# Periodontal Disease in the Pediatric Patient

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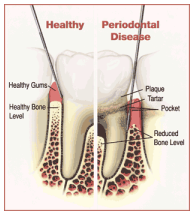
## Clinical Exam

- Gingiva
  - Color change
  - Edema
  - Bleeding on gentle probing
  - Gingival crevicular exudate
  - GI (Gingival Index) used to assess gingival health
- Plaque Accumulation
  - Several plaque indices assess portion of tooth covered in plaque
  - Disclosant provides excellent oral hygiene instruction tool
- Calculus
  - 10% of children, 1/3 of teens
  - Common areas- lingual of mandibular incisors and buccal of maxillary molars
- Bone loss
  - Height of interproximal crest 1-2 mm below CEJ on BW in health

## Clinical Exam

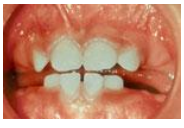
- Attachment Loss
  - Attachment Level- subtract the distance from the CEJ to the free gingival margin from the probing pocket depth (PPD) (distance from the free gingival margin to bottom of pocket)
  - Measure at 6 sites per tooth
  - Loss or gain of 2mm or more is clinically meaningful
  - Transient deep pockets are normal in transitional dentition and must be distinguished from true attachment loss by locating CEJ
  - More difficult to determine in younger patients because CEJ below free gingival margin
- Mucogingival problems
  - Determine width of attached gingiva- locate mucogingival junction and measure to free gingival margin
  - Note sites with less than 1 mm attached gingiva
- Periodontal Screening and Recording (PSR)
  - System designed for easier and faster screening of periodontal health for adults
  - Uses probe with colored band from 3.5 to 5.5mm
  - If band is even partially submerged, a complete periodontal exam is indicated
  - Can be used in children and adolescents, but erupting teeth give false positives





## Clinical Features of Gingiva in the Primary Dentition

- Papillary Gingiva
  - Interdental saddle area instead of cols where spacing exists
  - Well keratinized
- Marginal Gingiva
  - Sulcus depth greater
  - Free gingival margin thicker and rounder (due to cervical constriction of primary teeth)
  - Flaccid and retractable- immature connective tissue, immature gingival fiber system, increased vascularization
- Attached gingiva
  - Appears less dense and redder- thinner, less keratinized epithelium
  - Incidence of stippling- 35%
  - Greater width
  - Retrocuspid papilla-normal; 85% of children
- Alveolar mucosa
  - Redder
  - Width increases with age and eruption



## Radiographic Features of Periodontium in the Primary Dentition

- PDL
  - Wider and less dense
- Alveolar Bone
  - Less calcified, more vascular, fewer but thicker trabeculae
  - Larger marrow spaces, thinner lamina dura, flatter interdental crests
  - Blood supply, lymphatic drainage more extensive

## Host response

- Cell mediated: T-cell predominate response in children
- Sensitized lymphocytes produce bone resorbing lymphokines

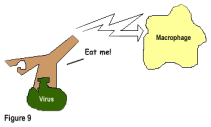


Figure 9

## Gingival Diseases

- Gingivitis does not always progress to periodontitis, but periodontitis is always preceded by gingivitis
- Plaque and non-plaque induced

## Gingival Diseases- Plaque Induced

- I-Gingivitis associated with plaque only
  - 1)With or without other local contributing factors
- II-Gingival diseases modified by systemic factors
  - 1)Associated with the endocrine system
    - A) Puberty
    - B) Menstrual cycle
    - C) Pregnancy
    - D) Diabetes
  - 2) Associated with blood dyscrasia
    - A) Leukemia
    - B) Others
- III-Gingival Diseases modified by medications
  - 1) Drug influenced gingival enlargements
  - 2)Drug influenced gingivitis
  - 3)Oral contraceptive associated gingivitis
  - 4)Others
- IV-Gingival diseases modified by malnutrition-
  - Ascorbic acid deficiency
  - Other

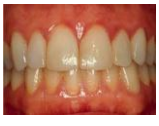
## Non plaque induced gingival lesions

- I) Gingival lesions of specific bacterial origin
- II) Gingival lesions of viral origin
- III) Gingival lesions of fungal origin
- IV) Gingival lesions of genetic origin
- V) Gingival manifestations of systemic conditions
  - 1) Mucocutaneous disorders
  - 2) Allergic reactions
- VI) Traumatic lesions
- VII) Foreign body reactions
- VIII) Not otherwise specified

