LEMBAR PENGESAHAN

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KETUA PROGRAM STUDI

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KLASIFIKASI PENYAKIT JARINGAN PERIODONTAL

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Dental Plaque–Induced Gingival Diseases

These diseases may occur on a periodontium with no attachment loss or on one with attachment loss that is stable and not progressing.

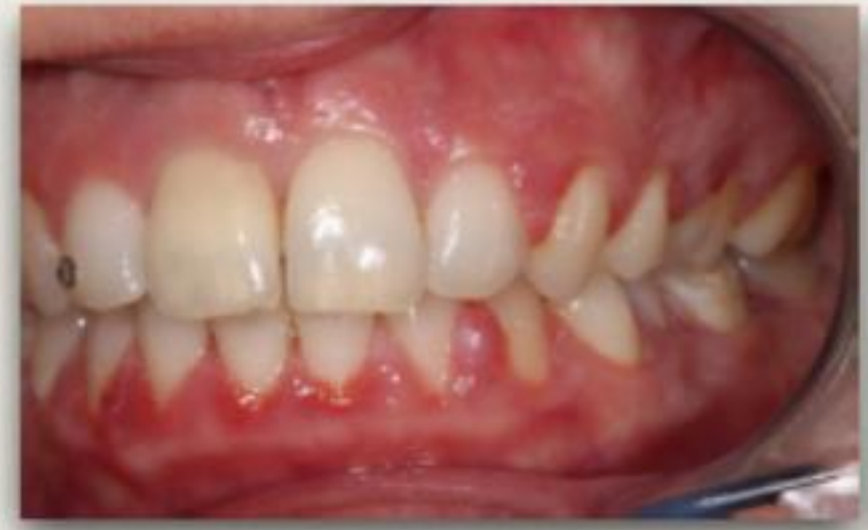
I. Gingivitis associated with dental plaque only
   A. Without local contributing factors
   B. With local contributing factors (see Box 4-4)
II. Gingival diseases modified by systemic factors
   A. Associated with the endocrine system:
      1. Puberty-associated gingivitis
      2. Menstrual cycle–associated 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
III. 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
IV. Gingival diseases modified by malnutrition
   A. Ascorbic acid deficiency gingivitis
   B. Other

