AGGRESSIVE PERIODONTITIS

Periodontology I - 4th year

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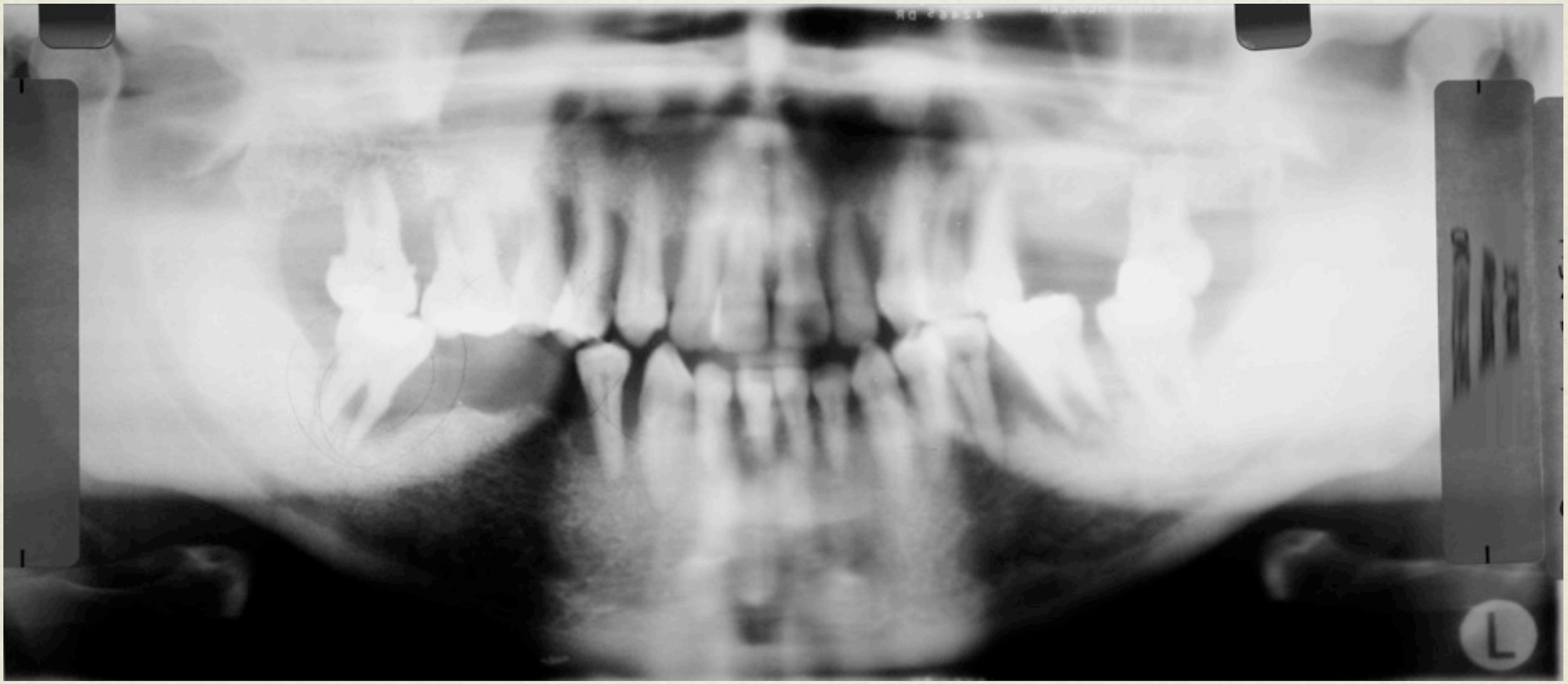
OUTLINE

