Gingival Problems

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Contents

- Chronic gingivitis
- Gingival enlargement
- Gingival recession
Objectives

- To know the differential diagnosis of the various forms of gingivitis
- To describe etiology and treatment options for the various forms of gingival enlargement
- To describe appropriate therapy of gingivitis and gingival enlargement
- To explain etiology, clinical relevance and treatment for gingival recession
Healthy Periodontium

- 0 - 3 mm sulcus depth
- No suppuration
- No bleeding
Chronic Gingivitis
Clinical and histopathological feature of Chronic gingivitis

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Healthy</th>
<th>Gingivitis</th>
<th>Histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid flow</td>
<td>Minimal</td>
<td>Increased</td>
<td>Increased vascular permeability</td>
</tr>
<tr>
<td>Bleeding on probing</td>
<td>No</td>
<td>Yes</td>
<td>Vascularity increased, Dilatation and engorgement of the capillaries, Epithelium degenerated and thinned with capillaries closer to surface</td>
</tr>
<tr>
<td>Color</td>
<td>Pink</td>
<td>Red to bluish red (erythema)</td>
<td>Result of: Vascular proliferation, Reduction of keratinization, Venous statis cause bluish red</td>
</tr>
<tr>
<td>Consistency</td>
<td>Firm, resilient</td>
<td>Soggy, puffy, pits on pressure (edema), flaccid</td>
<td>Infiltration by fluid and cells into the inflamed region, Flaccidity result of loss gingival fibers</td>
</tr>
</tbody>
</table>
# Clinical and histopathological feature of Chronic gingivitis

<table>
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<th>Clinical feature</th>
<th>Healthy</th>
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<th>Histopathology</th>
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<tbody>
<tr>
<td><strong>Texture</strong></td>
<td>Stippling (not always)</td>
<td>Loss of stippling, smooth and shiny</td>
<td>Thinning or atrophy of the epithelium, Degeneration associated with edema and leucocytic infiltration into the connective tissue</td>
</tr>
<tr>
<td><strong>Size</strong></td>
<td>Knife-edge margins, Papilla fills intedental space, Sulcus depth ≤ 3 mm</td>
<td>Swollen or balloning of interdental papilla and/or gingival margin, False pocket formation</td>
<td>Result of: Edema, New capillary formation, Vascular engorgement</td>
</tr>
<tr>
<td><strong>Shape/Contour</strong></td>
<td>Scalloped-troughs in marginal areas rise to peaks in interdental areas</td>
<td>Blunts the marginal and papillary tissues</td>
<td>Edema, New capillary formation, Vascular engorgement</td>
</tr>
</tbody>
</table>

*Remember B C C T S S*
STIPPLING

GINGIVAL MARGIN NOT STIPPLED
Distribution of gingivitis

- Localized \( \leq 30\% \)
- Generalized \( \geq 30\% \)
- Marginal
- Papillary
- Diffuse
Classification of Periodontal Diseases

I. Diseases of the Gingival Unit: Gingivitis
   1- Plaque-induced gingivitis.
   2- Non-plaque-induced gingivitis

II. Diseases of the Supporting Structures: Periodontitis
I. Gingival Diseases

Dental plaque-induced gingival disease
- associated with dental plaque
- modified by systemic factors
- modified by medications
- modified by nutrition
Characteristics of Plaque-Induced Gingivitis

- Plaque present at gingival margin
- Disease begins at the gingival margin
- Change in gingival color
- Change in gingival shape/contour
- Sulcular temperature change
Characteristics of Plaque-Induced Gingivitis

- Increased gingival exudates
- Bleeding upon provocation
- Absence of attachment loss
- Absence of bone loss
- Histological changes
  - Reversible with plaque removal
- Reversible with plaque removal
Characteristics of Plaque-Induced Gingivitis

- Microbial plaque is the DIRECT cause of gingivitis.