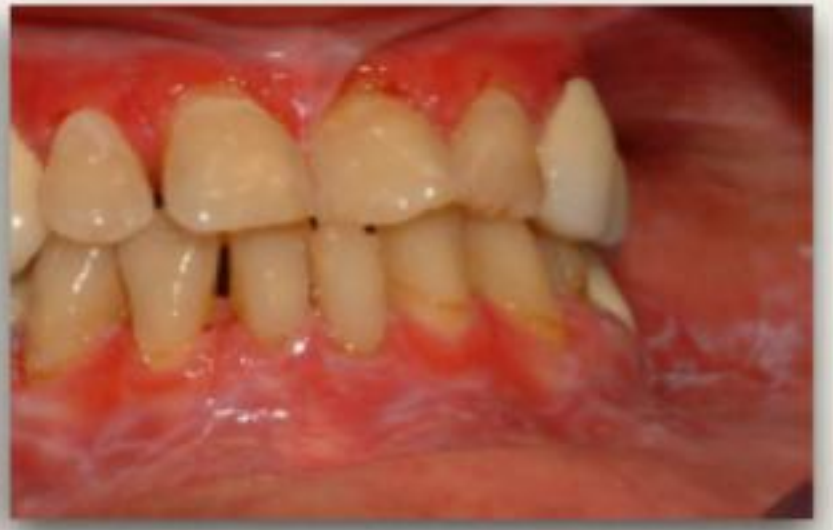
Non–Plaque-Induced Gingival Lesions

I. Gingival diseases of specific bacterial origin
   A. *Neisseria gonorrhea*
   B. *Treponema pallidum*
   C. Streptococcal species
   D. Other
II. Gingival diseases of viral origin
   A. Herpesvirus infections
      1. Primary herpetic gingivostomatitis
      2. Recurrent oral herpes
      3. Varicella zoster
   B. Other
III. Gingival diseases of fungal origin
   A. *Candida*-species infections: Generalized gingival candidosis
   B. Linear gingival erythema
   C. Histoplasmosis
   D. Other
IV. Gingival lesions of genetic origin
   A. Hereditary gingival fibromatosis
   B. Other
V. Gingival manifestations of systemic conditions
   A. Mucocutaneous lesions
      1. Lichen planus
      2. Pemphigoid
      3. Pemphigus vulgaris
      4. Erythema multiforme
      5. Lupus erythematosus
      6. Drug induced
      7. Other
   B. Allergic reactions
      1. Dental restorative materials
         a. Mercury
         b. Nickel
         c. Acrylic
         d. Other
      2. Reactions attributable to
         a. Toothpastes or dentifrices
         b. Mouthrinses or mouthwashes
         c. Chewing gum additives
         d. Foods and additives
      3. Other
VI. Traumatic lesions (factitious, iatrogenic, or accidental)
   A. Chemical injury
   B. Physical injury
   C. Thermal injury
VII. Foreign body reactions
VIII. Not otherwise specified

Diseases of the gum

- dental plaque induced gingival diseases
- non-plaque induced gingival lesions





Gingivitis -→ proses peradangan jaringan periodonsium terbatas pada gingiva, yang disebabkan oleh mikroorganisme yang membentuk suatu koloni serta membentuk plak gigi yang melekat pada tepi gingival.

Tanda /sign:

- Tak ada kehilangan perlekatan
- Kemerahan pada margin gingiva
- Pembengkakan bervariasi
- Perdarahan pada probing ringan
- Perubahan bentuk gingiva
- Kebanyakan tidak ada rasa sakit

symtomp: bau mulut, berdarah spontan/saat sikat gigi, permukaan gigi kasar

Gingivitis disebabkan plak → murni karena akumulasi plak dan diperparah adanya faktor lokal.--> Gingivitis marginalis kronis

Gingivitis karena faktor non plak → infeksi bakteri spesifik, infeksi virus atau jamur, alergi dan trauma, gangguan sistemik dg perdarahan spontan atau teriritasi, penggunaan obat , radiasi, siklus menstruasi dan genetik.

PATOGENESIS GINGIVITIS

Gambaran klinis gingivitis -→ kemerahan, perdarahan akibat stimulasi, perubahan kontur, adanya plak atau kalkulus dan secara radiografi tidak ditemukan kehilangan tulang alveolar. Pemeriksaan histologi jaringan gingiva yang mengalami peradangan menunjukkan ulserasi epitel.

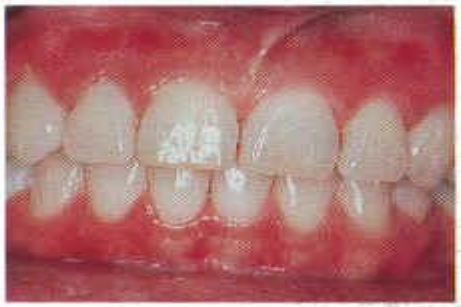
Gejala klinis gingivitis yang parah : eritema, edema, dan pembesaran hiperplastik

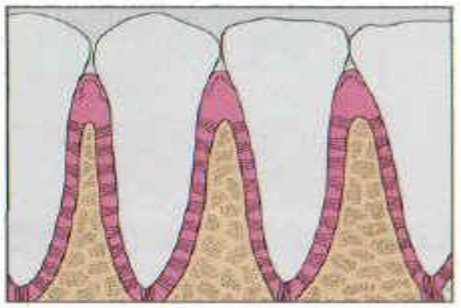
Perubahan patologis sehubungan jumlah MO dl sulcus ggv→mensintesis produk (PMNs, hialuronidase, protease, kondrotin sulfatase, endotoxin)→ kerusakan epithelial & jar ikat, kandungan interselular→ perluasan ruang antara sel *epithelial junction* ➔ masuk jar ikat.

