Aggressive Periodontitis

Localized & Generalized Forms

Periodontitis

- Destruction of the attachment apparatus
- Diagnosed by;
  - Probing pocket depth
  - Clinical attachment loss
  - Recession
  - Mobility
  - Furcation involvement
  - X-ray; bone loss

Chronic vs Aggressive

- Rate of destruction (RoD);
  - Faster in aggressive types; 3 – 4 X faster
- RoD depends on the;
  - Age
  - Severity
- Minor factors
  - Calculus, plaque; less in aggressive periodontitis
  - Incisor/first molar destruction; a feature of agg. Period.

Aggressive Periodontitis

- *Common Findings:*
  - Patient otherwise clinically healthy, usually <30 years of age
  - Characterized by rapid attachment loss (inconsistent with amount of plaque)
  - Absence of large amounts of plaque & calculus
  - Family history – genetic trait

Aggressive Periodontitis

- *Other Findings (not universal):*
  - *A.a.* found in diseased sites
  - Host response abnormalities (phagocytosis, chemotaxis)
  - Hyperactive macrophages
    - *Produce excess amounts of prostaglandins, interleukin – 1*
  - Disease may be self-arresting

Clinical Features of Localized Aggressive Periodontitis

- Formerly known as *localized juvenile perio*
- Onset of disease occurs between puberty and 20 years of age
- Attachment loss (3-4x faster than in chronic perio) affects:
  - First molars
  - Incisors
- Clinical inflammation may not be obvious
- Minimal plaque that rarely mineralizes
  - However contains elevated levels of *A.a.* and *P.g.*

Clinical Features of Localized Aggressive Periodontitis

- Maxillary incisors migrate in distolabial direction ↖ diastema
- Increasing mobility of affected teeth
- Periodontal abscess formation
- Sensitive root surfaces

Bacteria Associated with LAP

- Elevated levels of *A.a.* found in active sites (low numbers in healthy sites)
  - Produce leukotoxins, collagenase, & other immunosuppressive factors that help it to evade host defense mechanisms
- Incidence of *A.a.* found to be greater in younger persons compared to older ones
- Younger patients experience more destruction in a shorter period of time
- *Important to diagnosis condition in early stages*

Site Specific Destruction

- Some reasons why disease activity affects certain teeth:
  - *#1: A.a.* colonize first permanent teeth to erupt
    - *Evade host defenses*
    - *Following initial attack, host responds*
    - *Antibodies produce which improve phagocytosis of bacteria*
    - *This may prevent colonization of other sites*

Site Specific Destruction

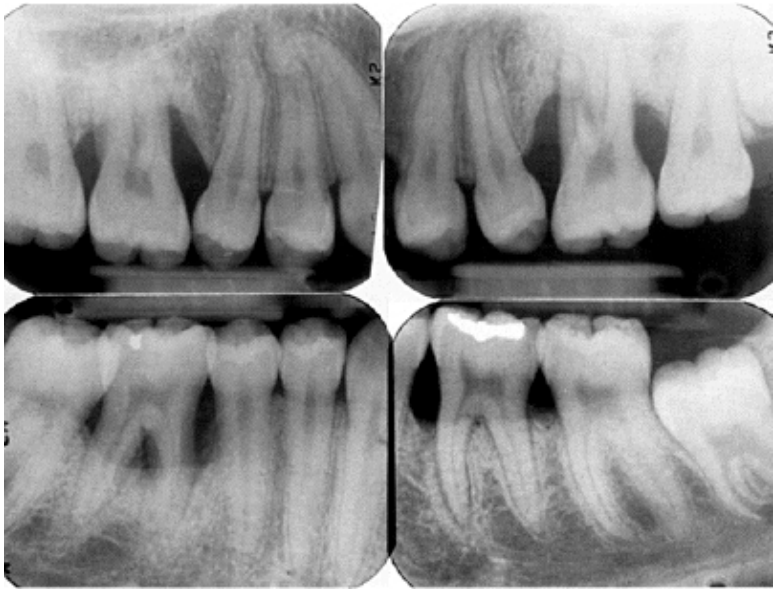
- Additional reasons:
  - #2: *A.a.* may lose its ability to produce leukotoxin
    - *This may slow or arrest the disease process*
  - #3: Antagonistic bacteria
    - *Anti-A.a. bacteria may colonize sites & prevent A.a. from colonizing other sites in mouth*
    - *Localizes the infection & tissue destruction*