- Loe et al, 1965 study: (classic evidence)
  - 12 individuals Abstained from all oral hygiene measures.
  - All individuals developed gingivitis within 10-21 days.
  - All individuals returned to healthy gingiva within one week of resuming oral hygiene measures
Epidemiology gingivitis

Is ubiquitous in population of children and adults globally

>82% of adolescent in US have overt gingivitis and signs of gingival bleeding (Albandar et al., 1996)

Similar or higher prevalence in other parts of the world

>75% of adults in US have gingival bleeding and dental calculus (Kingman and Albandar, 2002)
## Epidemiology CIPD: adults

(MOH Malaysia, 1990, 2000)

<table>
<thead>
<tr>
<th>Periodontal Status</th>
<th>1990(%)</th>
<th>2000(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects examined having periodontal diseases</td>
<td>92.8</td>
<td>90.2</td>
</tr>
<tr>
<td>Gingivitis (CPITN 1)</td>
<td>4.6</td>
<td>4.2</td>
</tr>
<tr>
<td>Calculus (CPITN 2)</td>
<td>65.1</td>
<td>56.9</td>
</tr>
<tr>
<td>Moderate Pocketing (CPITN 3)</td>
<td>17</td>
<td>20.8</td>
</tr>
<tr>
<td>Deep Pocketing (CPITN 4)</td>
<td>6.0</td>
<td>5.5</td>
</tr>
</tbody>
</table>
I. Gingival Diseases

Dental plaque-induced gingival disease
- associated with dental plaque
- modified by systemic factors
  - associated with endocrine system
    - Puberty-associated gingivitis
    - Pregnancy-associated:
      - gingivitis
      - pyogenic granuloma
    - Diabetes mellitus-associated gingivitis
  - associated with blood dyscrasia
    - leukemia-associated gingivitis
- modified by medications
- modified by nutrition
Puberty-associated gingivitis

- Clinical signs of gingivitis intensified.
- ↑ serum level of testosterone (boys) or estradiol (girls) results in:
  - ↑ level of Prevotella intermedia and P. nigrescens
Pregnancy-Associated Gingivitis

- ↑ prevalence of gingivitis.
- Prevalence range 30-100%
Gingivitis Caused by Hormonal Changes

Puberty

Pregnancy

REF: Color Atlas of Periodontology
Characteristics of Pregnancy-Associated Pyogenic Granuloma

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Can occur anytime during pregnancy
- More common in maxilla
- More common interproximally
- Sessile or pedunculated protuberant mass
- Not a neoplasm; has histologic appearance of a pyogenic granuloma
- Regresses following parturition
Characteristics of Leukemia-Associated Gingivitis

- Pronounced inflammatory response of gingiva in relation to the plaque present; however, plaque is not a prerequisite for oral lesions
- Gingival lesions are primarily found in acute leukemias
- Change in gingival color
- Change in gingival contour with possible modification of gingival size
- Enlargement first observed at the interdental papilla
- Bleeding upon provocation (may be one of the initial oral signs)
- Reductions in dental plaque can limit the severity of lesion

I. Gingival Diseases
   A. Dental plaque-induced gingival diseases*
      1. Gingivitis associated with dental plaque only
         a. without other local contributing factors
         b. with local contributing factors (See VIII A)
      2. Gingival diseases modified by systemic factors
         a. associated with the endocrine system
            1) puberty-associated gingivitis
            2) menstrual cycle-associated gingivitis
            3) pregnancy-associated
               a) gingivitis
               b) pyogenic granuloma
            4) diabetes mellitus-associated gingivitis
         b. associated with blood dyscrasias
            1) leukemia-associated gingivitis
            2) other
      3. Gingival diseases modified by medications
         a. drug-influenced gingival diseases
            1) drug-influenced gingival enlargements
            2) drug-influenced gingivitis
               a) oral contraceptive-associated gingivitis
               b) other
      4. Gingival diseases modified by malnutrition
         a. acerotic acid-deficiency gingivitis
         b. other*
Characteristics of Diabetes Mellitus-Associated Gingivitis

