Chronic Gingivitis ★
Chronic Periodontitis ★
Acute Periodontitis ★
Chronic Gingivitis, Chronic Periodontitis & Acute Periodontitis

For each of these conditions this presentation aims to improve your ability to:-

• Explain the disease process
• Know how to recognise
• Describe basic management
This BPE score is most likely to indicate a diagnosis of:-
A. Chronic Gingival Hyperplasia
B. Marginal Gingivitis
C. Generalised Chronic Gingivitis
D. Generalised gingivitis with localised mild chronic periodontitis
E. Necrotising Ulcerative Gingivitis
F. Difficult to decide, could be B or C.
Gingivitis

Necrotising-Ulcerative
Plaque-induced
Marginal

Eruptive Puberty-associated
Desquamative Drug-influenced
Non-plaque-induced

Menstrual-cycle-associated Pregnancy-associated
Oral-Contraceptive-associated
Ascorbic Acid Deficiency Leukaemia-associated
Diabetes-associated
Hormonal

Hyperplastic
Gingivosis
What is Gingivitis?

Gingivitis = Inflammation of the gingivae.
No loss of attachment; i.e. any pockets must be false.
No breakdown of PDL fibres.
No bone loss.
Changes are reversible.

The first stage of periodontal disease, although some patients never progress beyond gingivitis.

Gingival Disease = Any disease of the gingivae.
The Gingivae in Health

- Pink
- Firm
- Stippled
- Knife edge margin
- No tendency for bleeding
Health maintained by:-

- Intact junctional epithelium attached to tooth surface.
- Shedding of epithelial cells.
- Collagen fibres maintain form of tissues and aid attachment to tooth.
- GCF flows out through gingival sulcus.
- Antimicrobial effects of antibodies
- Phagocytosis by neutrophils and macrophages.
- Complement activity.
Clinical Signs of Gingivitis

• Redness (starting at the papillae and progressing along the gingival margin).
• Loss of stippling.
• Surface smooth and glossy.
• Swelling (oedema). The tissues become softer and ‘pit’ on pressing.
• Rolling of the gingival margin and loss of the triangular shape of the interdental papillae.
• Bleeding on gentle probing.
Early Gingivitis
Advanced Chronic Gingivitis
Additional Signs of Gingivitis

• Bad or metallic taste in mouth
• Halitosis
• False pocketing
• Fibrous tissue reaction
Histopathology of plaque induced gingivitis

- Vasodilation & ↑ capillary permeability
- Collagen breakdown
- More inflammatory cells
- ↑ GCF
Histopathology of plaque induced gingivitis – more detail

Page and Schroeder, 1976

Initial, early and established gingival Lesions

Studies of animals, not possible to diagnose clinically
Gingival Indices

Gingival Index – Loe and Silness, 1963
Modified Gingival Index – Lobene, 1986
Gingival Bleeding Index – Carter and Barnes, 1974
Eastman Interdental Bleeding Index – Caton and Polson, 1985
Papillary Bleeding Index – Muhlemann, 1977

.....to name a few
Loe and Silness 1963 Gingival Index

Subjective assessment of gingivitis based on colour, consistency and bleeding.

- 0 = normal
- 1 = mild inflammation, slight colour change and oedema, no bleeding
- 2 = moderate inflammation, redness, edema, bleeds on probing
- 3 = severe inflammation, marked redness and oedema, ulceration, spontaneous bleeding

4 sites on each tooth examined and divided by number of sites examined to give average score.
Why treat gingivitis?


• Gingivitis is a risk predictor for periodontal disease.

• Not all patients who have gingivitis develop periodontal disease but where gingivitis is consistently present the patient there is likely to be 70% more attachment loss than at healthy sites and the patient is 46 times more likely to lose the tooth over a 26 year period (Schätzle et al. 2003, 2004).
What treatment is indicated for plaque induced gingivitis?