- Localized Aggressive Periodontitis
    - Clinical & radiographic presentation
    - Prevalence and distribution among age and gender
- Generalized Aggressive Periodontitis
    - Clinical & radiographic presentation
    - Prevalence and distribution among age and gender
- Risk Factors for Aggressive Periodontitis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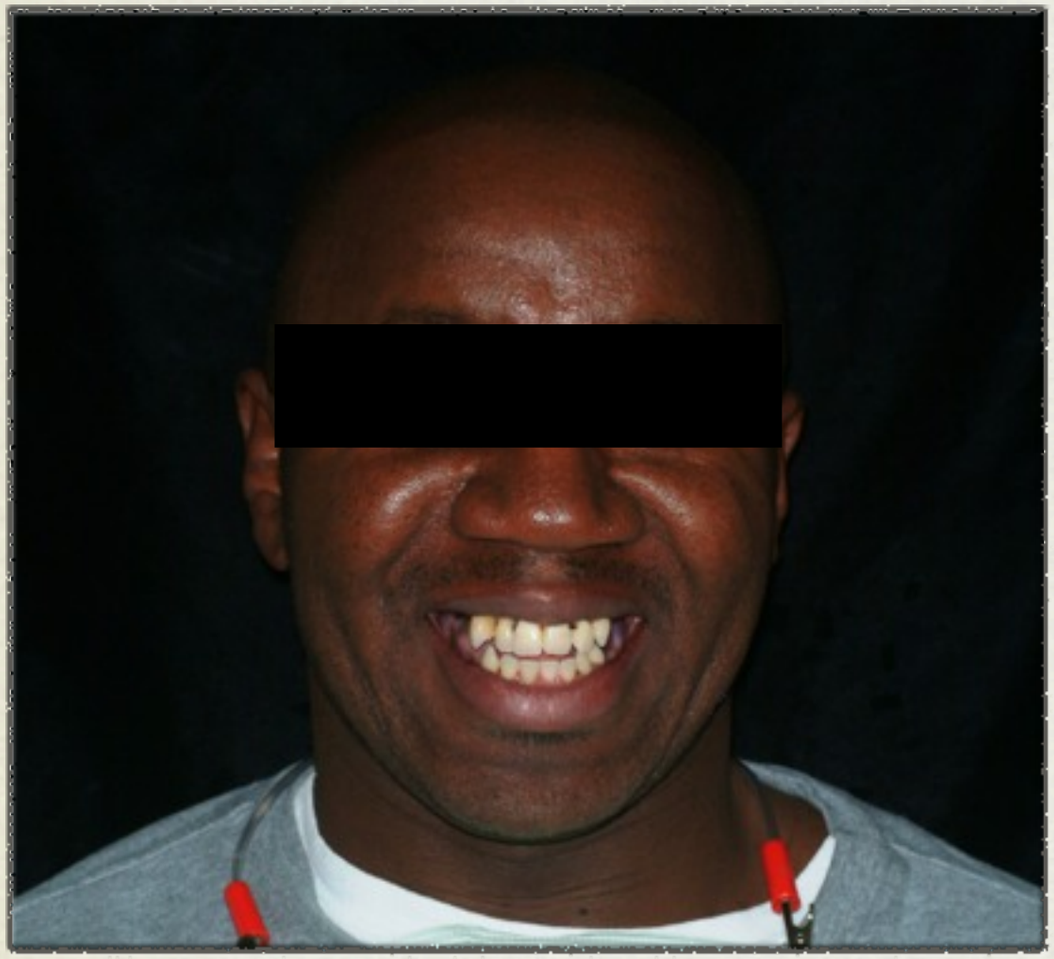


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AGGRESSIVE PERIODONTITIS

- Aggressive Periodontitis: inflammatory disease of the periodontium causing rapid bone loss and periodontal destruction around one or more permanent teeth in an otherwise healthy adolescent

    - First reports in 1923 (Gottlieb)

AGGRESSIVE PERIODONTITIS

- Re-classified in “The Workshop for the Classification of Periodontal Diseases” in 1999:

Localized Juvenile Periodontitis (LJP)

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Localized Aggressive Periodontitis (1999)

Generalized Juvenile Periodontitis

Rapidly Progressive Periodontitis

↘ ↙

Generalized Aggressive Periodontitis (1999)

AGGRESSIVE PERIODONTITIS

- Primary features:

1. Otherwise clinically healthy individual
2. Rapid attachment loss and bone destruction
3. Familial aggregation

AGGRESSIVE PERIODONTITIS

- Secondary features:

1. Amount of microbial deposits is inconsistent with amount of destruction

2. Elevated proportions of *Aa*, some populations *Pg*

3. Phagocyte abnormalities

4. Hyper-responsive macrophage phenotype including elevated levels of PGE-2 and IL-1b

5. Progression of attachment loss and bone loss may be self-arresting

AGGRESSIVE PERIODONTITIS

- Not all features have to be present to establish a diagnosis
- Combination of clinical, radiographic and historical findings (sometimes laboratory findings) will aid in establishing a diagnosis.

LOCALIZED AGGRESSIVE PERIODONTITIS

- Clinical presentation:
    - Onset around puberty (circumpubertal)
    - Robust antibody response to infecting agents
    - Localized molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors

LOCALIZED AGGRESSIVE PERIODONTITIS

- Reason for localization to 1st molar/incisors is not understood, theories are:

1. The development of adequate immune defenses to bacteria such as Aa after the initial colonization of the teeth to erupt first (1st molars and incisors) which prevents colonization of other sites.

2. Bacteria antagonistic to Aa may colonize the periodontal tissues and inhibit Aa from further colonization of periodontal sites in the mouth.