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Change in gingival color
- Change in gingival contour
- Increased gingival exudate
- Bleeding upon provocation
- Most commonly associated in children with poorly controlled Type-1 diabetes mellitus
- Absence of bone loss
- Absence of attachment loss
- Reversible with control of diabetic state
- Reduction of dental plaque can limit severity of lesion
(A) Gingival Diseases

modified by medications

- drug-influenced gingival enlargement
- drug-influenced gingivitis:
  - oral contraceptive-associated gingivitis
Gingival Disease Modified by Medications

DRUG-INDUCED ENLARGEMENTS

- dilantin, nifedipine, verpamil, cyclosporin
- free gingiva increases in size and thickness
- decrease in collagen turnover, increase interstitial ground substance
Characteristics of Drug-Influenced Gingival Enlargement

- Variation in inter patient and intra patient pattern
- Predilection for anterior gingiva
- Higher prevalence in children
- Onset within 3 months
- Change in gingival contour leading to modification of gingival size
- Enlargement first observed at the interdental papilla
- Change in gingival color
Characteristics of Drug-Influenced Gingival Enlargement

- Increased gingival exudate
- Bleeding upon provocation
- Found in gingiva with or without bone loss but is not associated with attachment loss
- Pronounced inflammatory response of gingiva in relation to the plaque present
- Reductions in dental plaque can limit the severity of lesion
- Must be using phenytoin, cyclosporine A, or certain calcium channel blockers
## Gingival enlargement

<table>
<thead>
<tr>
<th>Clinical conditions</th>
<th>Clinical feature</th>
<th>Histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical enlargement (hyperplasia) associated with Dilantin (Phenytoin), Cyclosporine and Calcium Channel Blockers (Nefedipine, Verapamil, Nitredipine, Felodipine, Diltiazem, Oxodipine)</td>
<td>Increased amount of gingival tissue, tissue usually firm</td>
<td>Hyperplasia of connective tissue. Increased response of gingiva to plaque</td>
</tr>
<tr>
<td>Pyogenic grauloma (pregnancy tumor) (gingivitis in pregnancy)</td>
<td>Red, soft gingiva. May be prominent enlargement interproximally</td>
<td>Typical inflammatory response. Increased response of gingiva to plaque</td>
</tr>
</tbody>
</table>
Gingival enlargement in a patient taking phenytoin
Table 1. Partial list of possible diagnoses for gingival diseases (1999 Classification) (10)

**Dental plaque-induced gingival diseases**
- Plaque-induced gingivitis (no other local contributing factors) (60)
- Plaque-induced gingivitis with local contributing factors (60)
- Necrotizing ulcerative gingivitis (NUG) (23, 80)
- Puberty-associated gingivitis (60, 93)
- Menstrual cycle-associated gingivitis (60)
- Pregnancy-associated gingivitis (54, 60)
- Pregnancy-associated pyogenic granuloma (60, 87)
- Diabetes mellitus-associated gingivitis (34, 60)
- Leukemia-associated gingivitis (60, 99)
- **Drug-influenced gingival enlargement** (60, 85)
- Oral contraceptive-associated gingivitis (60)
- Ascorbic acid-deficiency gingivitis (19, 60)
Various forms of gingival enlargement
Gingival enlargement

May be sign of an underlying systemic disorder

A full medical history should always be taken

Careful extraoral and intraoral examinations are necessary to determine the nature and extent of the lesion, additional signs and predisposing or traumatic factors.

Referral to a specialist centre for additional investigations may be appropriate.
Chronic hyperplastic gingivitis may occur following prolonged accumulation of dental plaque. It is frequently associated with concomitant systemic medications, though predisposing factors may not be identifiable. There is firm, pink gingiva, enlargement, particularly at interdental sites, although an inflammatory component may also be present. The gingiva may partially cover the crowns of teeth, resulting in aesthetic problems and cleaning difficulties.