Tahap Gingivitis

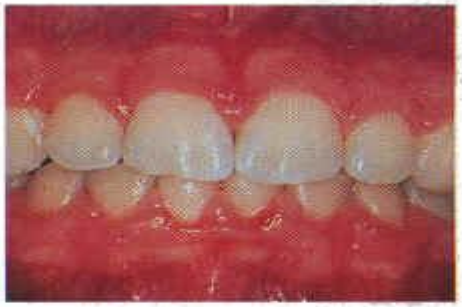
Tahap	Waktu	Pembuluh darah	Epitel sulkuler dan junction	Sel imun yg utama	Kolagen	Tanda klinis
1.*Initial lesion*	2-4 hr	Dilatasi PD dan vaskulitis	Infiltrasi PMNs	PMNs	Kehilangan perivaskuler	Aliran cairan ggv >
2.*Early lesion*	4-7 hr	Proliverasi vaskuler PD	Spt thp 1 pembentukan retepeg area atropi	Lymposit	Kehilangan perivaskuler > diarea infiltrasi	Erytema , perdarahan pada probing
3.*Established lesion*	14-21 hr	Spt thp2 ditambah stasis darah	Spt thp 2 lebh berkbg	Sel plasma	Kerusakan berlanjut	Perub warna, ukuran,tekstur

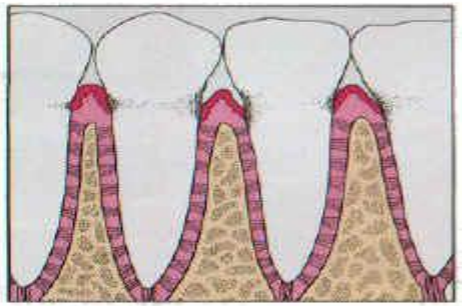
1. Normal, Healthy Gingiva (Gums)
Healthy gums and bone anchor teeth firmly in place.



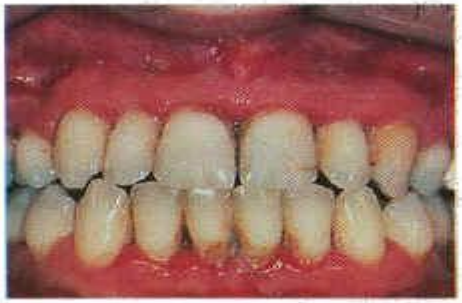


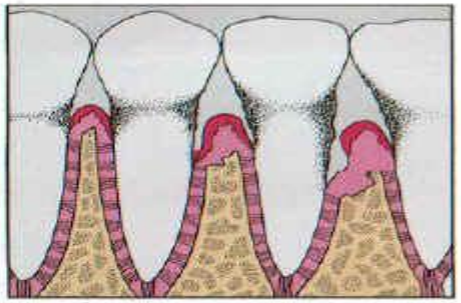
2. Gingivitis
Plaque and its byproducts irritate the gums, making them tender, inflamed, and likely to bleed.



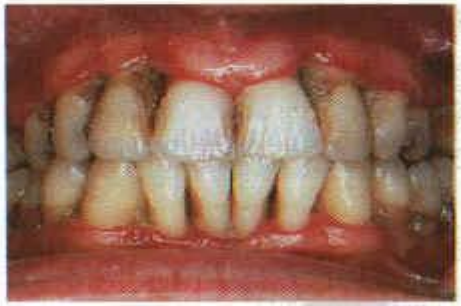


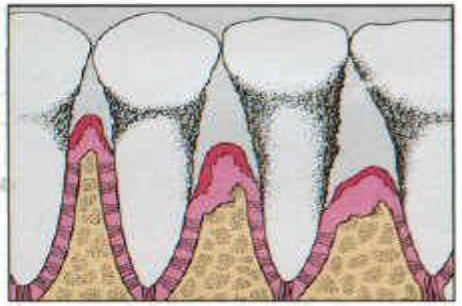
3. Periodontitis
Unremoved, plaque hardens into calculus (tartar). As plaque and calculus continue to build up, the gums begin to recede (pull away) from the teeth, and pockets form between the teeth and gums.