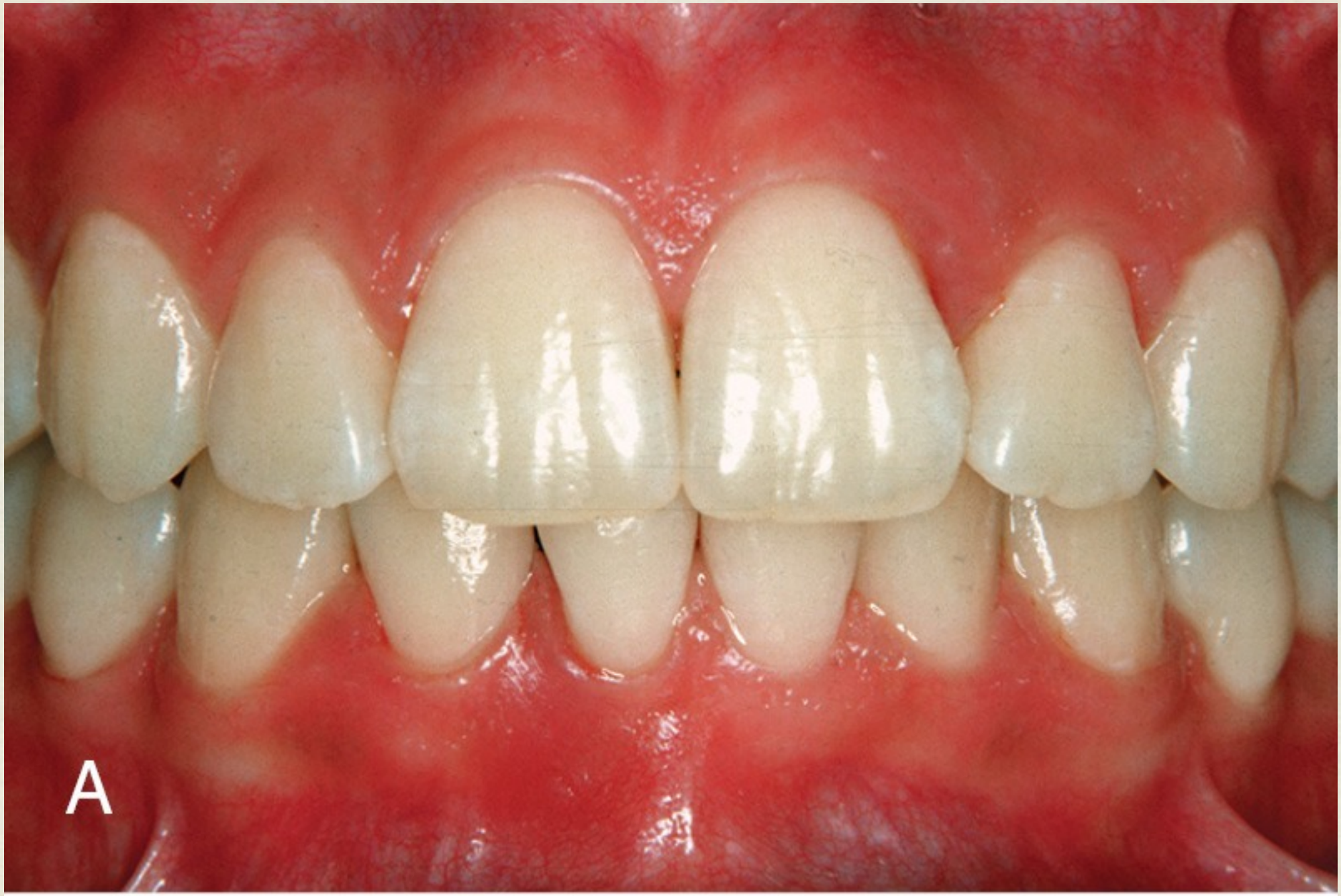
LOCALIZED AGGRESSIVE PERIODONTITIS

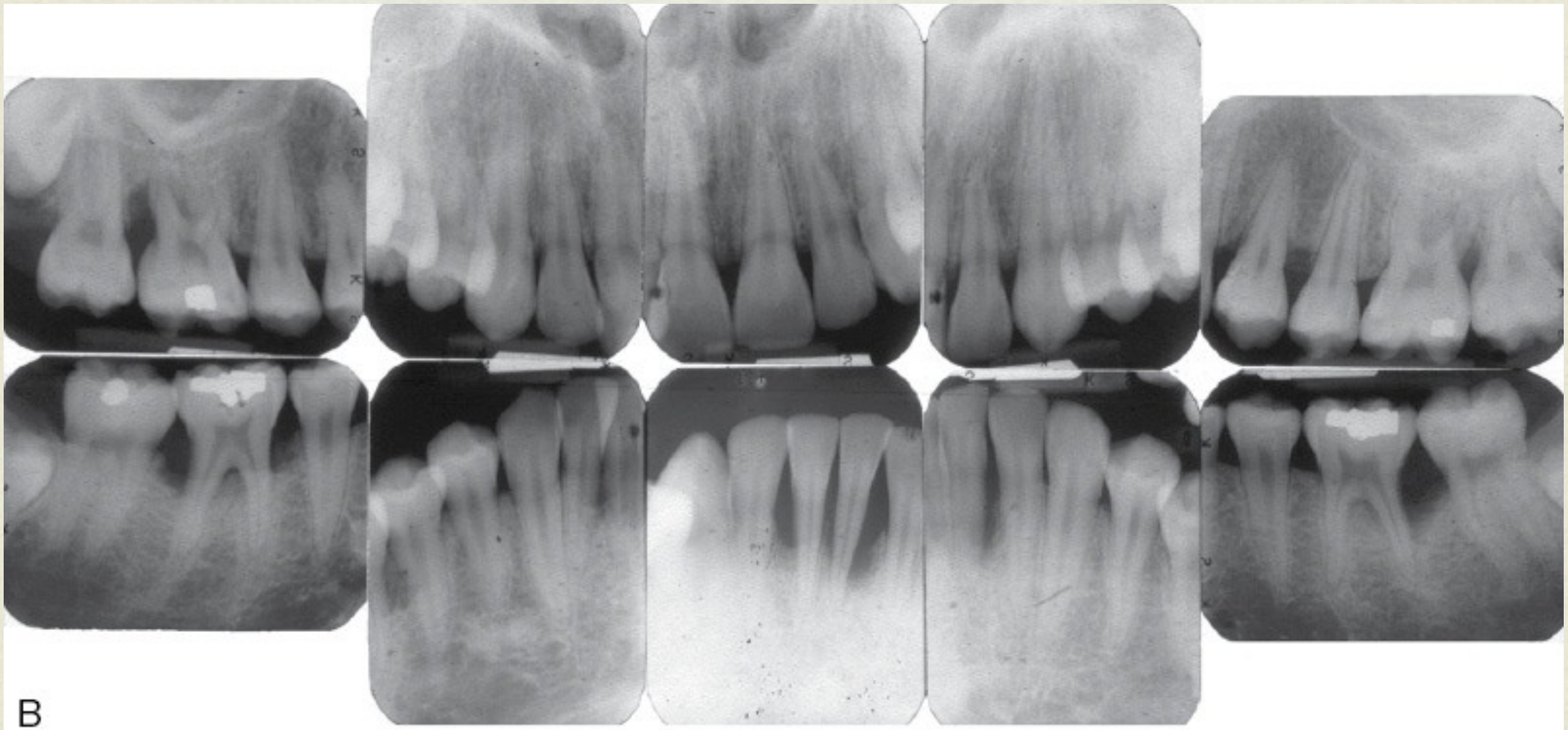
3. Aa may lose its leukotoxin-producing ability for unknown reasons which could impair or arrest the progression of the disease and avert the colonization of new periodontal sites.