## I-Gingivitis

- Definition: inflammation of gingival tissues
- Without bone loss or attachment loss
- Reversible with improved oral hygiene
  - Home care
  - Periodic professional reinforcement
- Clinical signs:
  - Erythema
  - Bleeding on probing
  - Edema
- Early primary dentition- gingivitis uncommon
- Peaks during puberty (60% of teenagers-bleeding on probing)
- After puberty declines slightly and constant into adulthood

## Gingivitis



- Etiology- bacterial plaque
  - Early species- gram positive cocci and small rods (streptococci and actinomyces)
  - Later replacements- filamentous forms, spirochetes
  - Reactivity gradually increases with age
  - May be related to steroid hormones
- Local factors that contribute to gingivitis in children:
  - Crowded teeth
  - Orthodontic appliances
  - Mouth breathing
  - Eruption
  - Calculus (10% of children, 1/3 of teens)

## Gingivitis in Children


- Occurs to varying degrees, increases with age, eruption, puberty
- Rounded gingival margins accentuate inflammatory changes
- Reversible with improved oral hygiene
- Does not occur to same degree as adults with comparable plaque

## Gingivitis-Treatment

- Scaling and root planing
- Topical antibacterial agents
- If gingivitis remains following removal of plaque and local factors, evaluate for systemic factors

## Objectives of Scaling and Root Planing

- Removal of dental calculus and plaque
- Reduce bacteria below a threshold level capable of initiating clinical inflammation
- Success is determined by evaluation of tissues following treatment and during maintenance phase


## Chronic Inflammatory Gingival Enlargement

- Long standing gingivitis in young patients
- Clinical features
  - Ballooning of interdental papilla and/or marginal gingiva
  - Gingiva is red or bluish/red, soft and friable with smooth shiny surface and bleeds easily
- May be generalized or localized
- Caused by prolonged exposure to plaque
- Common local contributory factors-
  - Mouth breathing
  - Orthodontic appliances
- Often resolves when plaque control is instituted

## Plaque Induced Gingival Enlargement

- Clinical features
  - Enlargement of interdental papilla and or marginal gingiva
  - Ranges from pale and fibrotic to red and friable
  - Generalized or localized
- Caused by prolonged exposure to plaque
- Common local contributing factors
  - Mouthbreathing
  - Orthodontic appliances
- Treatment
  - Thorough oral hygiene routines, use of powered toothbrush
  - Gingivectomy or gingivoplasty may be required

## II-Gingival Diseases Modified by Systemic Factors

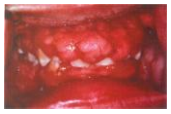
A) Associated with the Endocrine System

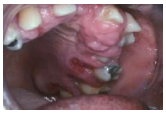
- 1) Puberty- dramatic rise in steroid hormone levels (esp. estrogen and progesterone) has transient effect on inflammatory status of the gingiva
- 2) Menstrual cycle- inflammatory changes occur at ovulatory surge
- 3) Pregnancy-during 2nd or 3rd trimester

## 4) Insulin Dependant Diabetes Mellitus (Type 1)

- Insulin Dependant Diabetes Mellitus Associated Gingivitis
  - Greater response to local factors
  - Consistent in children with poorly controlled type 1 diabetes
  - Level of diabetic control is most important aspect
  - Plaque control can limit severity
- Higher rates of periodontal disease in IDDM
- Increased risk and earlier onset of periodontitis in both IDDM and NIDDM

## III-Drug Induced Gingival Diseases 1)Drug Influenced Gingival Enlargements





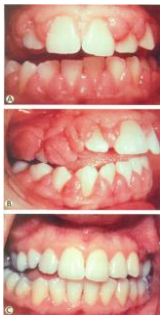
- **Phenytoin** (Dilantin, anti-epilectic)
- **Cyclosporin** (immunosuppressant)
- **Calcium channel blockers** (Nifedipine, Nitrendipine, diltiazem)

## III-Drug Induced Gingival Diseases 1)Drug Influenced Gingival Enlargements

- Inter and intra patient variation
- Higher prevalence in children
- Onset within 3 months of starting drug
- Change in gingival contour leads to modification of size
- Predilection for anterior gingiva
- Enlargement first observed at interdental papilla
- May progress to cover crowns
- Pronounced inflammation in relation to amount of plaque
- Reduction in plaque can limit severity
- Overgrowth is fibroepithelial
- Painless overgrowth, fibrous, firm, pale pink, little tendency to bleed

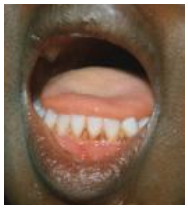
## Drug Influenced Gingival Enlargements Treatment