4. Advanced Periodontitis
The gums recede farther, destroying more bone and the periodontal ligament. Teeth—even healthy teeth—may become loose and need to be extracted.





Perubahan klinis konsistensi gingiva pd *Gingivitis kronis*

1. Margin lunak membulat pada penekanan
2. Konsistensi lunak dan gampang pecah
3. Konsistensi jaringan keras

Perubahan histopatologis konsistensi gingiva pd Gingivitis kronis

1.Infiltrasi cairan dan sel-sel dr eksudat inflamasi
2.Degenerasi jar ikat dan epitel.
3.Proliferasi epitel dan fibrosis dg inflamasi kronis yg lama

- *Faktor sistemik*

- Dalam beberapa kelainan sistemik, perdarahan gingiva dpt muncul tiba2 tdk didorong iritasi mekanis atau dpt jg setelah ada iritasi.

- Tendensi perdarahan mungkin disebabkan kegagalan mekanisme penjendalan, kelainan darah (hemofili, leukimia)

Perubahan warna gingiva

Perubahan warna pd gingivitis kr:juml dan ukuran pemblh drh, ketipisan epitel, kuantitas keratinasi Dan pigmentasi epith.

1. Gingivitis kronis :
   warna gingiva normal :coral pink
   - →Peningkatan vaskularisasi → merah
   - →Keratinisasi epitel → merah kebiruan
2. Gingivitis akut : warna merah marginal, difus, bernoda tgt kondisi akut
3. Logam : bismuth, arsen , merkuri, Pb, timah, perak dll

Resesi gingiva

Etiologi :

- Faktor fisiologis yg meningkat sesuai umur
- Kesalahan menyikat gigi
- Mal posisi gigi
- Jar lunak yg rusak
- Frenulum tinggi

Akibat resesi

- **Karies akar**
- **Sensitif permukaan gigi**
- **Hiperemi pulpa**
- **Retensi inter proksimal → tempat penimbunan plak**

Kronis (lokal/ general) Gambaran klinis :

Tahap awal penggembungan kecil papila inter dental dan atau margin ggv→ bertbh besar menutup permukaan mahkota gigi. biasanya pelan tanpa sakit kec ada komplikasi akut atau trauma.

Etiologi :

Plak dan OH jelek (iritasi tumpatan atau alat ortho)

- Pada peradangan kronis → monosit mll sirkulasi darah migrasi ke tempat radang→ menjadi makrofag.-→ aktifasi sistem imun spesifik maka makrofag memproduksi sitokin dan faktor pertumbuhan shg terbentuk jar fibrosis.
- 2 tipe dasar respon jaringan thd hiperplasi gingiva:

1. *Edematous* → gingiva halus, mengkilat, lunak , merah
2. *Fibrous* → gingiva lebih kenyal, hilangnya stippling, buram, lebih tebal, margin membulat (sewarna gusi sehat)