Site Specific Destruction

- Additional reasons:
  - #4: Denuded root surfaces
    - *The root surfaces of clients with LAP are often denuded (absence of cementum)*
    - *Allows bacteria to penetrate the root and colonize the site*

Radiographic Evaluation

- Vertical bone loss affecting:
  - Usually bilateral affecting first permanent molars & incisors,
  - Vertical loss of bone in an “arc-shape” extending from the distal of the 2nd premolar to the mesial of the 1st molar



Clinical Features of Generalized Aggressive Perio

- Limited information available due to reclassification of conditions
- Includes conditions formerly known as gen. juvenile and rapidly progressive periodontitis
- Usually affects persons 30 years & younger but can affect older persons
- Attachment loss (Bone loss) affects at least 3 teeth other than first molars & incisors
- Episodic nature to disease
  - Periods of inactivity may last weeks, months, or years

Clinical Features of Generalized Aggressive Perio

- Often plaque is minimal but contains high levels of:
  - *A.a.*
  - *P.g.*
  - *F.n. & C.r.*
  - *Spirochetes*
- Episodic nature of disease produces two different tissue responses

Clinical Features of Generalized Aggressive Perio

- *Destructive phase:*
  - Tissue appears severely inflamed, ulcerated & fiery red
  - Bleeding with or without stimulation
  - Suppuration
  - Active attachment & bone loss

Clinical Features of Generalized Aggressive Perio

- *Non-destructive phase:*
  - Tissues appear pink with some stippling
  - Lack of inflammation
  - Probing will reveal deep pockets
  - Bone & attachment levels relatively stable

Associated Systemic Complications

- Some individuals with GAP may exhibit:
  - Weight loss
  - Mental depression, general malaise
- Systemic conditions may predispose patient to GAP, these include:
  - Chronic neutrophil defects, leukocyte adherence deficiency
- Functional defects of PMNs, monocytes or both ↖ impaired chemotaxis & phagocytosis

Radiographic Evaluation

- Severe bone loss affecting minimal number of teeth *OR*
- Majority of teeth affected by advanced bone loss

Prevalence of Aggressive Periodontitis

- Prevalence estimates below 1% (U.S. & other countries)
- Prevalence for both types higher among African-Americans
- Gender differences unclear
- Distribution of disease by gender among race groups↖
  - Prevalence higher for African-American males compared to females
  - Reverse is true among whites

What Puts a Person at Risk?

- *A.a.* found in large numbers in LAP
- *A.a.* produces a strong leukotoxin; kills neutrophils
- Different strains of *A.a.* produce different levels of leukotoxin
  - Highly toxic strains (Jp2) produce greater numbers of leukotoxin
  - People with the disease more likely to have highly toxic strains (African-Americans in particular)

Risk

- Defective neutrophil function
- Depressed neutrophil chemotaxis & phacytosis common for both forms
- Neutrophil dysfunction has genetic basis
- *BUT,* not all people with this dysfunction have aggressive perio
- *AND* not all people with aggressive perio have the dysfunction

Aggressive Periodontitis - Treatment

- Depends on type and degree of destruction
- More severe forms have poorer prognosis
- Three phases;
  - Cause-related
  - Corrective
  - Maintenance

Treatment for LAP

- Extraction of involved teeth (depends on severity of tissue loss)
- Periodontal therapy:
  - Plaque control instruction + Motivation
  - Debridement with or without flap surgery
  - Irrigation with CHX, home rinsing with CHX
  - Bone grafts, root resections, hemisections
- Frequent maintenance visits
  - 1/month for 6 months, then every 3 months

Treatment for LAP

- **Antibiotic therapy:**
  - Adjunctive therapy often required to eliminate *A.a.* from tissues
  - Metronidazole combined with augmentin; 2 weeks
  - Tetracycline (250 mg qid for 2 weeks)
  - Doxycycline
- The earlier the condition is diagnosed, the sooner treatment can begin; outcome often more predictable

Treatment for GAP

- Careful monitoring of younger patients with GAP; rate of disease progression is often faster
- Maintenance every 3 weeks or less is recommended if disease in active phase
- Periodontal therapy:
  - Debridement in combination with antibiotic therapy, strict plaque control, CHX irrigation & rinsing
  - Periodontal surgery

Treatment for GAP

- Antibiotic therapy:
  - Highly recommended that microbial diagnostic & susceptibility testing be done
  - Combination therapies include:
    - Metronidazole/augmentin
    - doxycycline
    - tetracycline
  - Local therapies in the form of gels, chips or fibers (local antibiotics delivery)