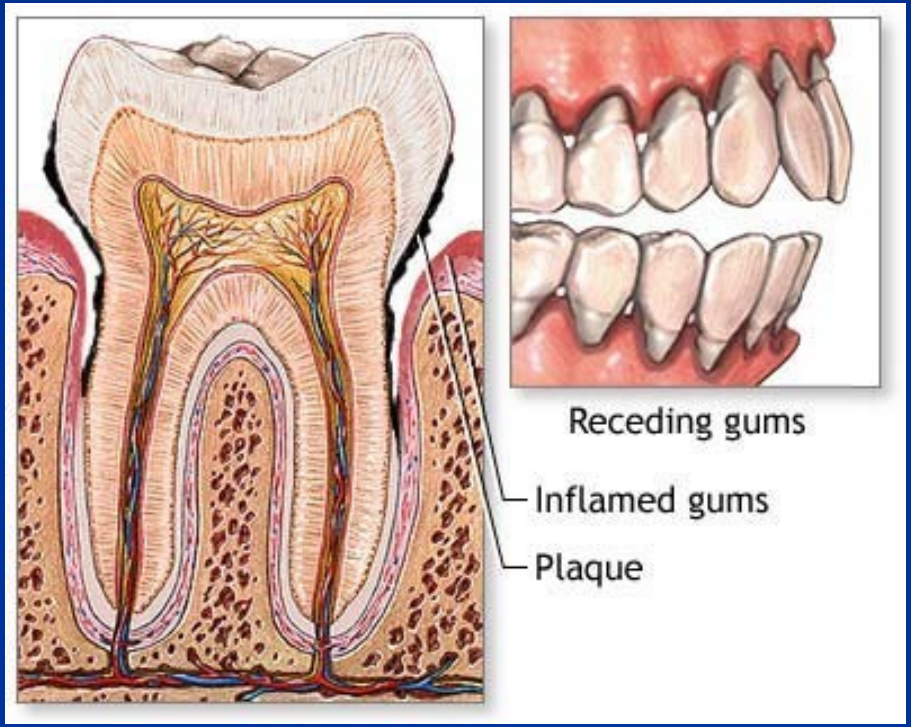
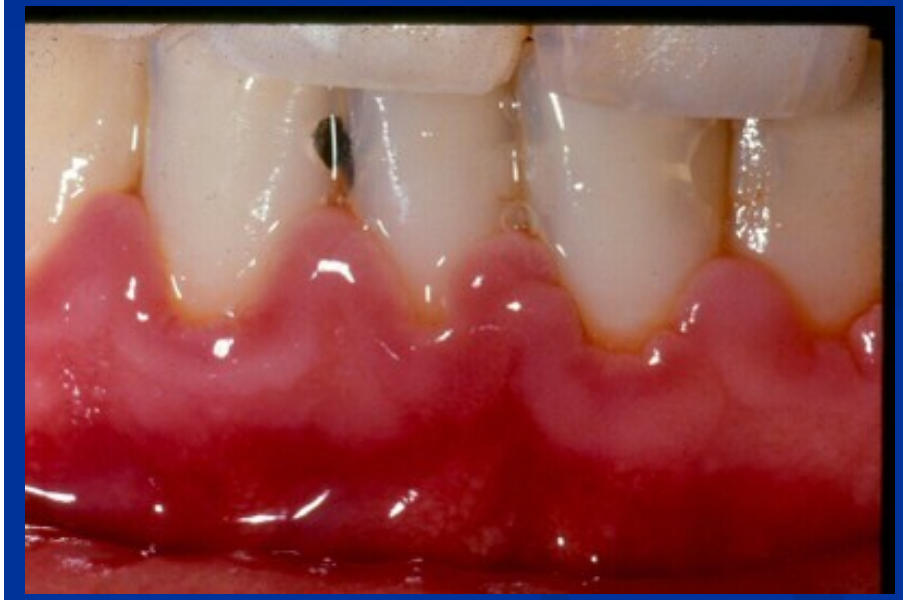

Healthy
Periodontal Disease
Healthy Gums
Healthy Bone Level
Plaque
Tartar
Pocket
Reduced Bone Level

- **Gingivitis:** inflammatory lesions confined to marginal gingiva

- **Periodontitis:** lesions associated with destruction of the connective tissue attachment of the tooth and loss of alveolar bone