Akut

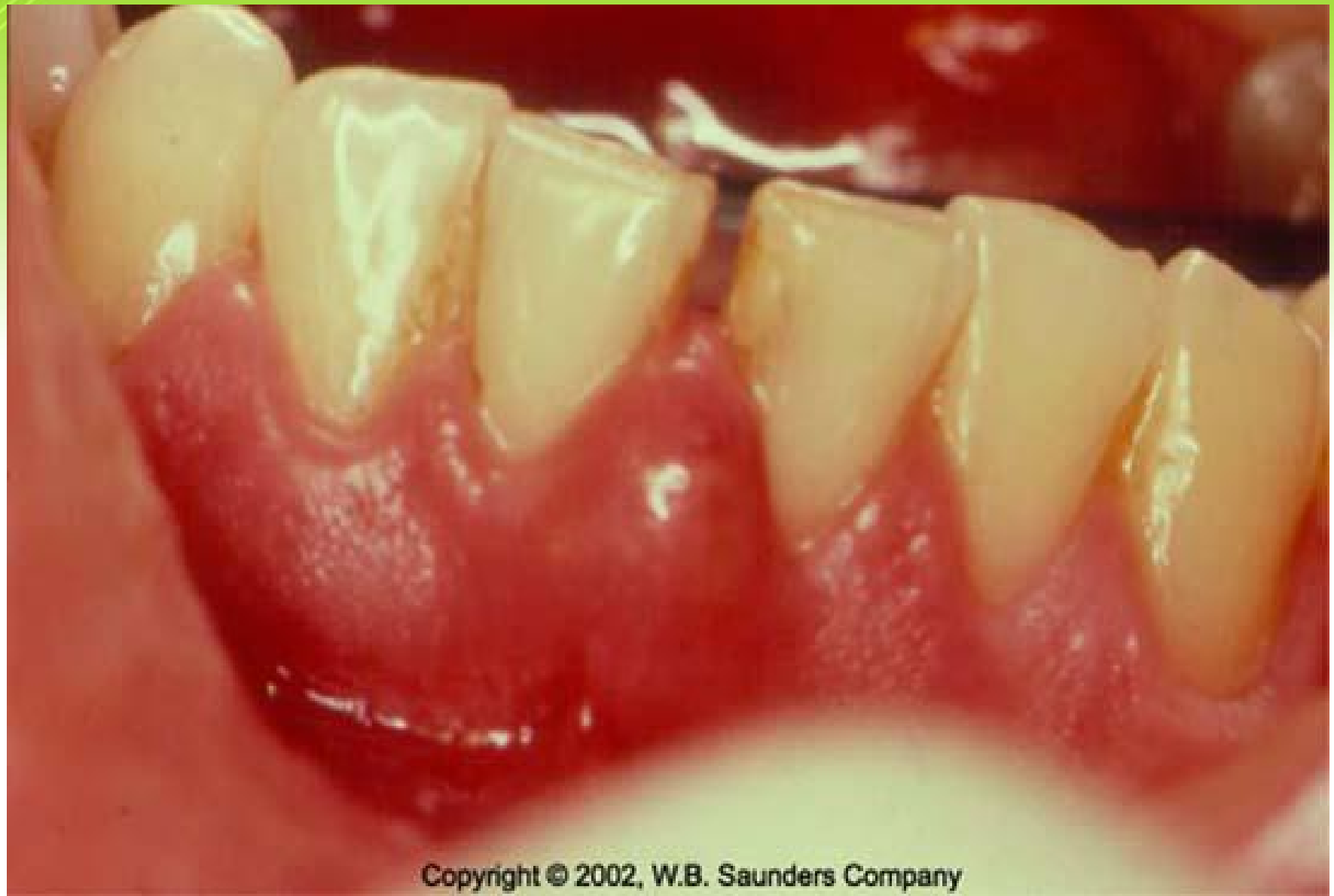
Gingival abses :

1. **Terlokalisir.**
2. **lesi berkembang cepat.**
3. **Terjadi mendadak.**
4. **Terbatas pada margin ggv. .**
5. **lesi biasanya hilang dg sendirinya**
6. **Etiologi : bakteri yg masuk**
   mel sikat gigi, apel, kulit lobster

Periodontal abses

- Biasanya lebih luas
- Telah tjd periodontitis sebelumnya
- Perluasan infeksi poket ke jar perio
- Biasanya akibat perluasan enlarge ment gingiva, ttp juga melibatkan jar periodontal
- Lesi harus dilakukan perawatan bahkan kadang diperlukan dg bedah
- Trauma gigi atau kesalahan perawatan endo

