Periodontology and oral diseases in childhood

Dr. Radó Zsuzsanna Stefánia, Dr. Löchli Heike

Semmelweis University,
Department of Paediatric Dentistry and Orthodontics

SEMMELWEIS
UNIVERSITY 1769

Overview

- Normal periodontal conditions
- Gingivitis
- Periodontitis
- Prevention
- Gingival recession
- Traumatic ulcerative gingival lesions

- Developmental disturbances
  - Cleft patients
  - Ankyloglossia
- Anomalies of the tongue
  - Lingua geographica
  - Acute and chronic inflammation of the tongue
- Diseases of the lip (Cheilitis exfoliativa, granulomatosa, angularis)
- Diseases of viral origin (HSV, EBV, HIV, HPV)
- Fungal infections
- Recurring ulcerative lesions (RAU,RAS, Mikulicz, Sutton, Cooke)

Goran Koch , Sven Poulsen: Pediatric Dentistry: A Clinical Approach

Fábián G., Gábris K., Tarján I.: Gyermekfogászat, fogszabályozás és állcsont-ortopédia

Normal periodontal conditions

- Primary dentition
  - Bulkier
  - Stippling develops gradually after the age of 2-3
  - Connective tissue is similar to permanent teeth but thicker junctional epithelium → more resistant to inflammation bc it is less permeable
- Permanent dentition
  - Healthy marginal gingiva becomes thinner and pinkish

GINGIVITIS

Clinical appearance

- Hard to distinguish between normal and pathologic reactions
- If plaque accumulation is minimal and the defense mechanisms work well, there will be no clinical symptoms
- More pronounced plaque accumulation or defects in defense reactions will result in clinical symptoms
- Diagnosis based on clinical symptoms (GBI)
- In healthy children gingival infections remain superficial
- If a child has long standing generalized gingivitis general health should be checked

Vascular response and accumulation of inflammatory cells

→Reddish gingiva

→Swollen appearance

→Papillae protruding from the interproximal spaces

→Increased volume, shiny surface

→Crevicular exudation

→Increased tendency of bleeding on probing

Age dependent tendency to develop gingivitis

- Preschool children are less susceptible than adolescents and adults
- Possible causes:
  - Spirochetes and black-pigmented bacteroides are less frequently found in children
  - Lower poportions of Fusobacterii, Eubacterii and Lactobacilli
  - Increased cell proliferation and turnover of collagene
  - Cellular infiltration is predominantly of T-lymphocytes (in adults it's B-s)
  - Lower permeability
  - Pubertal hormonal changes increase the risk

Etiology of gingivitis

- Unanimous agreement: **microbial plaque**

→Quantity of bacteria and bacterial products

- BUT must be regarded as **multifactorial** disease with intrinsic and extrinsic factors
  - Disturbances in enamel mineralization →rough surface
  - Manifest carious lesions
    - Cervical carious lesions are almost always accompanied with chronic ginigvitis
  - Restoration with defective margins/rough surfaces; braces
  - MALOCCLUSIONS are not dominant → depends on oral hygiene
  - Systematic factors

Factors modifying gingivitis I.

- Mouth breathing
- Hormonal changes (puberty gingivitis)
- Eruption gingivitis
  - Gingival response is often out of proportion to the degree of bacterial irritation
  - Epithelium displays degenerative changes
  - Cleaning is unpleasant

Diabetes mellitus
(Factors modifying gingivitis II.)

- More susceptible to develop periodontal diseases
- Tendency to develop chronic forms is higher
- Specially poorly controlled DM

Leukemia
(Factors modifying gingivitis III.)

- Most common form during childhood: acute lymphoblastic leukemia
- Often accompanied by severe oral symptoms
- Gingival margins are soft and swollen from the infiltration
- Cytotoxic treatments
- Drug interference with the replication of epithelial cells
- Plaque controll before the start of cytotoxic treatment!

Agranulocytosis
(Factors modifying gingivitis IV.)

- Malignant type of neutropenia
- Acute and very severe condition
- Etiology: drug induced or autoimmune
- Oral ulcerations and peridontal manifestations are common
- In chronic cases the gingiva becomes hyperplastic with granulomatous changes

Heart conditions
(Factors modifying gingivitis V.)