- Regresses and may disappear after cessation of drug therapy
- Replace with alternate drug if possible
- Professional prophylaxis and rigorous home care
- Daily use of chlorhexidine may be beneficial
- Surgery -gingivectomy or flap with internal bevel or gingivoplasty
  - Will recur
  - Surgery indicated- appearance unacceptable to patient, interferes with function, pocket depth cannot be maintained in healthy state



## IV-Gingival Diseases Modified by Malnutrition

- Rare in US or developing countries
- Precise role of nutrition in initiation/progression of disease yet to be determined
- Individuals on restricted diets may be at risk for ascorbutic gingivitis (avitaminosis C)
- May be a concern in anorexic/bulimic patients



## Vitamin C Deficiency Gingivitis

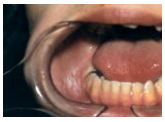
- Edematous, spongy gingiva, clinical appearance of non-specific gingivitis
- Spontaneous bleeding
- Impaired wound healing
- Dental management
  - Treatment of the underlying deficiency
  - Plaque control

## Non-Plaque Induced Gingival Lesions

- I-Gingival lesions of specific bacterial origin
  - Nieserria gonorrhea
  - Treponema pallidum
  - Streptococcal species
- II-Gingival lesions of viral origin
  - Primary herpetic gingivostomatitis
  - Recurrent oral herpes
  - Varicella zoster
- III-Gingival lesions of fungal origin
  - Candida species
  - Linear gingival erythema (manifestation of candidiasis in HIV positive individuals)
  - Histoplasmosis
- IV-Gingival lesions of genetic origin
  - Hereditary gingival fibromatosis

## Non- plaque Induced Gingival Lesions V-Gingival Manifestations of Systemic Conditions

1)Mucocutaneous disorders
- Lichen planus
- Pemphigoid
- Pemphigus vulgaris
- Erythema multiforme
- Lupus erythematous
- Drug induced
- Other



2)Allergic reactions
- Dental restorative materials
  - Mercury
  - Nickel
  - Acrylic
- Oral care products
  - Dentrifice
  - Mouthwash
  - Tartar control agents
- Chewing gum additives
  - Cinnamon
- Food and food additives

## Non-Plaque Induced Gingival Lesions

- VI-Traumatic lesions (factitious, iatrogenic, accidental, self mutilation)
  - Chemical injury
  - Physical injury
  - Thermal injury

## Herpes Simplex Virus Type I

- Includes gingivitis
- Febrile, pain, lymphadenopathy
- Diagnosis- clinical appearance of soft tissues
- Viral culture provides definitive diagnosis however not routinely performed
- Treatment- palliative therapy
- Self limiting and resolves 7-10 days
- Systemic acyclovir for immunocompromised patients with herpetic gingivitis

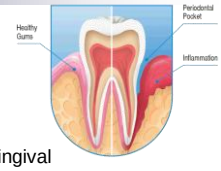
## Classification of Periodontitis

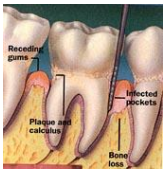
- I-Chronic Periodontitis (localized, generalized) (more common in adults)
- II-Aggressive Periodontitis (localized, generalized) (may be more common in children and adolescents
- III-Periodontitis as manifestation of systemic diseases
  - 1) Hematologic disorders
  - 2) Genetic disorders
  - 3) Not otherwise specified
- IV-Necrotizing periodontal diseases (NUG, NUP)
- V-Abscesses of the periodontium (gingival, periodontal, pericoronal)
- VI-Periodontitis associated with endodontic lesions
- VII-Developmental or acquired deformities and conditions
  - 1) Localized tooth related factors
  - 2) Mucogingival deformities/ conditions around teeth
  - 3) Mucogingival deformities/ conditions on edentulous ridges
  - 4) Occlusal trauma

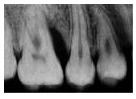
## Periodontitis- Further Characterized by:

- Degree of attachment loss
  - Slight
  - Moderate
  - Severe
- Extent
  - Localized
  - Generalized
- Post treatment status
  - Recurrent
  - Refractory

## Periodontitis



- Loss of gingival attachment
- Loss of bony support
- Chronic gingivitis (adults)
- Responds to local therapy
- Age 5-11 1%
- Age 12-17 20%

## Periodontitis- Therapeutic Approaches

- Anti infective treatment
  - Designed to halt progression of attachment loss by removing etiologic factors
- Regenerative therapy
  - Includes anti infective treatment and is intended to restore structures destroyed by disease
- Periodontal maintenance essential to both approaches