Periodontal abses (fasial)



1. **Phenytoin :**
   Obat anti convulsant utk terapi epilepsi. Sering pd pasien muda. keparahan enlargement sebanding dengan dosis obat yg diminum

2. **Cyclosporin:**
   Suatu agen imunosupresive utk menghindari adanya penolakan terhadap transplantasi organ. Berpengaruh pd respon seluler dan humoral imun respon. Dosis > 500 mg/hr membuat enlargement ggv

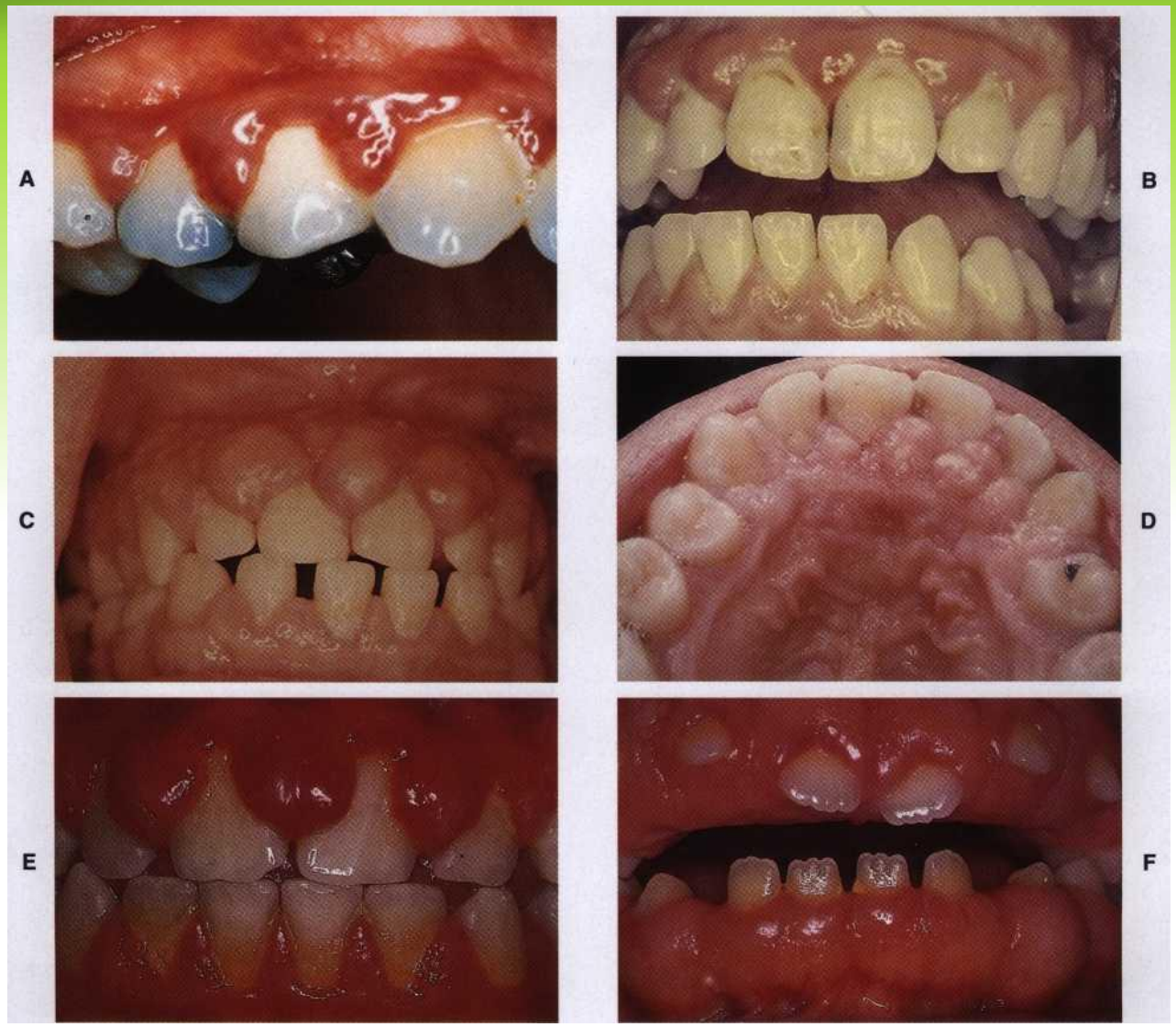
3. **Nifedipine,diltiazem, verapamil**
   calsium chanel bloker, menurunkan hipertensi dengan dilatasi pbl darah perifer
4. ***Idiophatic gingival fibromatosis :***
   suatu kondisi yg tidak diket penyebabnya (gingivomatosis, elephantiasis , fibroma diffuse)