Treatment: OHI, scaling and gingivectomy.
Epulides

- Localised hyperplastic lesions arising from the gingiva.
- Caused by trauma and chronic irritation from plaque and calculus → invoke a chronic inflammatory response → continued inflammation and repair occur concurrently → excessive production of granulation tissue → epulis.
Epulides

- Fibrous epulis
- Vascular epulis
- Peripheral giant cell granuloma
a) Fibrous epulis

- firm, pink, pedunculated mass that may be ulcerated if traumatized.
- histology: chronically inflamed, hyperplastic fibrous tissue which may be richly cellular or densely collagenous.
- metaplastic bone and/or foci of dystrophic calcification are common.
b) Vascular epulis

- Include pyogenic granuloma and pregnancy epulis.
- Characteristic: soft, purple/red swelling, frequently ulcerated which bleeds readily.
- Histology: proliferation of richly vascular tissue supported by a fibrous stroma, with a thin and often extensively ulcerated epithelium.
- Pregnancy epulis occurring in pregnant women.
c) Peripheral giant cell granuloma (GCG)

- Dark reddish/purple, ulcerated swelling, frequently arising interdentally and often extending buccally and lingually.
- May cause superficial erosion of crestal alveolar bone.
- Radiographs are essential to differentiate from a central GCG that has perforated the cortex to present as a peripheral swelling.
- Histology: contains multiple foci of osteoclast-like giant cells supported by a richly vascular and cellular stroma.
Treatment for epulides

- Surgical excision.
- Pregnancy epulis can be prevented by the removal of plaque and calculus, as well as OHI.
- In pregnancy, surgical excision without complete elimination of local irritants, can be recurrence
Iatrogenic gingival enlargement

- Denture induced enlargement
- Orthodontically induced enlargement
Denture induced enlargement

- chronic trauma from ill-fitting dentures + poor OH → hyperplasia of gingival tissues.
- Sign: edematous, erythematous and bleed readily.
- treatment: OHI, denture hygiene, SRP, and replacement of defective prostheses.
Orthodontically induced enlargement

- results in the ‘heaping-up’ of gingival soft tissues in the direction of tooth movement.
- More often with removal appliances
- Resolve on completion of orthodontic treatment.
- Treatment : OHI
Cystic lesions

- Gingival cysts
- Developmental lateral periodontal cysts
Cystic lesions

**Gingival cysts**

- < 1% of cysts of the jaws.
- common in neonates, tend to resolve spontaneously in early life.
- in adults, it is asymptomatic and found by chance in histological sections from gingivectomy specimens.
- probably odontogenic in origin, arising from remnants of dental lamina.
Developmental lateral periodontal cysts

- produces localized destruction of the periodontal tissues along a lateral root surface.
- may present with expansion of alveolar bone, but most are incidental findings on radiographs.
- resemble gingival cysts if arising near the alveolar bone crest.
- radiographically, appear as a radiolucency with well-defined bony margins.
- Treatment for cystic lesions: surgical excision
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         a. without other local contributing factors
         b. with local contributing factors (See VIII A)
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         a. associated with the endocrine system
            1) puberty-associated gingivitis
            2) menstrual cycle-associated gingivitis
            3) pregnancy-associated
               a) gingivitis
               b) pyogenic granuloma
            4) diabetes mellitus-associated gingivitis
         b. associated with blood dyscrasias
            1) leukemia-associated gingivitis
            2) other
      3. Gingival diseases modified by medications
         a. drug-influenced gingival diseases
            1) drug-influenced gingival enlargements
            2) drug-influenced gingivitis
               a) oral contraceptive-associated gingivitis
               b) other
      4. Gingival diseases modified by malnutrition
         a. ascorbic acid-deficiency gingivitis
         b. other
# Clinical and histopathologic feature of ANUG