## Periodontitis-Objectives of Therapy

- Halt disease progression
- Resolve inflammation
- Reducing etiologic factors below the threshold capable of producing breakdown
- Allow repair of the affected region

## Periodontitis- Treatment

- Scaling and Root Planing
- Pharmacological Therapy
  - Systemic drug administration
  - Local Delivery
- Surgical Therapy
- Regenerative Surgical Therapy

## Scaling and Root Planing

- Beneficial
  - Reduce clinical inflammation
  - Microbial shift to less pathogenic subgingival flora
  - Decreased probing depth
  - Gain of clinical attachment
  - Less disease progression
- Some sites do not respond to therapy
  - Root anatomy- concavities
  - Furcations
  - Deep probing depths
- Reevaluation several weeks later
- Must be combined with personal plaque control
- Contributing systemic factors must be addressed

## Systemic Drug Administration

- Systemic antibiotics indicated for:
  - Multiple sites unresponsive to mechanical debridement
  - Acute infections
  - Medically compromised patients
  - Presence of tissue invasive organisms
  - Ongoing disease progression
- Identify pathogenic organisms
- Antibiotic sensitivity testing
- NSAIDs
- Sub-antimicrobial dose doxycycline
- Risks and benefits
- **Since patients with chronic periodontitis respond to conventional therapy it is unnecessary to routinely administer systemic medications such as antibiotics, NSAIDs or sub-antimicrobial dosing with doxycycline**

## Local Delivery

- Controlled delivery of chemotherapeutic agents within periodontal pockets can alter pathogenic flora and improve clinical signs of periodontitis
- Benefits
  - Delivered at the site of disease activity at a bactericidal concentration
  - Facilitates prolonged drug delivery

## Surgical Therapy

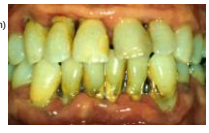
- Provides better access for removal of etiologic factors
- Reduce deep probing depths
- Regenerate or reconstruct lost periodontal tissues

## Regenerative Surgical Therapy

- Use of adjunctive surgical technique devices and materials
- Chemical agents that modify the root surface while promoting new attachment- variable results
- Bone grafting
- Guided tissue regeneration (GTR) with or without bone replacement grafts
- Biologically engineeered tissue inductive proteins (eg growth factors, extracellular matrix proteins and bone morphogenic proteins)
- NO smoking

## I-Chronic Periodontitis Formerly-Adult Onset Periodontitis

- Features
  - Loss of attachment and bone
  - Can be arrested
- Prevalence
  - ½ of adult population affected
  - 20% of 14-17 year olds have attachment loss of at least 2mm in one or more sites
  - Often begins in adolescence
  - Increases with age
  - Can be localized(<30% of dentition) or generalized >30% of dentition)
- Pathogenesis
  - Bacterial plaque dependant
  - Polymicrobial infection
  - Most common-Porphyromonas gingivalis
  - Neutrophil primary host defense mechanism
  - Host inflammatory response contributes to disease
- Treatment
  - OHI
  - Scaling/root planing
  - Correction of local contributory factors (overhang restorations, calculus)
  - Lasers (evidence insufficient to suggest any specific laser approach superior to traditional mechanical therapy)
- Prevention
  - Maintain good OH
- Smoking, Diabetes- RISK FACTORS
- Also increases with age and male gender

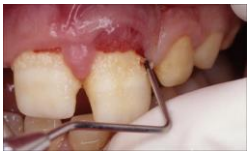


## Chronic Periodontitis

- Can be localized(<30% of dentition) or generalized (>30% of dentition)
- Most prevalent in adults
- Can occur in children and adolescents
- Low to moderate rate of progression that may include periods of rapid destruction
- Severity
  - Mild: 1-2mm of clinical attachment loss
  - Moderate: 3-4 mm of clinical attachment loss
  - Severe: >= 5 mm clinical attachment loss

## II-Aggressive Periodontitis

- Primary features
  - Otherwise healthy patient
  - Rapid loss of attachment and bone
  - Familial aggregation
  - ?Genetic Predisposition
  - Independent of age of onset
  - Localized and generalized forms
- Secondary features
  - Amounts of microbial deposits inconsistent with severity of periodontal tissue destruction
  - Elevated Aa (Actinobacillus actinomycetemcomitans) and P. gingivalis (some populations)
  - Phagocyte abnormalities
  - Hyperresponsive macrophage phenotype
  - Progression of attachment/bone loss may be self arresting



