AGGRESSIVE PERIODONTITIS

Dr. Chander Shekhar Joshi
Reader
Dept of Periodontology
Subharti Dental College & Hospital
Swami Vivekanand Subharti University
Meerut, UP
CLASSIFICATION OF PERIODONTITIS

- AAP WORLD WORKSHOP, 1989
  - ADULT PERIODONTITIS
  - EARLY ONSET PERIODONTITIS
    - PREPUBERTAL PERIODONTITIS
  - JUVENILE PERIODONTITIS
  - RAPIDLY PROGRESSIVE PERIODONTITIS
  - NECROTIZING PERIODONTITIS
  - PERIODONTITIS ASSOCIATED WITH SYSTEMIC DISEASE
  - REFRACTORY PERIODONTITIS
AAP INTERNATIONAL WORKSHOP, 1999

- CHRONIC PERIODONTITIS
- AGGRESSIVE PERIODONTITIS
- PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASE
DEFINITIONS

- **PERIODONTITIS**: An inflammatory disease of the supporting tissues of the teeth, caused by specific microbes / resulting in progressive destruction of Pdl & alv bone / with pocket formation, recession or both.

- **AGGRESSIVE PERIODONTITIS**: Comprises a group of rare, often severe, rapidly progressive forms of periodontitis / often characterized by an early age of manifestation / and a distinctive tendency to aggregate in families.
CRITERIA OF IDENTIFICATION
(Lang et al, 1999)

- PRIMARY FEATURES
  - Non-contributory medical history
  - Rapid attachment loss & bone destruction
  - Familial aggregation of cases
SECONDARY FEATURES

- Amounts of microbial deposits inconsistent with destruction
- Elevated proportions of *A. actinomycetemcomitans* & in some subjects *P. gingivalis*
- Phagocyte abnormalities
- Hyper-responsive macrophage: PGE$_2$ & IL-$1\beta$
- Progression of bone loss may be self-arresting
FORMS OF AGGRESSIVE PERIODONTITIS

- LOCALIZED AGGRESSIVE PERIODONTITIS
- GENERALIZED AGGRESSIVE PERIODONTITIS
LOCALIZED AGGRESSIVE PERIODONTITIS

- Circumpubertal onset

- Localized first molar / incisor presentation: involving no more than 2 other teeth

- Robust serum antibody response to infecting agents
GENERALIZED AGGRESSIVE PERIODONTITIS

- Age group under 30; may be older
- Bone loss on at least 3 other teeth
- Pronounced episodic nature of destruction
- Poor serum antibody response to infecting agents
LOCALIZED AGGRESSIVE PERIODONTITIS

**HISTORICAL BACKGROUND**

- **1923**: Gottleib reported first case; *diffuse atrophy of alveolar bone*
- **1947**: Goldman considered it degenerative, non-inflammatory – *Periodontosis*
- **1967**: Chaput et al introduced the term *Juvenile Periodontitis*
- **1989 AAP, 1993 European Workshop**: *Early Onset Periodontitis*
- **1999 AAP**: *Aggressive Periodontitis*
JUVENILE PERIODONTITIS

BAER (1971): A disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition. The amount of destruction manifested is not commensurate with the amount of local irritants.
REASONS FOR LOCALIZATION OF BONE LOSS

1. **I^ST** molars and incisors are first permanent teeth to erupt. A.a. colonize these sites / host immune defenses are stimulated

2. Colonization by bacteria antagonistic to A.a.

3. A.a. may lose its leukotoxin producing ability

4. Defect in cementum formation
CLINICAL FEATURES

- Lack of clinical inflammation despite the presence of deep periodontal pockets.

- Minimal plaque, which is inconsistent with the amount of periodontal destruction (contains elevated levels of A.a, and P. gingivalis).

- Rapid rate of progression: 3-4 times faster than in chronic Periodontitis.

- Distolabial migration of the maxillary incisors, increasing mobility of the first molars, sensitivity, & deep, dull, radiating pain
RADIOGRAPHIC FEATURES

- Vertical loss of alveolar bone around the first molars in otherwise healthy teenagers.

- *Arc-shaped loss* of alveolar bone extending from the distal surface of the second premolar to the mesial surface of second molar.
Low prevalence of about 0.2%.

Affects both males and females / most frequently in period between puberty and 20 years of age.

Blacks are at higher risk than whites with black males more than black females.
GINGIVAL TISSUE RESPONSE

- SEVERE, ACUTELY INFLAMED TISSUE, OFTEN PROLIFERATING, ULCERATED AND FIERY RED
  - Bleeding Spontaneously Or On Slight Stimulation
  - Suppuration May Be Present

- GINGIVAL TISSUES MAY APPEAR PINK, FREE OF INFLAMMATION / SOME DEGREES OF STIPPLING
  - Deep Pockets On Probing
GENERALIZED AGGRESSIVE PERIODONTITIS

- Age group under 30; may be older
- Bone loss on at least 3 other teeth
- Pronounced episodic nature of destruction
- Poor serum antibody response to infecting agents
RADIOGRAPHIC FEATURES

- Generalized extensive bone loss

PROGNOSIS

- Poor as compared to localized aggressive
RISK FACTORS

- MICROBIOLOGIC FACTORS
- IMMUNOLOGIC FACTORS
- GENETIC FACTORS
- ENVIRONMENTAL FACTORS
Several microorganisms have been shown to have tissue invasive properties

A. actinomycetemcomitans, P gingivalis, Capnocytophaga sp., Prevotella intermedia & Campylobacter rectus
EVIDENCE OF ETIOLOGIC AGENT

- Aa is found in high frequency in lesions characteristic of LAP (approx. 90%)

- Significantly elevated serum antibody titers to Aa in many patients

- Reduction in subgingival load of Aa during treatment

- Produces a no. of virulence factors that may contribute to the disease process
IMMUNOLOGIC FACTORS

- **Functional defects of PMNs & monocytes**: impaired chemotaxis & phagocytosis

- **Hyper responsiveness of monocytes**: PGE$_2$ & IL-1$\beta$.

- **Autoimmunity**: antibodies to host collagen, DNA & IgG
GENETIC FACTORS

- Specific genes have not been identified

- Some immunological defects may be inherited

- E.g. antibody response to Aa is under genetic control

- However it is unlikely that all the patients affected have the same genetic defect
ENVIRONMENTAL FACTORS

- **SMOKING**: Identified as a major risk factor
- Frequency & duration of habit
- More attachment loss in smokers than non-smokers.
TREATMENT

- Scaling & root planing alone is not sufficient to remove the tissue invasive microorganisms

- Antibiotic therapy has to be combined
CURRENT APPROACH TO THERAPY

- SRP with antibiotics for least 1 week
- Surgery in cases of extensive bone loss
- Clinical & microbial monitoring every 3 weeks when in active state.
ANTIOBIOTIC REGIMENS

- TETRACYCLINE : 250 mg QID
- DOXYCYCLINE : 100 mg OD
- AMOXICILLIN & METRONIDAZOLE