Receding gums
Inflamed gums
Plaque



Epidemiology of periodontal disease

- Early periodontitis involves some of the teeth in the majority of adults
- The prevalence of pocketing/loss of attachment increases with age
- The proportion of teeth affected by periodontitis increases with age
- Advanced periodontal disease affects only a small percentage of the population

Classification of plaque-associated periodontal diseases

- **Gingivitis**
  - Associated with dental plaque only
  - Modified by systemic factors
  - Modified by medication
  - Modified by malnutrition
- **Chronic periodontitis**
  - Localised
  - Generalised
- **Aggressive periodontitis**
  - Localised
  - Generalised
- **Periodontitis in systemic diseases**
  - Immunocompromised patients
  - Genetic disorders

Based on International Workshop for a Classification of Periodontal Diseases and Conditions, 1999)

Plaque microorganisms in health, gingivitis, and periodontitis

Main species	% aerobic/ anaerobic	% Gram+/ Gram -	Motile/ non-motile
Healthy gingiva *Streptococcus* *Actinomyces*	75/25	90/10	1:40
Chronic gingivitis *Actinomyces* *Streptococcus* *Porphyromonas* *Prevotella*	60/40	65/35	Number of motile rods and spirochaetes increases with disease
Chronic periodontitis *Actinobacillus* *Porphyromonas* *Bacteroides* *Prevotella* *Fusobacterium*	20/80	25/75	1:1 Abundant motile rods and spirochaetes

Summary: microbiology of periodontal disease

- Gram-positive cocci decrease as gingivitis progresses to periodontitis
- Gram-negative anaerobic bacilli increase as disease progresses
- Motile forms increase as disease progresses
- Periodontal disease involves interactions of mixtures of bacteria forming complexes in plaque
- Certain species (periodontal pathogens) are prevalent in destructive lesions

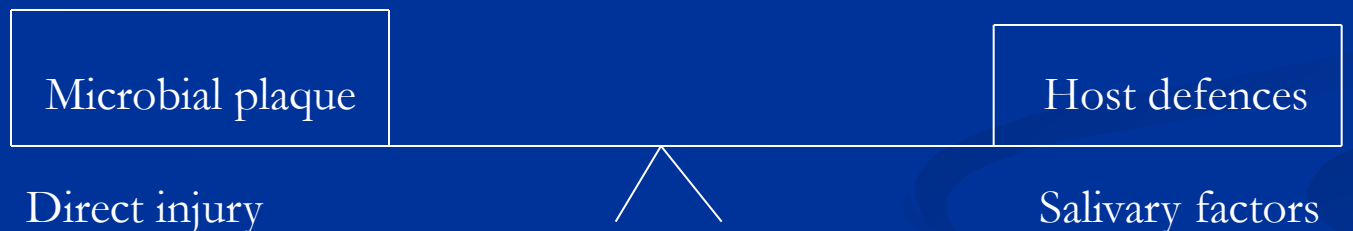
Other risk factors for periodontal diseases

- **Local factors**
  - pre-existing anatomy of the teeth, gingiva, and alveolar bone
  - alignment and occlusal relationships of teeth
- **Systemic factors**
  - Diabetes mellitus
  - Pregnancy and sex hormones
  - Nutrition (avitaminosis C)
  - Blood diseases
  - Drugs
  - AIDS
  - Smoking

Drugs affecting periodontal tissues and the activity of periodontal disease

Anti-epileptics	Phenytoin	Gingival hyperplasia
Immunosuppressants	Azathioprine Corticosteroids Cyclosporin	Equivocal reduction of disease activity Gingival hyperplasia
Non-steroidal anti-inflammatory drugs	Indomethacin Ibuprofen	Equivocal reduction of disease activity
Calcium channel blockers	Nifedipine Verapamil	Gingival hyperplasia
Sex hormones	Oestrogen Progesterone	Exacerbation of pre-existing gingivitis

Host-parasite equilibrium at the plaque-gingival interface: chronic periodontal disease = disturbance of this balance = a dynamic process reflecting changes in the balance of the host-parasite relationship with time



Direct injury
Toxic products
Enzymes
Antigenic challange

Salivary factors
Crevicular fluid
Epithelial barrier
Migrating neutrophils
Immune response
Potential for tissue regeneration and repair