## Localized Aggressive Periodontitis (LAgP)- Primary Dentition

- Formerly Localized prepubertal periodontitis (LPP)
- Features
  - Attachment loss and bone loss around primary teeth
  - Affects only some deciduous teeth
  - Most commonly affects primary molars
  - Mild to moderate inflammation (not prominent feature)
  - May have less plaque and inflammation than seen in chronic periodontitis
  - Heavier than average plaque deposits
  - Most commonly in African Americans
  - Children otherwise systemically healthy
  - Commonly diagnosed during late primary or early transitional dentition
  - Prevalence less than 1%
  - May progress to LAgP in the permanent dentition

## Localized Aggressive Periodontitis (LAgP)- Primary Dentition

- Suggested etiologic factors
  - Leukocyte chemotactic defect
  - Cementum defect
  - Usually (but not always) associated with Aa
  - Has not been studied as much as LAP in permanent dentition
    - Causative bacteria has not been identified
- Dental Management
  - Little data
  - Scaling and root planing, extraction of primary teeth
  - Antibiotic therapy: amoxicillin +/- metronidazole for 7-10 days or azithromycin for 3-5 days
    - Not tetracycline

## Localized Aggressive Periodontitis (LAgP)-Permanent Dentition

- Formerly Localized Juvenile Periodontitis (LJP)
- Prevalence is 1%
  - .2% Whites
  - 2.6% in African Americans
  - Increase in Latinos
- Features:
  - Circumpubertal onset typically (not age dependant) (10-15 years of age)
  - Robust serum antibody response to infecting agents (Aa)
  - Localized to permanent first molar/incisor (loss of attachment and bone) and no more than 2 other teeth
  - Minimal inflammation
  - Conflicting data on plaque and calculus
  - Patient otherwise systemically healthy
  - Frequently preceded by bone loss in primary dentition (LPP) (50%)
  - Probably same disease as LAgP in primary dentition
  - May have less plaque and inflammation than seen in chronic periodontitis-often first detected at 10-15 years olf

## Localized Aggressive Periodontitis (LAgP)-Permanent Dentition

- Radiographic signs (Distinctive):
  - Vertical bone loss around molars
  - Horizontal bone loss around incisors
  - Rapid rate of progression-3x that of chronic periodontitis
- Etiology
  - Genetic basis? Familial aggregation
  - Aa (most but not all cases)
  - No single species in all cases
  - Depressed neutrophil chemotaxis in 70%
  - Possible defect in phagocytosis
  - Over-reactive monocyte response
  - Genetic defects in gene coding IgG2

## Localized Aggressive Periodontitis (LAgP)-Permanent Dentition

- Diagnosis
  - Clinical: attachment loss on at least two permanent first molars and incisors with and no more than 2 teeth other than those molars or incisors
  - Laboratory: cultures and/or DNA probes
- Treatment:
  - Scaling, curettage, root planing
  - Antibiotic therapy (metronidazole most effective, may be used in combination with amoxicillin, tetracycline) (local antibiotic therapy not effective)
  - Microbiologic monitoring needed to ascertain eradication of Aa
  - Surgery
  - Regenerative techniques: root conditioning composite graft, ePTFE membranes

## Generalized Aggressive Periodontitis (GAgP)

- Previously Generalized Juvenile Periodontitis
- Prevalence 0.15% or less in US
- Greater prevalence in males, African Americans
- No association with age?
- Considered a disease of adolescents and young adults (12-30), onset often circumpubertal
  - In the US prevalence in adolescents(14-17) is 0.13 percent
- Features
  - Poor serum antibody response to infecting agents
  - Pronounced episodic nature of attachment/bone loss

## Generalized Aggressive Periodontitis (GAgP)

- Rapid, severe periodontal destruction around most teeth
- Clinical diagnosis:4 or more mm of attachment loss around 8 or more teeth, at least 3 teeth other than molars and incisors
- More gingival inflammation than LAgP
  - Inflammation can be severe
- More local irritants (plaque, calculus) than LAgP
- Probably progression of LAgP
- Smoking is a risk factor
- Radiographic signs: rapidly progressing bone loss around multiple teeth including molars/incisors

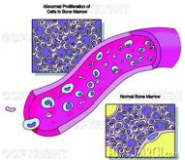
## Generalized Aggressive Periodontitis (GAgP)

- Etiology:
  - Neutrophil chemotactic disorder
  - Aa, P. gingivalis, E. corrodens
  - Microbiological profile similar to chronic disease
  - Subgingival bacterial cultures: non motile facultatively anaerobic gram - rods
- Treatment
  - Scaling, surgery, curettage, root planing, antibiotics
  - Does not always respond to conventional mechanical and antibiotic therapy
  - Culture and sensitivity helpful in refractive cases