4. A defect in cementum formation may be responsible for the localization of the lesions.

LOCALIZED AGGRESSIVE PERIODONTITIS

- Lack or minimal clinical inflammation
- Minimal plaque, rarely mineralizes to calculus
- Elevated levels of *Aa*
- Rapid progression (3-4 times the rate of progression in chronic periodontitis)





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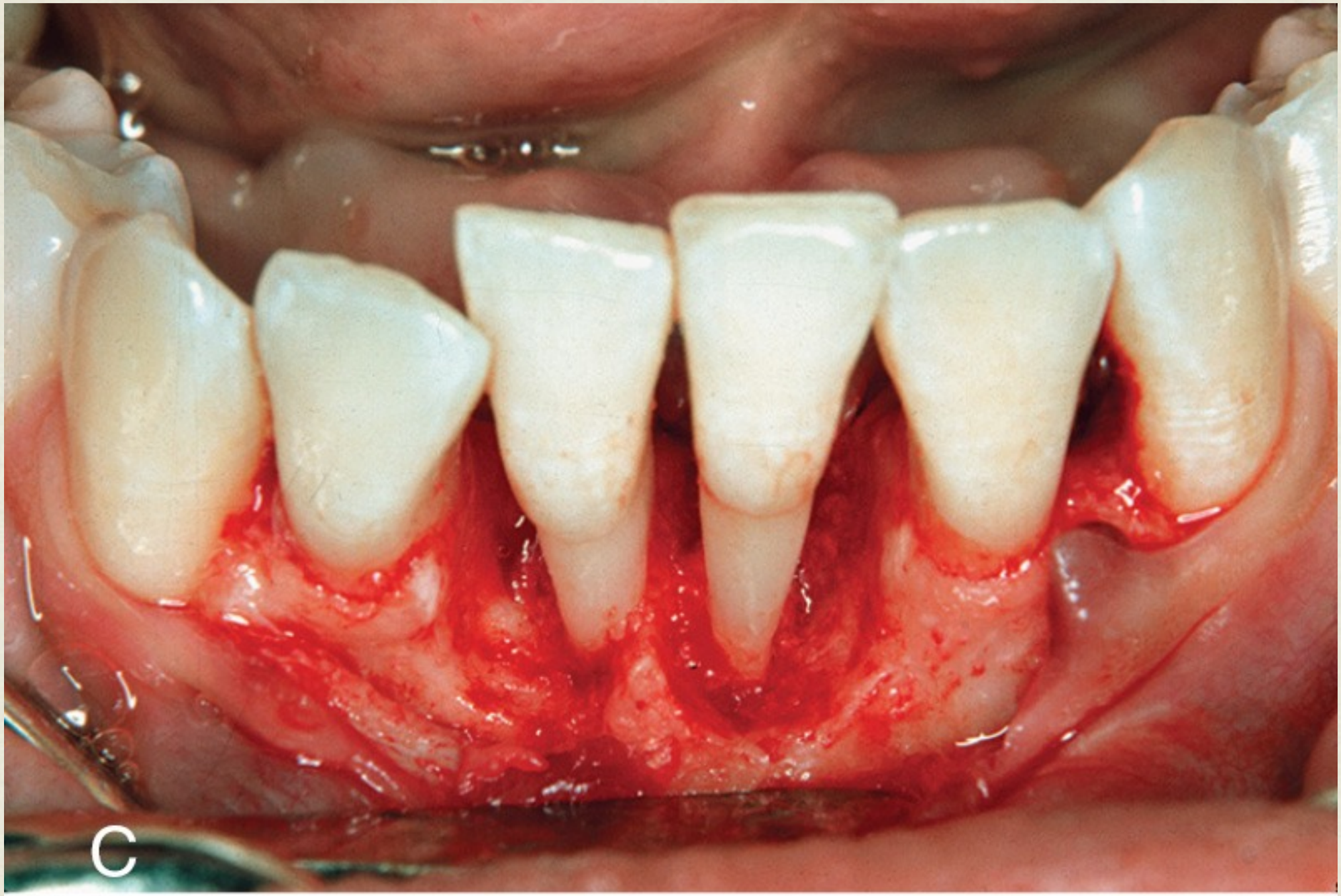
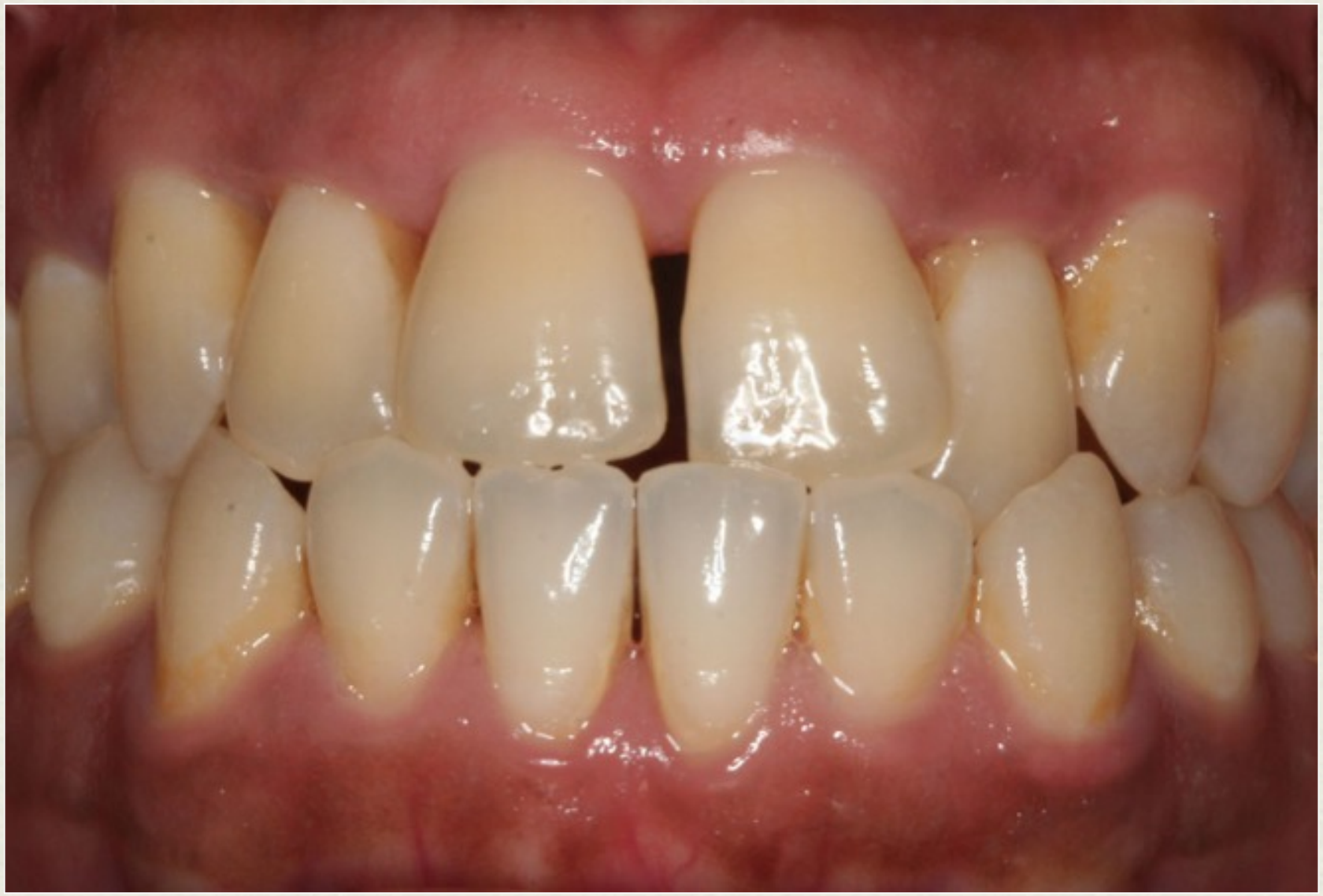