A. Patient dental education.
B. Oral Hygiene Instruction.
C. Encourage behavioural change.
D. Debridement of calculus.
E. Removal of plaque retentive factors where possible.
F. All of the above.
Gingival Diseases - 1999 International Workshop

**Plaque-induced Gingival Diseases**

- **With local contributing factors**
  - Associated with Plaque Only
  - Modified by Systemic Factors
  - Drug influenced gingival enlargement e.g. Nifedipine, Amlodipine
  - Modified by Malnutrition
- **Without local contributing factors**
  - Associated with blood dyscrasias i.e. Leukemia associated or other
  - Drug influenced gingivitis e.g. OCP
  - Other
## Non-Plaque induced Gingival Lesions

### Specific Bacterial
- Neisseria gonorrhoea associated
- Treponema pallidum associated
- Streptococcal associated
- Other

### Viral
- Herpes
- Other

### Fungal
- Candida associated
- Other

### Genetic
- Hereditary gingival fibromatosis
- Other

### Gingival manifestation of Systemic Condition
- Mucocutaneous disorder
- Allergic reaction to restorative materials
- Allergic reaction to foodstuffs, additives, oral hygiene products

### Traumatic
- Chemical
- Physical
- Thermal

### Foreign body reactions

### Not otherwise specified
Periodontitis

Chronic

Recurrent

Rapidly-progressive

Necrotising-Ulcerative

Juvenile

Early-Onset

Acute

Associated-with-Systemic-Disease

Prepubertal

Aggressive

Localised

Generalised

Refractory
Periodontal Diseases - 1999 International Workshop

- **Chronic Periodontitis**
  - Localised or Generalised

- **Aggressive Periodontitis**
  - Localised or Generalised

- **Periodontitis as a manifestation of systemic disease**
  - Associated with **haematological** e.g. leukaemia, neutropenia or **genetic** disorders e.g. Down syndrome or hypophosphatasia or others not specified

- **Developmental or acquired deformities and conditions**
  - Localised tooth-related factors
  - Mucogingival deformities around teeth
  - Mucogingival deformities of edentulous ridges
  - Occlusal trauma

- **Abscesses of the Periodontium**
  - Gingival
  - Periodontal
  - Pericoronal

- **Necrotising Periodontal Diseases** NUG/NUP

- **Periodontitis associated with endodontic lesions**
Clinical signs of Periodontitis

- Some or all the signs of gingivitis
- True pocketing on probing
- Recession
- Suppuration
- Mobility above physiological levels
- Drifting of teeth
- Exposure of furcations
- Radiographic evidence of bone loss
Distinguishing between chronic and aggressive forms of periodontitis

• Most patients with plaque induced periodontitis will have the chronic form of the disease.

• The typical patient with chronic periodontitis is over 35 yrs, with substantial deposits of plaque and calculus associated with gingival inflammation, periodontal pockets and attachment loss. In most cases the disease is slowly progressing but short periods of attachment loss can occur.

• When distinguishing consider:
  ✴ Amount, pattern and rate of progress of the condition in relation to microbial deposits and presence of inflammation.
  ✴ Patient’s age and medical status and familial tendency.
Historical vs. Active disease: Does it affect our treatment?

• Scenario 1
  A 5mm pkt with bleeding on probing and purulent discharge where subgingival deposits can be detected.

• Scenario 2
  A 5mm pkt with no bleeding on probing and no purulent discharge where subgingival deposits can be detected.

• Scenario 3
  A 5mm pkt with no bleeding on probing and no purulent discharge where no subgingival deposits can be detected.
Treatment for Chronic Periodontitis

- Minimise risk factors where possible
- Maximise plaque control
- Full mouth debridement
- Reassess, if not stabilised disease process, determine reason where possible prior to proceeding
- Consider indications and suitability for surgical/adjunctive therapies
- Supportive therapy
Chronic Periodontitis

Aim to control not cure the condition
This is the gingival appearance around a 26 year old male patient’s lower incisors, you see a yellow/grey slough which covers the gingival tissues, the gingival tissues bleed readily, and the patient reports that his ‘gums’ are extremely painful. You also notice the patient’s breath has a particularly pungent malodour. What is the most likely diagnosis?