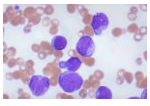
## III-Periodontitis as a Manifestation of Systemic Disease

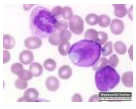
- 1) Hematological Disorders
  - Acquired neutropenia
  - Leukemia
  - Others
- 2) Genetic Disorders
  - Familial/cyclic neutropenia
  - Down Syndrome
  - Leukocyte adhesion deficiency syndromes
  - Papillon-Lefevre syndrome
  - Chediak Higashi syndrome
  - Langerhans cell Histiocytosis
  - Infantile genetic agranulocytosis
  - Hypophosphatasia
  - Glycogen Storage Disease
  - Cohen syndrome
  - Ehlers Danlos syndrome (Type IV and VIII)

## Leukemia

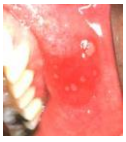


- Most common from of childhood cancer
- ALL (acute lymphoblastic leukemia)
  - Most common
  - Best prognosis
- AML (acute myeloid leukemia)
  - 20% of childhood leukemia
  - Poorer long term survival rate
- AML (not ALL) may present with gingival enlargement (infiltrates of leukemic cells), Lesions- blue/red and may invade bone
- Gingiva hyperplastic, edematous, bluish red
- Fever, malaise, bone or joint pain, gingival or other bleeding
- Petechiae and mucosal ulcerations in any form of leukemia
- Initial diagnosis by CBC





## Neutropenia



- Decreased circulating PMNs
- Several forms
  - Cyclic neutropenia
  - Chronic benign neutropenia of childhood
  - Chronic idiopathic neutropenia
  - Familial benign neutropenia
- Can be cyclic, transient or persistent
- Cyclic/ Familial Neutropenia
  - Reduction of PMNs every 19-21 days typically (14-30 days more inclusive)
  - Neutropenia 5-10 days duration
  - Severe ulcerative gingivitis
  - Alveolar bone loss

## Neutropenia

- Periodontal symptoms
  - Severe gingivitis with ulcerations
  - Attachment loss and alveolar bone loss
  - Early loss of primary teeth
  - Severe periodontal disease in the permanent dentition
- History of other recurrent soft tissue infections
  - Otitis media, respiratory and skin infections
- Diagnosed by white blood cell differential count
  - Depressed neutrophils
- Dental management
  - Rigorous local measures to control plaque
  - Antibiotic therapy
  - Extraction of affected teeth
  - Periodontal treatment as indicated
  - Systemically administered granulocyte colony stimulating factor (G-CSF) to treat underlying cause

# Down Syndrome

- Prevalence 60-100%, all under age 30, increases with age
- Primary dentition involved in 36%
- Mandibular incisors often affected
- Higher prevalence in trisomy 21 than general MR population
- Higher rates of ANUG in institututionalized
- Susceptibility to periodontitis appears to be due to innate immune system abnormalities
- Dental management
  - Oral hygiene routines
  - Periodontal treatment as indicated based on periodontal diagnosis
- Probable pathogenesis
  - Capillary fragility
  - Chemotactic/phagocytic defects of PMNs ("lazy leukocyte syndrome")
  - Low numbers of mature T cells
  - Abnormal thymus
  - Early colonization by pathogenic bacteria

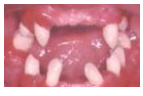
# Leukocyte Adhesion Deficiency (LAD)

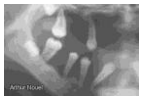
- Rare, autosomal recessive genetic disease
- Etiology:
  - Leukocyte surface glycoprotein defect
    - Poor leukocyte adherence
    - Poor migration to infection sites
    - Impaired phagocytic function
- Patients susceptible to bacterial infections
  - Including periodontitis (in primary and young permanent dentition)
  - Frequent respiratory, ear, skin and other soft tissue bacterial infections

# LAD

- Formerly Generalized Prepubertal Periodontitis
- Dental symptoms manifest in early primary dentition
- Involves all primary teeth, if untreated will involve permanent teeth
- Highly acute inflammation, clefting, recession
- Rapid destruction of bone
- Severe generalized periodontitis refractory to treatment
- Stem cell transplantation can be curative
- Treatment
  - Oral hygiene measures
  - Antibiotic therapy
  - Extractions of affected teeth
- Due to chronic problems with illness, adequate compliance difficult

