Biofilm levels of discernable plaque would match the disease state of the patient
Supra and sub gingival calculus

Anticipate gram - anaerobes but no characterization to the flora

Overhangs, furcations, grooves

(more rapid)
More putative response (difference in ability to mount an appropriate defense for the microflora present)
Primary means no systemic disease

Expect but may not be universally present
Unlike chronic where there is a match with the microbial deposits/local factors, there is an inconsistent relationship
Not see the signs of inflammation and evidence of the bioburdent that we may see in a chronic periodontitis patient
Defense not as effective (phagocyte abnormalities)
Greater overall response cascade but phagocytes not phagocytosing
Occasionally patients "burn out": may have at young age, but once hit 20s gets quiet

Diagnosis based on:
clinical, radiographic and historical data

There are two forms:
- Localized Aggressive Periodontitis
- Generalized Aggressive Periodontitis
- Old scheme = Early Onset Periodontitis
  Early onset, pre-pubertal, juvenile (other names)

Common features:
- Primary
  - except for Periodontitis, they are healthy
  - rapid attachment loss and bone destruction
  - familial aggregation

Common features:
- Secondary
  - elevated proportions of Aa and in some Pg
  - phagocyte abnormalities
  - hyper-responsive macrophage phenotype, including levels of PGE2 and IL-1B
  - progression of attachment loss and bone loss may be self-arresting

If you are going to diagnose someone as primary aggressive periodontitis they must have the three listed bullet points

You can only surmise, you can confirm this
Based on clinical progression of the disease and overall response of the patient to the therapy
Young boy: 7.5 yo

Are some signs of inflammation along the permanent teeth

In looking at BW and clinical assessment
Notice level of bone on primary molar and canine: evidence of bone loss and furcation involvement on the primary molars as well as clinical probings of 4-5mm with lack of clinical inflammation

What do we need to do?
1. Rule out systemic disease
2. Evaluate family
3. Chronic perio you don't necessary use antibiotics, but aggressive we do
When determining a periodontal diagnosis, can break down into 2 pathways:

 localized vs. generalized

 If there is a generalized presentation in a pre-pubescent individual, first thing in differential is a systemic disease because obviously unlikely losing bone and teeth at such a young age.

 So want to rule out systemic.

 Chroic does not always have to be in an adult.

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**Periodontitis as A Manifestation of Systemic Disease**

**2. GENERALIZED:** associated with systemic disease at young age

- Leukocyte adhesion deficiency (LAD)
- Papillon Lefevere
- Cherubism
- Hypophosphatemia
- Leukemia
- Cyclic neutropenia
- Hypoiodotyosis X

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**Current Classification**

- Chronic Periodontitis
- Aggressive Periodontitis
- Periodontitis as a Manifestation of Systemic Disease
- Necrotizing Periodontal Disease
- Abscesses of the Periodontium
- Periodontitis Associated with Endodontic Lesions
- Developmental or Acquired Deformities and Conditions

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**Periodontitis noted prior to puberty**

(used to be identified as Prepubertal)

- Management
  - Rule out systemic disease
  - Systemic antibiotic and scaling and root planing
  - Extractions?

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All associated with early tooth loss at such a young age.

She is not sure why this slide was placed here (didn't say anything).

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If such severe bone loss, may consider extractions to prevent population/flora on erupting dentition.
Localised Aggressive Periodontitis

- Circumpubertal onset
- Strong serum antibody response
- Localised first molar / incisor
- Interproximal attachment loss on at least two permanent teeth
  - One has to be a first molar (permanent)
  - But no more than two additional teeth other than JP teeth

Anticipate localised permanent first molar and incisors involved in this disease

Aggressive Periodontitis

- Localised
- Generalised

Localised Aggressive Periodontitis

Illustrates clinically lack of inflammation
Nice scalloped gingiva, some stippling, some rolling in mandibular anterior and erythema but nothing dramatic
Could be simple gingivitis involving supracrestal tissues

Localised Aggressive Periodontitis

Found multiple intrabony defects
Only came in because of pericoronitis then discovered this after taking a PAN

Localised Aggressive Periodontitis

1. General characteristics
   - Circumpubertal onset
   - Permanent first molars/incisors

Usually see intrabony defects and bilateral symmetry
3-5x rate of chronic periodontitis
1. General characteristics
- Circumpubertal onset
- Permanent first molars/incisors
- 70-90% exhibit altered neutrophil chemotaxis
- Bilaterally symmetric involvement
- 3-5x rate of attachment loss as chronic periodontitis (4-5μ)

Localized Aggressive Periodontitis
- Vertical bony defects
- Affects females more than males
- Affects African-Americans more than Caucasians
- Familial tendency
- Flora (Aa)

Almost 80% bone loss on maxillary and mandibular arch

Same pt BW and clinical view
Clinical view does not match the BW
No significant signs of clinical inflammation
No radiographic evidence of deposits
Pt characterized with symmetric intrabony defects on mesial of first molars
This was her brother (10.5 yo)

1. Intrabony defect

LEFT:

2. Intrabony defect

RIGHT:

9.5 yo sister

Lots of signs of inflammation (erythema, edema, bulbous papilla)

Her BWs: definite alteration to her bone height

Bone loss on the primary molars

BONE LOSS ON PRIMARY MOLARS

9 year old sisling:

ABOVE:

• No root resorption due to bone loss (no odontoclasts to resorb)
• Losing primary molar early

BELOW:

Rolled margins, but had intrabony defects on those teeth

30 year old man:

MANAGEMENT

Finding which antibiotic would be effective to control the pathogens

Rational behind culture and sensitivity tests:

Disrupt biofilm and deliver antibiotic at same time

Often move into surgical intervention

Localized Aggressive Periodontitis

Generalized Aggressive Periodontitis

16. Usually under 30
16. Poor serum antibody response
16. Episodic nature of the destruction of attachment and alveolar bone
16. Generalized interproximal destruction
   • at least three teeth other than JP teeth
Can see overall the amount of destruction of those maxillary incisors
Overall there is a generalized loss of bone height throughout the dentition
Intrabony defects on the molars
Bilaterally symmetric
Max and mandibular incisors almost floating
26-27 yo patient

1. CHARACTERISTICS
   - Age of onset
   - Is there a clinical pattern?
   - Extent of involvement
   - Flora
   - Immune Response?

1. MANAGEMENT

Management is similar as previously described
1. Antibiotic
2. Culture sensitivity
3. Look at family members

Already lost posterior teeth and soon to be losing incisors
Came in to get teeth cleaned for a wedding
Learned she was going to lose those teeth

Current Classification

- Chronic Periodontitis
- Aggressive Periodontitis
- Periodontitis as a Manifestation of Systemic Disease

Why is therapy different?

- Host response
- Microbial Flora

Neutrophil Chemotaxis

- Neutrophils obtained from heparinized venous blood
- Chemotaxis performed in microchemotaxis chambers using $4 \times 10^6$ cells/mL
- N-formyl-methionyl-leucyl-phenylalanine (20nM) used as the chemoattractant

Usually neutrophils are first response
Goal to look for antibody titers to pathogens and to evaluate neutrophil response
Spun venous blood down and then assessed chemotaxis of neutrophils using microchemotaxis chamber
Wells beneath mimics bacterial peptides (NFMLP)
Needs to be done on individuals with normal neutrophil numbers
In the wells is NFMLP
You plate the neutrophils on top
Look at the migration compared to the control
When see pseudopodal extensions, see extension
Reduced migration to the bacterial peptide compared to controls

Additionally also looked for antibody titers for putative pathogens
Looked for markers of Aa, Pg, ...

Supragingival flora

Expect to see Aa and Pg in this group
Why Aa is so important
Leukotoxin neutralizes effect of neutrophil
Tissue invasive (doesn’t just stay in biofilm)—invades pocket wall and can evade the immune response

When look at presentation in the families overall, family members also have high antibody titers to Aa
Reduced neutrophil chemotaxis
Risk among family members is high and identifiable before they may present with the disease

Proband with arrow
Yellow = decreased neutrophil chemotaxis
Two sibblings have disease
One has altered neutrophil chemotaxis

3 siblings have disease
All have high antibody titers and altered neutrophil chemotaxis
One sibling may be at risk
Mom does not have the disease

More often than not require surgical intervention
Treatment of Aggressive PD

Premise:
Early recognition and treatment is key to more predictable outcome
Do not respond as well to conventional therapy
Must have an all encompassing approach and be aggressive with therapy not conservative

Non-surgical Therapy
Subgingival scaling and root planing
Systemic antibiotic therapy
Local antimicrobial therapy

May use mouth rinses

Signs of inflammation
Can see that there would be disease
In starting treatment:
1. Non surgical tx
2. Expected good result because horizontal pattern and spacing allowed instrumentation
3. Right image is how she appeared after re-evaluation
4. Continued active therapy and instrumentation
5. Able to control disease and pockets just with non-surgical therapy, antibiotic and home care

Generalized Aggressive Periodontitis

All views, no signs of clinical inflammation
FMS illustrates destruction so significant almost to apex of teeth and requiring endo therapy to control disease process

Tissue color is not that severe
1. Had to hemisect the tooth to make a premolar out of it because there was no way to treat the distal aspect of the molar
2. Placed a bridge to restore missing tooth structure

Overall:
30 yo female
Intrabony defect
Required grafting to treat that area

As a review...
**Treatment Plan**

- Culture and sensitivity
  - Microbial sensitivity to antibiotics
  - Select based on sensitivity results
- SC/RP with selected systemic antibiotic

**Why antimicrobial therapy?**

- Tissue invasion of putative pathogens
  - P. gingivalis
  - Aa
  - Treponema
- Some strains of Aa produce a highly toxic leukotoxin

**Antibiotic Therapy**

- Tetracycline 250 mg QID for 2 to 3 weeks
- Doxycycline 100mg BID for 20 days
- Combination of amoxicillin/metronidazole

**Treatment Plan**

- Re-evaluation (4-6 weeks post SC/ RP)
  - as in conventional therapy for Chronic Periodontitis

**Surgical Therapy**

Flap debridement therapy

- Resective
- Regenerative procedures -
  - guided tissue regeneration
  - freeze-dried bone allografts
  - autogenous grafts
  - alloplasts

Combine with systemic antibiotic
Maintenance

Overall, treatment is all encompassing and maintenance needs to be comprehensive
- Antimicrobial (topical and local delivery)
- Host modulation
- Frequency
- May need to extract severely involved and consider implant placement earlier than would in a Chronic Periodontitis patient

Study Questions

1. What three characteristics must be present to identify a patient with Aggressive Periodontitis.
2. Can you test for the systemic/host response alterations of the Aggressive Periodontitis patient?
3. Why is Aggressive periodontitis treated with Antibiotics as part of the non-surgical therapy?
4. What makes therapy of Aggressive Periodontitis different than that of Chronic Periodontitis?