Hipotesis mekanisme :
(1) *Pengaruh obat/metabolit secara tidak langsung.Obat/metabolit* menstimulasi diproduksinya IL-2 oleh sel-T, atau diproduksinya metabolit testosteron oleh fibroblas gingiva menstimulasi proliferasi dan/atau sintesa kolagen oleh fibroblas gingiva;

(2) *Pengaruh obat/metabolit secara langsung.*
Obat/metabolit sec. langsung menstimulasi proliferasi fibroblas gingiva, sintesa protein, dan produksi kolagen;

(3) *Penghambatan aktivitas kolagenase.*
Obat/metabolit →menghambat aktivitas kolagenase → penghancuran matriks akan terhambat;

(4) *Penghambatan degradasi kolagen.*
Obat/metabolit menstimulasi terbentuknya kolagenase fibroblastik inaktif, dengan akibat degradasi kolagen akan terhambat;


A
B
C
D
E
F

Enlargement yg berhub dg penyakit / kondisi sistemik

Terjadi jika kondisi sistemik pasien terpacu oleh iritasi lokal : hormonal(kehamilan, pubertas, nutrisi), def vit C, alergi.

- Enlargement pd pubertas

  Terjadi selama masa pubertas baik laki dan perempuan. Sering pd perm fasial jarang lingual. Setelah melewati pubertas enlargement berkurang dan hilang bila iritasi lokal dihilangkan

- Def vit C

  Def vit C tidak menyebabkan enlargement, tp menyebabkan hemorhagi, deg kolagen , odema jar ikat → plak.

  def vit C + inflamasi → enlargement

Disamping menyebabkan *scurvy, def. Vit. C*
dikaitkan peny. periodontal memperhebat respon gingiva thd plak dan memperparah oedema, pembesaran, dan pendarahan yg terjadi akibat inflamasi.

Hipotesa mengenai mekanisme berperannya vit. C pd penyakit periodontal:

(1) Level vitamin C ↓↓→ mempengaruhi metabolisme kolagen → mempengaruhi kemampuan regenerasi jaringan
(2) Defisiensi vit. C→ menghambat pembentukan tulang

(3).Peningkatan level vitamin C → meningkatkan aksi khemotaksis dan aksi migrasi lekosit, tanpa mempengaruhi aksi fagositosisnya

(4) Level vitamin C yg optimal diperlukan utk memelihara integritas mikrovaskular periodonsium.

(5) Penurunan level vitamin C yang drastis bisa mengganggu keseimbangan ekologis bakteri dalam plak sehingga meningkatkan patogenitasnya

EG yang berhubungan penyakit sistemik:
DM penyakit yg penting pd periodonsia.

Ada dua tipe diabetes mellitus primer :
(1) *Insulin-dependent diabetes mellitus (IDDM/Tipe I),*
dinamakan diabetes juvenil → tidak adanya insulin; sangat tidak stabil dan sulit dikontrol; ketosis dan koma; tdk didahului
kegemukan; butuh injeksi insulin utk kontrol;
sulit dikontrol; ketosis dan koma; tdk didahului
disertai simtom klasik : polifagia, polidipsia,
poliuria, cenderung mudah infeksi, dan anorexia.