LOCALIZED AGGRESSIVE PERIODONTITIS

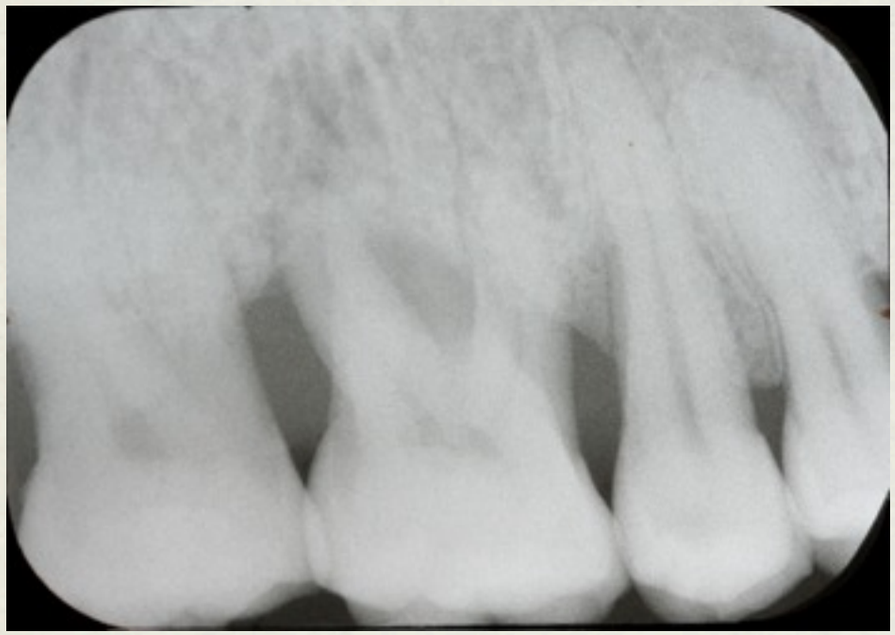
- Other features:

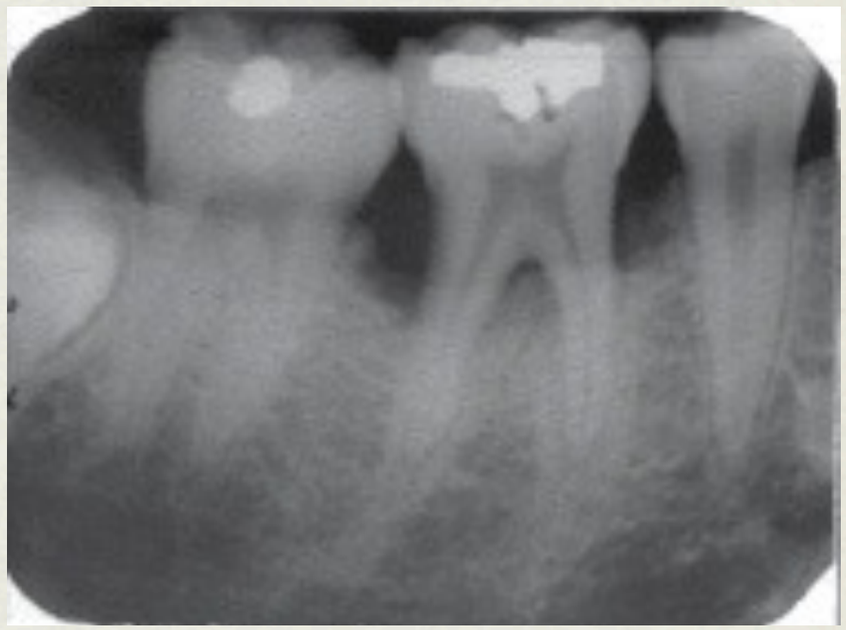
1. Distolabial migration of the maxillary incisors with concomitant diastema formation
2. Increasing mobility of the maxillary and mandibular incisors and first molars
3. Sensitivity of denuded root surfaces to thermal and tactile stimuli
4. Deep, dull, radiating pain during mastication, probably caused by irritation of the supporting structures by mobile teeth and impacted food
5. Periodontal abscesses may form at this stage, and regional lymph node enlargement may occur



LOCALIZED AGGRESSIVE PERIODONTITIS

- Radiographic findings:
  - Vertical loss of alveolar bone around the first molars and incisors.
  - “Arc-shaped” defects





LOCALIZED AGGRESSIVE PERIODONTITIS

- Prevalence distribution by age and gender
    - <1% in a diverse population
        - <0.2% in whites
        - ≈ 2% in blacks
    - Historically was thought to be more prevalent in females but studies correcting for ascertainment bias show no sex predilection
    - Black males ⇒ black females ⇒ white females ⇒ white males

LOCALIZED AGGRESSIVE PERIODONTITIS

- Management:
  - Non-surgical and surgical periodontal therapy
  - Oral hygiene instructions
  - Systemic antibiotics

GENERALIZED AGGRESSIVE PERIODONTITIS

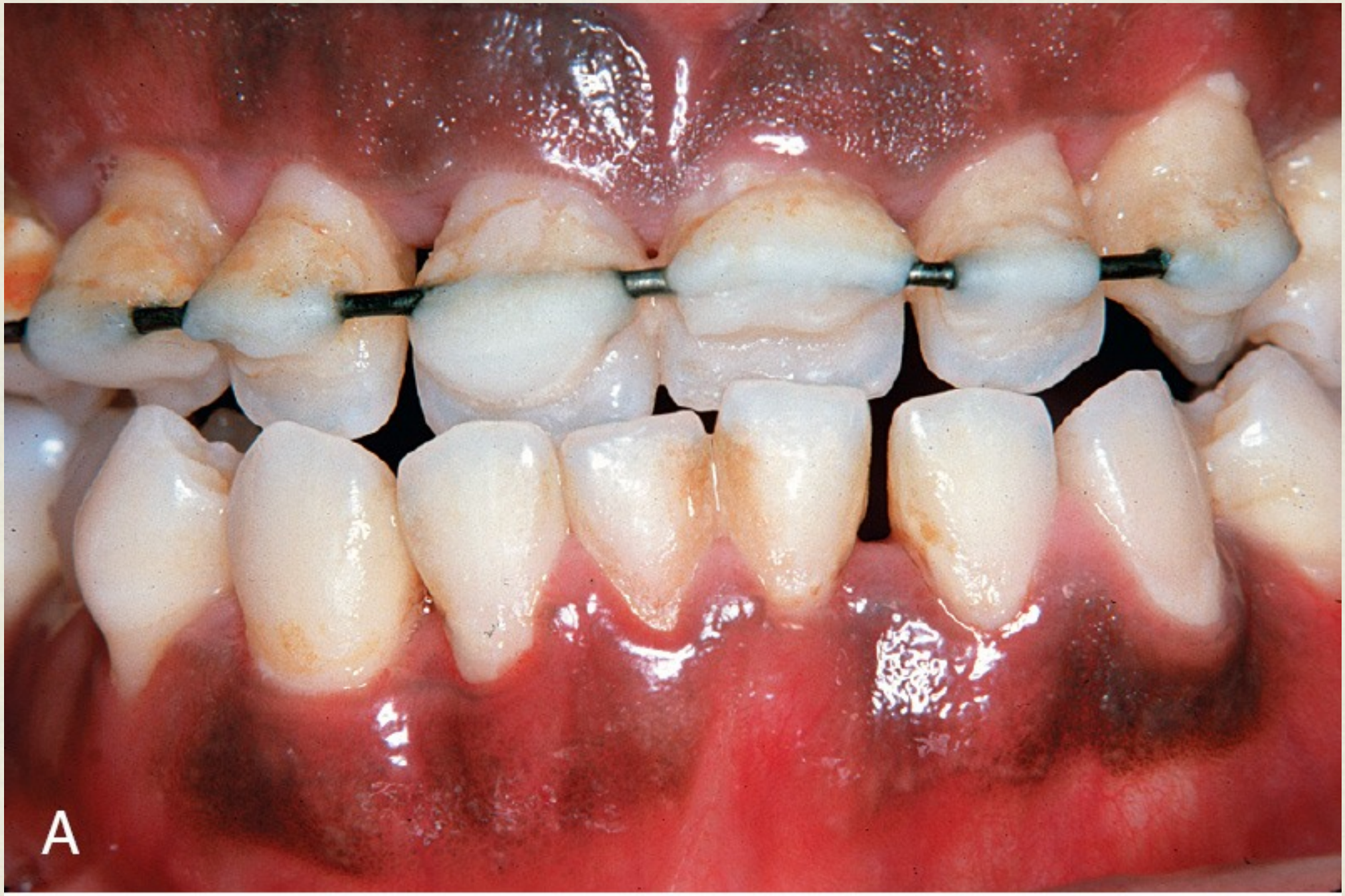
- Clinical presentation
    - Affects individuals under age 30
    - Poor antibody response to the infecting agents
    - Minimal plaque, elevated *Pg, Tf and Aa*
    - Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors

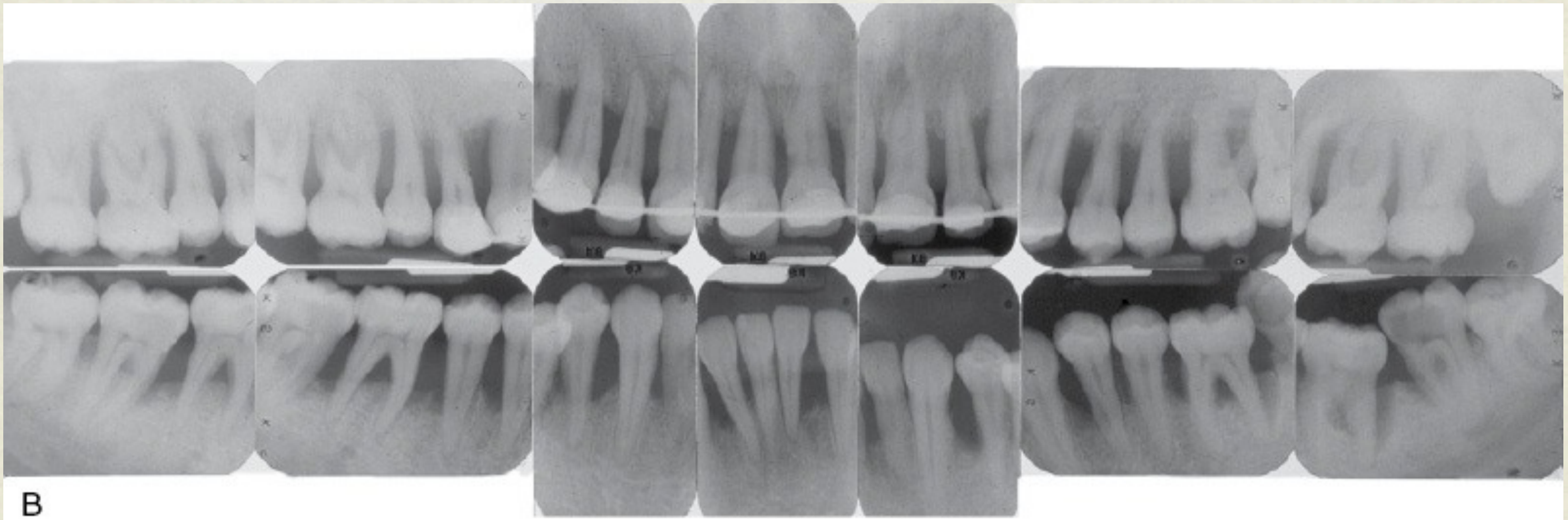
GENERALIZED AGGRESSIVE PERIODONTITIS

- Clinical presentation
    - Two types of gingival response:
        1. Severe, acutely inflamed tissue, often proliferating, ulcerated, and fiery red and bleeding may occur spontaneously or with slight stimulation, suppuration may be an important feature.
        2. Tissues may appear pink, free of inflammation, and occasionally with some degree of stippling, although stippling may be absent.

GENERALIZED AGGRESSIVE PERIODONTITIS

- GAP may be arrested spontaneously or after therapy while other cases continue to progress and eventual tooth loss despite intervention.
- New cases should receive medical checkup to rule out systemic diseases.