<table>
<thead>
<tr>
<th>Clinical conditions</th>
<th>Clinical feature</th>
<th>Histophatology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Necrotizing ulcerative gingivitis</td>
<td>Fetid odor, painful gingiva, sudden onset, punched-out interdental papillae, marginal and interdental papilla affected. Pseudomembranous slough. Local lymphadenopathy and slight elevation in temperature might be present</td>
<td>Surfaces epithelium degenerated and replace by pseudomembrane. The connective tissue is hyperemic with numerous capillaries and a dense infiltration of PMN. The layer between necrotic and living tissue contains numerous number of fusiform bacilli and spirochetes in addition to leucocyte. Spirochetes has been show to invade the underlying living tissue</td>
</tr>
</tbody>
</table>
Necrotizing Ulcerative Gingivitis

- Pain and (spontaneous) bleeding
- Fetor exoris
- Punched out papillae
- Grey pseudo-membrane
- Fusospirochetal infection
Necrotizing Ulcerative Gingivitis

Microbiology

- *Fusobacterium* sp. (spirochetes)
- *Treponema* sp.
- *Porphyromonas gingivalis*
- *Prevotella intermedia*
Necrotizing Ulcerative Gingivitis

Secondary Etiology
- Impaired Chemotaxis
- Poor Oral Hygiene
- Alcohol
- Smoking
- Malnutrition
- Stress
Necrotizing Ulcerarive Gingivitis (Healing)
**Figure 1.**
Classification of periodontal diseases and conditions.

*Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing.*

<table>
<thead>
<tr>
<th>I. Gingival Diseases</th>
<th>3. Gingival diseases of fungal origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Dental plaque-induced gingival diseases*</td>
<td>a. <em>Candida</em>-species infections</td>
</tr>
<tr>
<td>1. Gingivitis associated with dental plaque only</td>
<td>1) generalized gingival candidosis</td>
</tr>
<tr>
<td>a. without other local contributing factors</td>
<td>b. linear gingival erythema</td>
</tr>
<tr>
<td>b. with local contributing factors (See VIII A)</td>
<td>c. histoplasmosis</td>
</tr>
<tr>
<td>2. Gingival diseases modified by systemic factors</td>
<td>d. other</td>
</tr>
<tr>
<td>a. associated with the endocrine system</td>
<td></td>
</tr>
<tr>
<td>1) puberty-associated gingivitis</td>
<td></td>
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<tr>
<td>2) menstrual cycle-associated gingivitis</td>
<td></td>
</tr>
<tr>
<td>3) pregnancy-associated</td>
<td></td>
</tr>
<tr>
<td>a) gingivitis</td>
<td></td>
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<tr>
<td>b) pyogenic granuloma</td>
<td></td>
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<tr>
<td>4) diabetes mellitus-associated gingivitis</td>
<td></td>
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<tr>
<td>b. associated with blood dyscrasias</td>
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<tr>
<td>1) leukemia-associated gingivitis</td>
<td></td>
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<tr>
<td>2) other</td>
<td></td>
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<tr>
<td>3. Gingival diseases modified by medications</td>
<td></td>
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<tr>
<td>a. drug-influenced gingival diseases</td>
<td></td>
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<tr>
<td>1) drug-influenced gingival enlargements</td>
<td></td>
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<tr>
<td>2) drug-influenced gingivitis</td>
<td></td>
</tr>
<tr>
<td>a) oral contraceptive-associated gingivitis</td>
<td>b) other</td>
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<tr>
<td>4. Gingival diseases modified by malnutrition</td>
<td></td>
</tr>
<tr>
<td>a. ascorbic acid-deficiency gingivitis</td>
<td></td>
</tr>
<tr>
<td>b. other</td>
<td></td>
</tr>
<tr>
<td>B. Non-plaque-induced gingival lesions</td>
<td></td>
</tr>
<tr>
<td>1. Gingival diseases of specific bacterial origin</td>
<td></td>
</tr>
<tr>
<td>a. <em>Neisseria gonorrhoea</em>-associated lesions</td>
<td></td>
</tr>
<tr>
<td>b. <em>Treponema pallidum</em>-associated lesions</td>
<td></td>
</tr>
<tr>
<td>c. streptococcal species-associated lesions</td>
<td></td>
</tr>
<tr>
<td>d. other</td>
<td></td>
</tr>
<tr>
<td>2. Gingival diseases of viral origin</td>
<td></td>
</tr>
<tr>
<td>a. herpesvirus infections</td>
<td></td>
</tr>
<tr>
<td>1) primary herpetic gingivostomatitis</td>
<td></td>
</tr>
<tr>
<td>2) recurrent oral herpes</td>
<td></td>
</tr>
<tr>
<td>3) varicella-zoster infections</td>
<td></td>
</tr>
<tr>
<td>b. other</td>
<td></td>
</tr>
<tr>
<td>6. Traumatic lesions (factitious, iatrogenic, accidental)</td>
<td></td>
</tr>
<tr>
<td>a. chemical injury</td>
<td></td>
</tr>
<tr>
<td>b. physical injury</td>
<td></td>
</tr>
<tr>
<td>c. thermal injury</td>
<td></td>
</tr>
<tr>
<td>7. Foreign body reactions</td>
<td></td>
</tr>
<tr>
<td>8. Not otherwise specified (NOS)</td>
<td></td>
</tr>
</tbody>
</table>
Non-plaque-induced gingival lesions