- Severity of oral manifestations is directly proportional to the cyanosis
- Bluish-red gingiva
- Sometimes antibiotic prophylaxis is indicated

Drug-induced gingival overgrowth

- Calcium channel blockers (nifedipine)
- Immunosuppressives (cyclosporinA)
- Anticonvulsants (phenytoin)
  - Overgrowth occurs more frequently in children than in adults
  - Plaquecontrol program before start of therapy!!!!
- Pseudopockets (over 4mm)
- Altered tissue composition: more glycosaminoglycans

Non-plaque-induced gingival lesions

- Specific bacterial origin:

Neisseria gonorrhoea, Treponema pallidum, Actinomyces israelii, Mycobacterium tuberculosis, Streptococcus

- Viral origin:

Herpes simplex 1-2 (primary herpetic gingivostomatitis, recurrent oral herpes), Varicella-zoster, HPV

- Fungal origin
- Gingival manifestation of systematic conditions – desquamative gingivitis:

lichen planus, pemphigoid, pemphigus vulgaris, erythema mutliforme…

- Genetic origin: hereditary gingival fibromatosis

Gingival fibromatosis

=diffuse, non inflammatory gingival enlargement

- Autosomally inherited
- Generalized/localized
- Enlargement is pale and very firm
- Retarded eruption

Gingivitis treatment

- Marginal gingivitis
  - Plaque control
    (parents in preschool children, modified Bass technique with soft brush)
- Severe forms of gingivitis
  - Professional tooth cleaning (potentially in local anesthesia)
  - Chemical plaque control
  - Education

PERIODONTITIS

Dr. Radó Zsuzsanna Stefánia,
Dr. Löchli Heike

Clinical picture

- Ongoing inflammatory process involving deeper parts of the periodontium with loss of tooth support
- Histological appearance of inflammation is different
  - Larger proportion of plasma cells and B-lymphocytes (↔gingiv.)
- Few subjective symptoms

→Clinical diagnosis based on

- probing depth
- attachment loss
- marginal bone assesed on radiographs

→evaluation of the inflammatory status

- Bleeding on probing
- Subgingival calculus

Classification

1. Early, chronic periodontitis with minimal attachment loss (stage I, grade A)
2. Localized, rapid (stage I, grade C)
3. Generalized, rapid (stage III-IV, grade C)
4. Juvenile necrotizing periodontitis
5. Periodontitis associated with systematic diseases and genetic factors

Early, chronic periodontitis with minimal attachment loss (stage I, grade A)

- Minor loss of periodontal support
- Slow progression rate
- Considerable plaque accumulation
- Can be of local origin (trauma, development)
- Mostly in late adolescent patients
- May represent intital stage of progressive periodontal disease

Localized (stage I-II, grade C) and generalized (stage III-IV, grade C) rapid periodontitis

- No strict definition on level of attachmentloss or number of teeth involved
- Prevalence less than 0,5%
- Most cases show localized lesions with moderate signs of inflammation
- Generalized forms are often associated with systemic diseases
  - →underlying cause examination by pediatrician
  - →destruction starts early after eruption and may lead to premature loss of teeth

Localized (stage I-II, grade C) and generalized (stage III-IV, grade C) rapid periodontitis

- Onset in early permanent dentition
- Amount of plaque is not in accordance with the severity of attachment loss
- Often preceded by boneloss in primary dentition
- Localized: first molars and incisors
  - Radiographs show vertical or arch-shaped pattern of bone loss
- Generalized form
  - At least 3 teeth that are not molars or incisors
  - Severe inflammation
  - Usually in association with systematic diseases/ genetic disorders

Systematic use of bitewing radiographs (caries diagnosis) may help identify patients →early treatment

Etiology and risk factors-Microbiology

- Localized: Aggregatibacter actinomycetemcomitans
  - Leukotoxin
  - Cytolethal-distending toxin reduces the content of collagen in tissues
  - Capacity to invade periodontal tissue
  - Variation of virulence between different clones
- Generalized:
  - A.actinomycetemcomitans
  - Porphyromonas gingivalis
  - Prevotella intermedia

Image of Aggregatibacter actinomycetemcomitans colony grown on selective agar from UCL Eastman Dental Institute

P. gingivalis colonies grown on blood agar.
Heme from the media is oxidized by the bacteria to produce hemin which accumulates on the cell surface producing a characteristic black pigment after about 7 days of anaerobic incubation.