Initial gingivitis

- Microscopic area around base of gingival sulcus
- Acute inflammatory changes
  - Cellular exudate: enhanced migration of neutrophils
  - Fluid exudate: increased crevicular fluid flow
  - Number of chemical mediators of inflammation responsible

Early gingivits

- Lymphocytic infiltration
- Impairment of barrier function of junctional epithelium
- Gingival pocket formation; growth of subgingival plaque

Established gingivitis

- Expansion of area of inflammation and destruction of gingival connective tissue
- Predominance of plasma cells in inflammatory infiltrate
- Deepening of gingival pocket; thining/ulceration of pocket epithelium

Chronic periodontitis

- Apical extension of destructive inflammation
- Loss of connective tissue attachment and destruction of alveolar bone
- Apical migration of junctional epithelium and pocket formation
- Periods of quiscence/stability; random bursts of destructive activity

Degradation of the extracellular matrix (ECM) of gingiva, periodontal ligaments, and the destruction alveolar bone

- Matrix metallo-proteinases (MMPs) degrade ECM
- Tissue inhibitors of metalloproteinases (TIMPs) inhibit MMPs
- Activity of MMPs and TIMPs in balance in health
- Increased MMPs activity in disease; reflects fluctuations in cytokine activity (IL-1)
- Local mediators affecting bone resorption:
  - Cytokines (IL-1, IL-6, TNF)
  - Prostaglandins (PGE_2)
  - Growth factors (e.g. from osteblasts which regulate the osteoclast recruitment)

Pathogenesis of periodontal disease

- Disturbance of host-parasite balance
- Activation of host inflammatory and immune response
- Enhanced synthesis of inflammatory mediators/cytokines
- Periodontal connective tissue degradation/bone resorption
- New equilibrium in host-parasite realtionship as host response contains the challange for plaque bacteria

Clinical forms of periodontitis

- Chronic periodontitis
- Aggressive periodontitis
- Periodontitis in systemic disease

Aggressive periodontitis

- Usually juvenile
- F>M
- Rapid destruction of alveolar bone, vertical bone loss, deep intrabony pockets
- First molars and/or maxillary incisors
- Pathogenesis obscure; inflammatory and bacterial plaque??? (G-anaerobic rods *(Actinobacillus actinomycetemcomitans)*, genetic factors, abnormalities in cell-mediated immunity)

Periodontitis in systemic diseases

- **Diseases associated with major abnormalities of neutrophils**
  - Agranulocytosis
  - Cyclic neutropenia (AD, mutation in the gene for neutrophil elastase)
  - Chediak-Higashi syndrome (AR, mutation in lysosomal trafficing regulatore gene)
  - Job syndrome (hyper IgE syndrome, hereditary)
- **Diseases in which there may be associated neutrophils dysfunctions**
  - Papillon-Lefevre syndrome (palmar and plantar hyperkeratosis, severe periodontal destruction; AR, mutation in lysosomal enzyme cathepsin C gene)
  - Down syndrome
  - Juvenile-onset diabetes mellitus
- **Other systemic diseases**
  - Hypophosphatasia
  - Langerhans cell histiocytosis (histiocytosis X)
  - Ehlers-Danlos syndrome

Gingival enlargement

- **Fibrous overgrowths**
  - Gingival fibromatosis (hereditary, AD)
  - Chronic hyperplastic gingivitis
  - Drug associated hyperplasia (epanutin (anti-epilepticum), verapamil, nifedipin (cardiovascular diseases), cyclosporin (immunosuppressive drug))
- **Oedematous enlargement**
  - Oedematous gingivitis in puberty, pregnancy, oral contraceptives, scurvy (avitaminosis C)
- **Systemic disease**
  - Acute leukaemias
  - Wegener´s granulomatosis

Desquamative gingivitis

- **Gingival manifestation of several different diseases:**
- **Mucous membrane pemphigoid**
- **Lichen planus**
- **Local hypersensitivity reaction**
- **Orofacial granulomatosis** (in Crohn´s disease, sarcoidosis, other causes of granulomatous inflammation (infection, foreign bodies), idiopathic, Melkersson-Rosenthal syndrome (recurring facial paralysis, swelling of the face and lips, and the development of folds and furrows in the tongue), allergic reaction,…)

Thank you for your attention …