- Gingival diseases of specific bacterial origin e.g. N. gonorrhoea, T. pallidum, and Streptococcal species
- Gingival diseases of viral origin e.g. primary herpetic lesion, recurrent oral herpes, varicella-zoster
- Gingival diseases of fungal origin e.g. generalized gingival candidosis, linear gingival erythema, histoplasmosis
- Gingival lesion of genetic origin e.g. hereditary gingival fibromatosis
- Gingival manifestations of systemic conditions e.g. mucocutaneous disorders, allergic reactions to metals and dentifrices
- Traumatic lesions of chemical, physical, and thermal origin
- Foreign body reaction
- Not otherwise specified (NOS)
## Clinical and histopathologic feature of Acute Herpetic Gingivostomatitis

<table>
<thead>
<tr>
<th>Clinical conditions</th>
<th>Clinical feature</th>
<th>Histophatology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Herpetic gingivostomatitis</td>
<td>Involvement of gingiva and may include mucosa and lips. Appear vesicular, erythemathous, shiny with varying degree of edema and gingival bleeding. After 24 hours vesicles may rupture forming painful ulcers with a hello-like margin and depressed grayish white central portion. More often in children with a duration on of 7-10 days</td>
<td>Etiology-Herpes Simplex. Ulceration that result from rupture of vesicles resulting in a central portion of acute inflammation with purulent exudates surrounded by a zone rich a blood vessels</td>
</tr>
</tbody>
</table>
HIV-associated gingivitis

Linear Gingival Erythema:
- Persistent, linear, erythematous gingivitis.
- Localized/generalized
- Erythematous gingiva may be:
  - Limited to marginal gingiva.
  - Extend into attached gingiva and/or alveolar mucosa.
Gingival fibromatosis

is an uncommon condition with autosomal dominant inheritance pattern.

There is generalised fibrous enlargement of the gingiva as a result of the accumulation of bundles of collagen fibres.

It is frequently associated with fibrous enlargement of the maxillary tuberosities.