# Papillon-Lefevre syndrome





- Autosomal recessive
- 1 of 19 different forms of palmoplantar keratoderma
- Rare
- Erythematous hyperkeratosis of palms and soles (variable)
- Nail dystrophy
- Ectopic calcification of dura periodontitis
- Rapid loss of bone and attachment
- Inflammation can be severe
- Premature loss of primary and permanent teeth
- Aa infection, also Bacteroides and Fusobacterium
- Treatment-
  - Antibiotic therapy
  - Extraction of affected teeth
  - Local measures to control plaque

# Chediak Higashi Syndrome

- Autosomal recessive
- Rare
- Neutrophils with giant cytoplasmic granules
- Recurrent infections
- Severe gingivitis and periodontitis
- Oculocutaneous albinism
- Photophobia
- Nystagmus
- Peripheral neuropathy

# Langerhans Cell Histiocytosis
# Formerly Histiocytosis X
# Non Lipid Reticuloendothelioses

- Group of disorders with variable symptoms resulting from abnormal proliferation and dissemination of histiocytic cells of the Langerhans system
- Disorder of mononuclear phagocytes
- Multiple hard and soft tissue lesions containing histiocytes and eosinophils
- Diagnosis by biopsy
- Oral manifestations in 10% of patients
  - Necrotic gingivitis
  - Furcation bone loss
  - Radiolucent lesions of mandible and cranium
  - Bone lesions produce "floating teeth"
  - Gingival enlargement
  - Ulceration
  - Mobility of teeth with alveolar expansion

Treatment-radiation, surgery, systemic chemotherapy

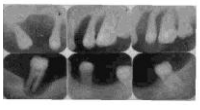


Figure 2 - Periapical radiographs showing well-defined radiolucent images comprising alveolar bone, providing the radiographic image of "floating teeth".

## Histiocytosis

- Rare disorder of childhood
- Presentation as infiltration of bones, skin, liver and other organs by histiocytes
- 10-20% of cases initial infiltrates are in oral cavity- usually the mandible

## Langerhans Cell Histiocytosis

- Letterer-Siwe (acute disseminated)- most severe, affects infants, prominent skin and visceral involvement
- Hand-Schuller-Christian (chronic disseminated)- children>3 years, mostly bony sites, skull lesions, diabetes insipidus, exopthalmos
- Eosinophilic Granuloma (acute localized)- older children, most benign form

## Hypophosphatasia – Rathbun Syndrome

- Autosomal recessive
- 5 groups: perinatal (lethal), infantile (severe), childhood (milder), adult (tarda) and odontohypophosphatasia
- Phenotypes range from premature loss of primary teeth to severe bone abnormalities leading to neonatal death
- Early loss of primary teeth may be first clinical sign in mild forms
- Earlier presentation of symptoms the more severe the disease
- Low serum tissue-non specific alkaline phosphatase
- High urinary phosphoethanolamine

## Hypophosphatasia

- Genetic disorder
- Premature loss of primary teeth (especially single rooted teeth)
- Loss of teeth due to cementum defect
  - Weakened attachment of tooth to bone
- Clinical features in permanent dentition similar to LAP
- Dental prognosis for permanent teeth is good
- Typical presentation
  - Primary incisors exfoliate before age 4
  - Teeth exfoliate with intact roots, usually before root formation complete
  - Teeth lost in order of eruption
  - Other primary teeth are affected to varying degrees
  - Permanent dentition may be normal in odontohypophatasia
- Enlarged pulp chambers, acementogenesis, dentinal dysplasia

## Hypophosphatasia

- Systemic treatment
  - Enzyme replacement therapy, Asfotase Alpha, approved for perinatal, infantile and juvenile onset hypophasphatasia
- Dental management
  - Supportive therapy to address concerns and prevent long term consequences or early tooth loss

## IV-Necrotizing Periodontal Diseases (NUG, NUP)(NPD)

- Rapid Onset
- Painful gingivitis with interproximal and marginal necrosis and ulceration
- NUG may progress to NUP in immunocompromised individuals
- Febrile
- Incidence
  - Late teens and early 20s in North America and Europe
  - Young children in undeveloped countries
  - Varying but low frequency in North America and Europe (<1%)
  - 2-5% in developing areas of Africa, Asia and South America
- Predisposing factors
  - Malnutrition
  - Viral infection(including HIV)
  - Stress
  - Lack of sleep
- Spirochetes and Prevotella intermedia
- Treatment
  - Local debridement (ultrasonic scaling excellent)
  - Antibiotic therapy may be indicated (Penicillin, Metronidazole)
  - NSAIDs for pain