→ Periodontitis is a polyinfection with varying efficiency of the host response

Etiology and risk factors – Host-defense factors

- Polymorphonuclear neutrophil cells (PMNcells)
  - Abnormalities of adherence, chemotaxis, phagocytosis, bactericidal activitiy
  - Defect chemotaxis mainly in African-Americans
- Serum Immunoglobulin G levels high (particularly to AA)

Genetic factors and ethnicity

- Markedly increased incidence within families
- AD, AR, X-linked
- 8-63% of near relatives have severe periodontitis too
- Black or Hispanic adolescents 5-15x compared to caucasians

Etiology and risks-Modifying factors

- Restaurations, manifest caries
- Ectopic eruption
- Obesity (inflammatory molecules)
- Smoking
  - Vasoconstrictor effect → anaerob colonization
  - Substances affect fibroblasts and inflammatory cells
  - Passive smoking decreases protective LL37-protein

Necrotizing periodontal diseases (NG)

Acute necrotizing ulcerative gingivitis (ANUG) →ANUP

- Rapid onset
- Painful necrotic ulcerative gingival lesions
- Affected interdental papillae
- Foetor ex ore
- Mostly seen in children suffering malnutrition/ immunodeficiency/ stress factors/ smoking

- Professional plaque removal
- Mouthrinsing with 0,5% hydrogenperoxid or 0,1%chlorhexidine
- Antibiotics in cases of non-response to the above

Systemic diseases and syndromes connected to periodontal disease

- Diseases influencing the periodontal inflammation (immune response, tissue developement): agranulocytosis, leukaemia, congenital diseases (neutropenia, lazy leukocyte syndrome, PLS, Down, hypophosphatasia, EDS)
- Systemic diseases/factors modifying the pathogenesis of periodontitis: DM, obesity, smoking
- Diseases causing direct tissue destruction: Langerhans cell histiocytosis

Down syndrome

- Marginal bone loss
- Severe in the anterior segment, especially mandible
- Impaired phagocytic function, quantity
- Increased MMP-8
- Poor oral hygiene

Picture source:
https://www.intechopen.com/books/prenatal-diagnosis-and-screening-for-down-syndrome/oral-health-in-individuals-with-down-syndrome

Hypophosphatasia

- Low serum alkalin phosphatase
- Ricket-like skeletal changes
- Loss of alveolar bone, early tooth loss
- Anterior primary teeth
  - Aplasia and hypoplasia of root cementum
  - Large pulp chambers

Papillon-Lefevre syndrome
(keratosis palmaris et plantaris)

- Fulminant types of periodontitis with rapid bone destruction (catepsin C nonfunction)

Histiocytosis-X (reticuloendotheliosis)

- Eosinophilic granuloma in bone → more frequent in mandible than maxilla
- Hand-Schüller-Christian disease (→disseminated form)
- Treatment of the disease (steroids, irradiation, cytostatics) produce secondary negative effects

Screening

- Organized dental health care helps a lot
- Full mouth probing is debated
  → partial periodontal probing
  →first molars
- Radiographic analysis of marginal bone level
- More thorough examination in risk groups

Treatment

Initial therapy

- Plaque control
- Professional scaling
- Root planning
- Systemically administered antibiotics (aggressive P) → succesfull outcome has been reported without it

Reevaluation

- 4-6 weeks after scaling and root planning

Regular maintenance

- Subgingival sampling
- Extraction of severely affected primary teeth
- Surgery in adolescents

Prevention

Mechanical plaque control

- Parents have to brush their children's teeth
- Modified Bass technique
- Toothbrush: small, soft, big handle
- Quality is more important than quantity

(Chemical plaque control)