Treatment is usually not required, unless access for cleaning is impaired or aesthetics are compromised. It tends to recur following surgical excision.
# Clinical and histopathologic feature of Desquamative Gingivitis and Pericoronitis

<table>
<thead>
<tr>
<th>Clinical conditions</th>
<th>Clinical feature</th>
<th>Histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desquamative Gingivitis</td>
<td>Intense redness and desquamation of the epithelium. Gingiva and mucosa involved</td>
<td>Disruption of the epithelial-connective tissue with the formation of subepithelial bullae. These lesions characteristically have immunoglobulins bound</td>
</tr>
</tbody>
</table>
Table 1. Systemic factors shown to modulate gingivitis expression

<table>
<thead>
<tr>
<th>Factor/condition</th>
<th>Gingival inflammation modulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic</td>
<td></td>
</tr>
<tr>
<td>puberty</td>
<td>+</td>
</tr>
<tr>
<td>pregnancy</td>
<td>+</td>
</tr>
<tr>
<td>diabetes</td>
<td>+</td>
</tr>
<tr>
<td>Genetic</td>
<td></td>
</tr>
<tr>
<td>Down’s syndrome</td>
<td>+</td>
</tr>
<tr>
<td>Environmental</td>
<td></td>
</tr>
<tr>
<td>smoking</td>
<td>-</td>
</tr>
<tr>
<td>vitamin C deficiency</td>
<td>+</td>
</tr>
<tr>
<td>antibiotics</td>
<td>-</td>
</tr>
<tr>
<td>calcium channel blockers</td>
<td>+</td>
</tr>
<tr>
<td>corticosteroids</td>
<td>-</td>
</tr>
<tr>
<td>cyclosporin</td>
<td>+</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>-</td>
</tr>
<tr>
<td>phenytoin</td>
<td>+</td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>immune (PMN) deficiency</td>
<td>+</td>
</tr>
<tr>
<td>leukemia</td>
<td>+</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>+</td>
</tr>
<tr>
<td>psychological stress</td>
<td>+</td>
</tr>
</tbody>
</table>

+ Heightened gingival response to plaque.
— Reduced gingival response to plaque.
NSAIDs: non-steroidal anti-inflammatory drugs.
PMN: polymorphonuclear granulocytes.
HIV: human immunodeficiency virus.
AIDS: acquired immune deficiency syndrome.
Treatment for chronic gingivitis
Treatment

THERAPEUTIC GOALS

- The therapeutic goal is to establish gingival health
- through the elimination of the etiologic factors: plaque, calculus, and other plaque-retentive factors
Contributing systemic risk factors may affect treatment and therapeutic outcomes for plaque-induced gingivitis. These may include diabetes, smoking, and certain periodontal bacteria, aging, gender, genetic predisposition, systemic diseases and conditions (immunosuppression), stress, nutrition, pregnancy, substance abuse, HIV infection, and medications.
A treatment plan for active therapy should be developed that may include the following:

- Patient education and customized oral hygiene instruction.
- Debridement of tooth surfaces to remove supra and subgingival plaque and calculus.
- Antimicrobial and antiplaque agents or devices may be used to augment the oral hygiene efforts of patients who are partially effective with traditional mechanical methods.
Treatment

Correction of plaque-retentive factors such as over-contoured crowns, open and/or overhanging margins, narrow embrasure spaces, open contacts, ill-fitting fixed or removable partial dentures, caries, and tooth malposition.

In selected cases, surgical correction of gingival deformities/enlargement that hinder the patient’s ability to perform adequate plaque control may be indicated.

Following the completion of active therapy, the patient’s condition should be evaluated to determine the course of future treatment.
Treatment