## NUG/NUP

- NUG
  - Specific bacterial accumulations
  - Lowered host resistance
  - Personal plaque control and professional debridement
  - Systemic antibiotics if lymphadenopathy or fever with oral symptoms
  - Chemotherapeutic rinses during initial treatment stages
- NUP
  - Manifests as rapid necrosis and destruction of gingiva and periodontal attachment apparatus
  - Gingival bleeding and pain and represents an extension of NUG
  - HIV + and –
  - Management
    - Debridement
    - Irrigation with antiseptics(eg povidone iodine), antimicrobial mouth rinses (eg chlorhexidine) and systemic antibiotics

## V-Abscesses of the Periodontium (Gingival, Periodontal, Pericoronal)

- Gingival abscess
  - Localized, painful lesion of marginal gingiva or interdental papilla
  - Sudden onset
  - Bacterial infection following gingival trauma , typically caused by embedded foreign object
    - Popcorn hull
    - Fingernail fragment
  - Treatment-
    - Debridement and establish drainage
    - Chlorhexidine irrigation

## Pericornitis

- Inflammation of gingiva covering partially erupted tooth
- Most common around erupting 3rd molars
- Food trap, environment conducive to bacterial growth
- Pericoronal flap becomes inflamed and swollen
- Enlarged flap traumatized by occlusion, very painful
- Dental Management- debridement, antibiotic therapy for systemic involvement, chlorhexidine irrigation

## Mucogingival Defects

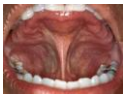
- Pocket depth exceeds width of attached keratinized gingiva
- Lower incisor most common location
- May result from labial positioning of tooth through band of attached gingiva

## Defects of Attached Gingiva

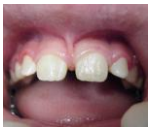
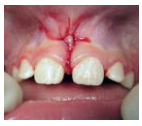
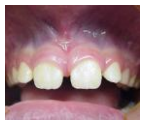
- Mandibular incisors can erupt labial to alveolar ridge leading to a narrow band of attached gingiva
- Small loss of attachment -> mucogingival defect and recession
- Loss of attachment and recession with a labially malpositioned tooth may be called **stripping**
- Other factors that cause recession
  - Smokeless tobacco
  - Habit related to self induced injury
  - Nail biting
  - Tooth brushing
  - Orthodontic movement
- Treatment for severe recession:
  - Gingival graft (commonly from palate)
    - If defect not severe should postpone until after ortho
  - Ortho movement of labially malpositioned tooth

## Frena

- Common finding in children
- Prominent maxillary frenum and midline diastema
  - Immediate treatment usually unnecessary
  - Treatment delayed until permanent incisors and cuspids are erupted (allows natural closure)
  - If ortho planned- surgical treatment should be postponed until diastema closed
  - If appearance unacceptable after closure then frenectomy indicated
  - Laser
- Maxillary and mandibular labial frena-
  - Treatment indicated to prevent
    - Stripping of the labial tissue
    - Loss of alveolar bone
    - Loss of tooth
- Restrictive lingual frenum
  - Tongue tie or Ankyloglossia
  - If limits normal tongue mobility treatment may be indicated
  - Will also make speech easier



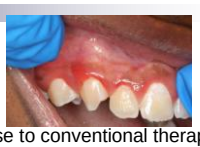
## Frenectomy

## Localized Juvenile Spongiotic Gingival Hyperplasia (LJSGH)

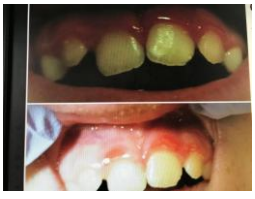
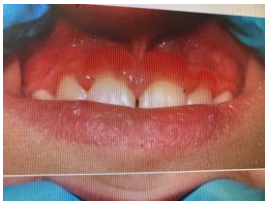
- Recent
- Benign condition
- Affects the gingiva of children and young adults
- Clinically distinctive
  - Presents as a localized area of erythema on the attached gingiva, with a subtly papillary surface architecture
- Generally biopsied
- Prominent intercellular edema (spongiosis) and neutrophil infiltrate
- Lack of resolution with conservative oral hygiene therapeutic measures
- Esthetic concerns
- Histopathology
  - Subtle papillary epithelial hyperplasia
  - Acute inflammation
  - Numerous engorged capillary vascular spaces in the lamina propria
  - Clinical correlation is necessary to make the diagnosis

## LJSGH



- Lack of a good clinical response to conventional therapy
- Excisional biopsies were performed to establish the diagnosis
- Plaque control reinforced
- Additional antiseptic local treatment was administered
- Persistence of some bright reddish gingival masses in sone of the patients these lesions were treated by surgical excision
- Overall clinical outcome was good and stable after one year

## LJSGH

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