Gingival recessions

- Localized GR in 10-15% of teenagers
- Labial and irregular position of teeth, traumatic brushing
- History of orthodontic therapy
- Poor plaque control
- Therapy: underlying cause

Traumatic ulcerative gingival lesions

- Bacterial superinfection of traumatized gingival tissue
- Morsicatio buccae
- Infection is caused by the normal mixed flora of the oral cavity
- Ddg: HSV infection and ANUG
  - no affection of the papillae
  - localisation

2nd part ☺
ORAL DISEASES IN CHILDHOOD

Dr. Radó Zsuzsanna Stefánia,
Dr. Löchli Heike

Developmental problems

Cleft lips and palate

Ankyloglossia

- Impairment of speech
- Different levels of restriction
- Surgical treatment

Anomalies of the tongue

Dr. Radó Zsuzsanna Stefánia,
Dr. Löchli Heike

Lingua geographica (glossitis migrans)

- Smooth redish areas without papillae
- Borders are white and curly – hence the name
- Often hereditary
- Depappillated areas can be painful

Acute and chronic inflammation of the tongue

Acute inflammation
usually accompanies some general infectious disease

- scarlet fever (strawberry tongue)
- Herpetiform stomatitis

Chronic inflammation

Anaemia perniciosa

Candidiasis

- Tongue becomes red and smooth “mirrortongue”

Diseases of the lip

Dr. Radó Zsuzsanna Stefánia,
Dr. Löchli Heike

Cheilitis exfoliativa

Cheilitis exfoliativa

- Due to exsiccation of the lip (fever) or to chewing on lip
- The lip is bright red, exfoliates, cracks and bleeds
- Possible superinfection

Cheilitis acuta

- Sunburn, wind, allergy etc

Treatment: coating and moisturizing

Cheilitis granulomatosa

- Isolated symptom or part of Melkersson-Rosenthal syndrome
- Painless granulomatous enlargement of lips
- Can be regressing and recurring

Cheilitis angularis/angulus infectiosus

- Usually starts with a sense of dryness, then exfoliation, then cracking of the corners of the mouth
- Very painful
- Etiology: SLS allergy, fungal infections + vitamin B deficiency

Diseases of viral origin

Dr. Radó Zsuzsanna Stefánia,
Dr. Löchli Heike

Herpes simplex virus (HSV)

- Herpetic gingivostomatitis
- Gingivostomatitis herpetica et ulcerosa →bacterial superinfections
- Herpes simplex

Herpangina

- CoxsackievirusA
- Sudden fever with sore throath
- 1-2 mm diameter grayish lumps form and develop into vesicles with red surrounding
- Over 24 hours they become shallow ulcers
- Vesicles typically found on the posterior oropharynx

Mononucleosis infectiosa

Aka. Glandular fever

Epstein-Barr virus

- Infection in childhood produces milder symptoms
- In young adults it causes fever, sore throat, enlarged lymph nodes
- Spontaneous recovery within 2-4 weeks

HIV

- Infection from mother during birth or through breastfeeding
- Well controlled HIV doesn’t produce symptoms
- Known HIV+ doesn’t pose risks since medicated individuals have low virus count and are not contagious
- Unknow disease represents the real threat
- Virus has a low virulence

HPV

Fungal infections

Candida albicans

- Is part of normal oral flora and only invades mucosa if there is some change in the immunological or humoral environment (antibiotics or immunosuppresives)
  - Pseudomembranous candidiasis (thrush)
    - Common disease in newborns and children with chronic disease
    - Raised pearly white patches that can be rubbed off, leaving an erythematous or bleeding mucosa surface
    - Treatment: antifungal medication (nystatin, miconazole) systemically or topically applied

Recurring ulcerative lesions

Benign and non-contagious ulcers in otherwise healthy individuals

→multiple, erythematous, recurrent, small, round or ovoid ulcers with circumscribed margins, typically presenting first in childhood or adolescence

- Mikulicz aphta – most common
  - Separate multiple ulcers 1-2 mm diameter
- Recurrent aphtous ulcer major (Sutton)
  - Typically single and 2-3 cm, may cause scarring
- Cook aptha
  - Multiple small lesions in groups, very similar to herpes

Literature