A. Chronic Severe Periodontitis
B. Necrotising Ulcerative Gingivitis
C. Necrotising Ulcerative Periodontitis
D. Lateral Periodontal Abscess
Necrotising Ulcerative Gingivitis

- Previously known as ANUG, Trench mouth, Vincent’s disease
- Systemic changes predispose the gingivae to invasion by the oral flora, in particular spirochaetes and fusiform bacteria
- Contributory systemic factors include:
  - Severe malnutrition
  - Heavy smokers
  - Emotional stress
  - Compromised immune system
  - Blood dyscrasias e.g. acute leukaemia
Prevalence NUG/NUP

- Most prevalent 16-30 years
- Military recruits
- Prevalence reduced over last 30 years due to improved general health, nutrition and plaque control
- Seen in patients with HIV and AIDS, should be considered where NUG/NUP diagnosed with no other predisposing factors
Histology of NUG

• Necrotic surface epithelium and underlying connective tissue.
• Dense acute inflammatory infiltration of underlying tissues - PMNLs
• Bacteria, particularly spirochaetes invade the tissues
• Underlying viable tissue with increased numbers of plasma cells, lymphocytes and macrophages
<table>
<thead>
<tr>
<th>Necrotising Ulcerative Gingivitis – Diagnostic features</th>
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<tbody>
<tr>
<td><strong>Site of Ulcers</strong></td>
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<td><strong>Character of Ulcers</strong></td>
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<td><strong>Fever</strong></td>
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<td><strong>Symptoms</strong></td>
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<td><strong>Duration of Ulcers and Discomfort</strong></td>
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<td><strong>Breath odour</strong></td>
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<td><strong>Lymphadenopathy</strong></td>
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</tbody>
</table>
Necrotising Ulcerative Periodontitis – Diagnostic features

• Deep interproximal craters with denudation of interdental alveolar bone.

• Sequestration of interdental and possibly buccal and/or lingual alveolar bone.

• Loss of attachment.
Possible causes of gingival ulceration that is not characteristic of NUG/NUP

**Viral Infections**
- Acute herpetic gingivostomatitis
- Recurrent intraoral herpes
- Varicella
- Herpes Zoster
- Infectious mononucleosis

**Bacterial Infections**
- Streptococcal gingivitis
- Gonococcal gingivitis
- Syphilis
- Tuberculosis
- Leprosy

**Mucocutaneous conditions**
- Desquamative gingivitis
- Pemphygoid
- Pemphigus
- Erythema multiforme
- Oral lichen planus
- Lupus erythematosus

**Traumatic Conditions**
- Traumatic ulcerative gingival lesions
- Toothbrushing
- Flossing
- Toothpicks/woodsticks
- Factious gingival ulceration
Management of NUG/NUP

**Acute Phase**
- Irrigation ideally with ultrasonic scaler
- Use of oxygen releasing mouthwash e.g. hydrogen peroxide 1.5% w/v e.g. Colgate Peroxyl or a sodium perborate mouthwash
- Metrinidazole 200mg TDS for 3-5 days is the antibiotic of choice
- Chlorhexidine mouthwash can be used in the short term where oral hygiene is important
- Consider predisposing factors and counsel where appropriate

**Residual Condition**
- Aim: minimise risk of recurrence
- Meticulous supra and subgingival debridement
- Remove any local predisposing factors
- In extreme cases gingivoplasty may improve gingival contour
- Regular review to maintain high standard of OH
- If apparently unexplained cause consider medical examination and blood screening for major predisposing factor
Noma - Cancrum Oris

‘The ulcer of extreme poverty’