<table>
<thead>
<tr>
<th>Trial author</th>
<th>Trial length (months)</th>
<th>No. of patients</th>
<th>Agent used</th>
<th>Plaque reduction (%)</th>
<th>Gingivitis reduction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grossman et al. (1989)</td>
<td>6</td>
<td>481</td>
<td>CHX 0.12%</td>
<td>49</td>
<td>31</td>
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<tr>
<td>Grossman et al. (1986)</td>
<td>6</td>
<td>380</td>
<td>CHX 0.12%</td>
<td>61</td>
<td>39</td>
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<tr>
<td>Loe et al. (1976)</td>
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<td>CHX 0.2%</td>
<td>45</td>
<td>27</td>
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<tr>
<td>Lang et al. (1982)</td>
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<td>CHX 0.1%</td>
<td>16</td>
<td>67</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>CHX 0.2%</td>
<td>19</td>
<td>80</td>
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<tr>
<td>Gordon et al. (1985)</td>
<td>9</td>
<td>85</td>
<td>Listerine (EO)</td>
<td>19.5</td>
<td>23.9</td>
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<td>Lamster et al. (1983)</td>
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<td>145</td>
<td>Listerine</td>
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<td>Overholser et al. (1990)</td>
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<td>124</td>
<td>Listerine</td>
<td>36.1</td>
<td>35.9</td>
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<tr>
<td>Charles et al. (2001)</td>
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<td>Listerine</td>
<td>56.1</td>
<td>22.9</td>
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<tr>
<td>DePaola et al. (1989)</td>
<td>6</td>
<td>107</td>
<td>Listerine</td>
<td>34</td>
<td>34</td>
</tr>
</tbody>
</table>

CHX = chlorhexidine; EO = essential oil.
Treatment for gingival enlargement

A strict programme of OHI and plaque control must be implemented. Overgrown tissues should be surgically excised.
Gingival recession
POSITION

- Level of the gingival margin which is attached to the tooth
- Abnormality – gingival recession
GINGIVA RECESSION
GINGIVAL RECESSION

Recession is exposure of the root surface by an apical shift in the position of the gingiva.

- Actual position – level of the epithelial attachment on the tooth
- Apparent position – level of the crest of the gingival margin
- Severity of the recession is determined by the actual position not apparent position.
APPARENT & ACTUAL LENGTH
ETIOLOGY

- Faulty tooth brushing technique (gingiva abrasion)
- Tooth malposition
- Friction from soft tissue (gingiva ablation)
- Gingival inflammation
- Abnormal frenum attachment
- Usage of a particular abrasive dentrifice
ETIOLOGY

- Traumatic incisor relationship
- Habits such as rubbing gingiva with a finger nail or end of a pencil
- Orthodontic movement in labial direction
- Smoking
- Aging - not due to physiological shift of the gingival epithelium but cumulative effect of minimal pathologic involvement
CLINICAL FEATURES

- Exposed root surface due to apical migration of the gingival complex
- Stillman’s cleft – an incipient lesion, a narrow, deep and slightly curved cleft extending apically from the free gingival margin
- McCall’s festoon – rolled, thickened band of gingiva
STILLMAN’S CLEFT & MCCALL’S FESTOON

Stillman’s Cleft

McCall’s festoon
PREDISPOSING FACTORS

Natural defects in labial alveolar plates -
**Dehiscences** (clefts) or **fenestrations** (windows)
CLINICAL SIGNIFICANCE

- Exposed root surface is susceptible to caries
- Abrasion or erosion of the cementum which subsequently cause hypersensitivity due to exposed dentinal tubules
- Hyperemia of the pulp
- Interproximal recession causes plaque accumulation
TREATMENT

- Record the magnitude of recession to assess progression or stability (clinically or on study models)
- Eliminate etiological factors
- Oral Hygiene Instruction
- Topical desensitising agent / fluoride varnish
- Gingival veneer to cover exposed roots / embrasure spaces
TREATMENT

Crown teeth with extreme caution to prevent exposure of coronal pulp at level of radicular preparation.

Mucogingival surgery to correct the recession either a lateral pedicle graft, double papilla flap or coronally repositioned flap.

Mucogingival surgery to provide a wider and functional zone of attached gingiva using free gingiva flap.
MUCOGINGIVAL SURGERY USING FREE GINGIVAL FLAP

BEFORE

AFTER
REFERENCES

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THANK YOU