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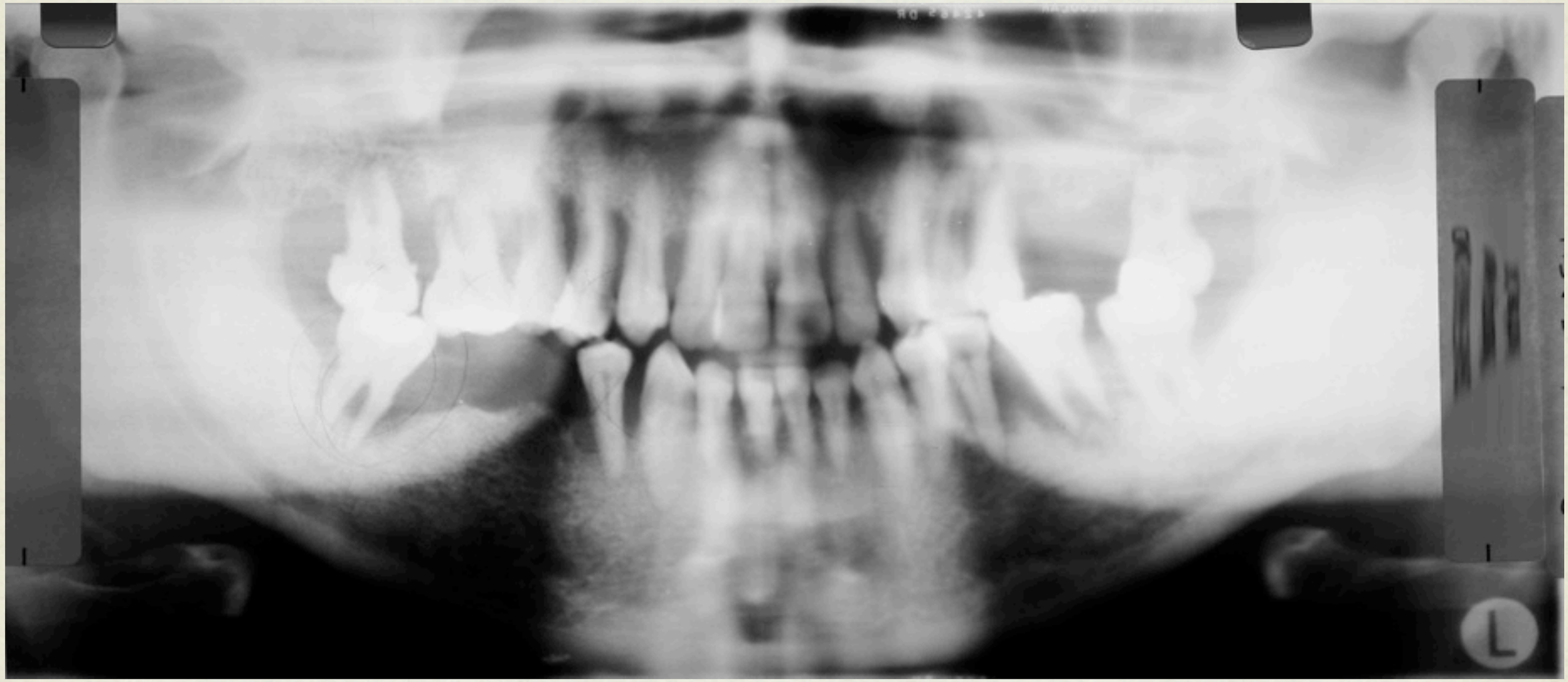
GENERALIZED AGGRESSIVE PERIODONTITIS

- Radiographic presentation:
  - Bone loss of varying severity.
  - Longitudinal comparison of x-rays in some cases shows extreme rate of bone loss.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GENERALIZED AGGRESSIVE PERIODONTITIS

- Prevalence and distribution by age and gender:
    - 8% of Sri-lankan tea laborers had progression of attachment loss of about annual 0.1 - 1.0 mm
- 0.13% of adolescents 14-17 yrs of age had GAP
- Higher risk in blacks than whites and in males than females

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

- Microbiologic factors:
  - *Aa* has been implicated as the primary pathogen associated with LAP:

1. *Aa* is found in high frequency (approximately 90%) in lesions characteristic of LAP.
2. Sites with evidence of disease progression often show elevated levels of *Aa*.
3. Many patients with the clinical manifestations of LAP have significantly elevated serum antibody titers to *Aa*.
4. Clinical studies show a correlation between reduction in *Aa* during treatment and a successful clinical response.
5. *Aa* produces a number of virulence factors that may contribute to the disease process.

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

- Microbiologic factors:
  - In some reports, Aa was not detected and in other reports, Aa was detected in healthy sites.
  - Serotype b has been shown to be the virulent type of Aa in US studies and elsewhere.
  - Aa has been shown to have the ability to invade periodontal tissues.

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

- Immunologic factors:
- Aggressive periodontitis patients display functional defects of polymorphonuclear leukocytes (PMNs), monocytes, or both. These defects could:
    - Impair the chemotactic attraction of PMNs to the site of infection
    - Impair the ability to phagocytose and kill microorganisms.
- Hyper-responsiveness of monocytes, HLA markers, autoimmunity.

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

- Genetic factors:
- Autosomal dominant inheritance in LAP (studies done in african-americans)
- Immunologic PMN defects cluster in LAP families which indicated they might be related to gene clusters and inherited
- Different patterns of inheritance might exist in other populations.

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

- Environmental factors:
- Smoking in GAP patients is associated with more CAL and more teeth being affected
- Smoking has less impact on attachment levels in young patients with LAP.

THE END

REFERENCES

- Carranza's Clinical periodontology, 11th edition, chapter 18