Cases in concentration camps of WW2, in association with intense poverty, immunosuppressive therapy in patients with HIV or AIDS; remains prevalent in Sub-Saharan Africa.
In 1998 the WHO estimated 140,000 children contract Noma each year and 79% of them die from the disease and associated complications.
Cancrum Oris

- Diseases that commonly precede Noma are: measles, malaria, severe diarrhoea, malnourishment, poor sanitation, compromised immune system and **Necrotising Ulcerative Gingivitis**

- Early features include: soreness of the mouth, pronounced halitosis, foetid taste, tenderness of the lips or cheek, cervical lymphadenopathy, a foul smelling purulent oral discharge and a swollen blue-black discolouration of the skin of the affected area

- Multiple bacteria associated particularly fusiform bacilli and spirochaetes.

- hCMV, HSV and measles virus also associated.
Recommendations for management of Acute Noma

- Correction of dehydration and electrolyte imbalance
- Nutritional rehabilitation
- Treat predisposing diseases
- Antibiotics (penicillin and metronidazole)
- Local wound irrigation with antiseptic
- Physiotherapy to reduce fibrous scarring
- Removal of any remaining slough and sequestra
- Serological test for HIV and appropriate referral if positive.

Enwonwu et al. 2006
Acute Periodontal Abscesses

- Can be acute or chronic with acute exacerbations.
- Pain
- Lateral gingival oedema
- Erythema
- Bleeding and/or suppuration on probing
- Tooth hypermobility
- Tooth may be tender to percussion, particularly to lateral forces
- Lymphadenopathy, fever and malaise rare
- Radiographs may show bone loss depending on stage of abscess
Predisposing factors for the formation of a periodontal abscess

- Periodontal disease
- Calculus remaining in pocket after debridement
- Penetration of bacteria into soft tissue wall of periodontal pocket
- Recent course of antibiotics often for non-oral reasons
- Diabetes mellitus
- Traumatic impaction of foreign body
**Differential Diagnoses for Periodontal Abscesses when the presentation is a red painful swelling of the periodontal tissues**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiation</th>
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<tr>
<td>Gingival Abscess</td>
<td>Location, Periodontal health</td>
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<tr>
<td>Periapical Abscess</td>
<td>No Pulpal response, Apical lesion on radiograph, No or narrow increased probing depth</td>
</tr>
<tr>
<td>Pericoronal Abscess</td>
<td>Partially erupted tooth, Adjacent vital teeth with no increased pocket depths</td>
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<tr>
<td>Incomplete Root Fracture</td>
<td>Clinical finding of fracture, Radiographic finding of fracture</td>
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<tr>
<td>Endodontic / Post Perforations</td>
<td>Radiographic features (parallax technique)</td>
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Gingival Abscess

- Confined to marginal gingival tissues often at previously non-diseased sites.
- Often an acute inflammatory response to the impaction of a foreign body or material into the gingival sulcus.
- Removal of offending material, history of 1-2 days localised pain associated with localised gingival swelling with a red, shiny surface often diagnostic.
Periodontal abscess caused by tooth fracture
Combined Perio-Endo Lesions

Can have a periodontal or endodontic cause

Simon, Glick & Frank’s 1972 Classification

- Class 1 - Primary endodontic lesion draining through PDL
- Class 2 - Primary endodontic lesion with secondary periodontal involvement
- Class 3 - Primary periodontal lesion
- Class 4 - Primary periodontal lesion with secondary endodontic involvement
Treatment Options for a Combined Perio-Endo lesion

• If class 1 endodontic treatment and reassessment
• If class 3 periodontal treatment and reassessment
• If combined endodontic and periodontal treatment

• Root resection
• Extraction

The best option will depend on a number of local and general patient factors which should be considered during treatment planning
Know how to recognise and treat:

- Chronic Gingivitis
- Chronic Periodontitis
- NUG/NUP
- Periodontal abscesses

- Be aware of perio-endo lesions