(2) *Non-insulin-dependent-diabetes-mellitus*(NIDDM/Tipe II)

Beberapa hipotesa mengenai keterlibatan DM sbg faktor etiologi penyakit gingiva dan periodontal:

(1) **Terjadinya penebalan membran basal**.
Membran basal kapiler gingiva menebal→ lumen kapiler menyempit →terganggunya difusi O2, ekspresi limbah metabolisme,
migrasi PMN, dan difusi faktor-faktor serummigrasi PMN, dan difusi faktor-faktor serum termasuk antibodi

(2**) Perubahan biokimia**.
Level *cyclic adenosine monophosphate (cAMP),*
yg mengurangi inflamasi, pada penderita DM ↓↓→
memperparah inflamasi gingiva

(3) *Perubahan mikrobiologis.*

↑↑ level glukosa dlm cairan sulkular m'pengaruhi lingk. Subgingival menginduksi perubahan kualitatif bakteri.

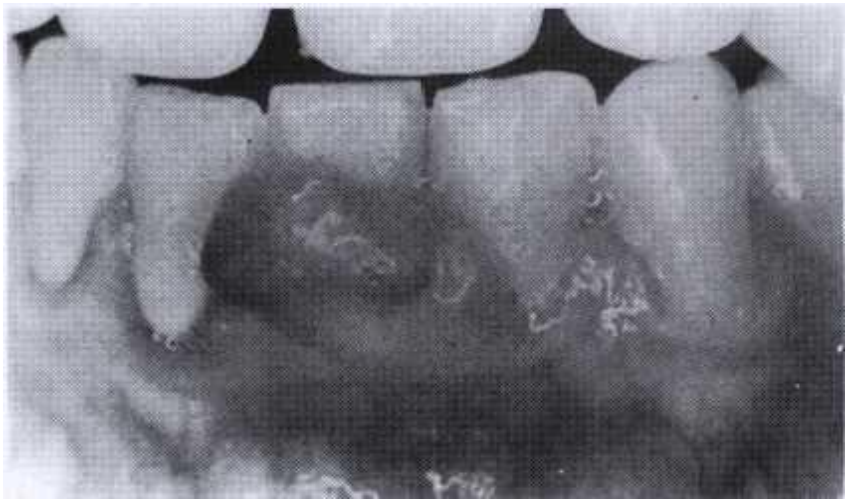
Flora mikroba subgingival pasien periodontitis penderita DM berbeda dgn flora mikroba pasien periodontitis bukan DM.

Mikroba dominan pd pasien periodontitis penderitaDM → spesies *Capnocytophaga, Actinomyces, dan vibrio*anaerob; *Porphyromonas gingivalis, Prevotella intermedia. dll*

(4) *Perubahan imunologis*
Meningkatnya kerentanan penderita DM thd inflamasi kr defisiensi fungsi lekosit polimorfonukleus (LPN) berupa terganggunya khemotaksis, kelemahan daya fagositosis, atau terganggunya kemampuannya untuk melekat ke bakteri
(5) *Perubahan berkaitan dengan kolagen.*
Peningkatan level glukosa menyebabkan berkurangnya produksi kolagen dan peningkatan aktivitas kolagenase pada gingiva.

Enlargement pd kehamilan :

Terjadi pd marginal gingiva dan biasanya general. Bisa terjadi singel atau multipel tumor. Ggv merah, mengkilat, lunak dan halus. Sering terjadi perdarahan spontan. Biasa tjd 3 bl kehamilan. Reduksi tjd setelah selesai kehamilan. Hilang setelah iritasi lokal dihilangkan



PERIODONTITIS

DRG HARTANTI SP.PERIO

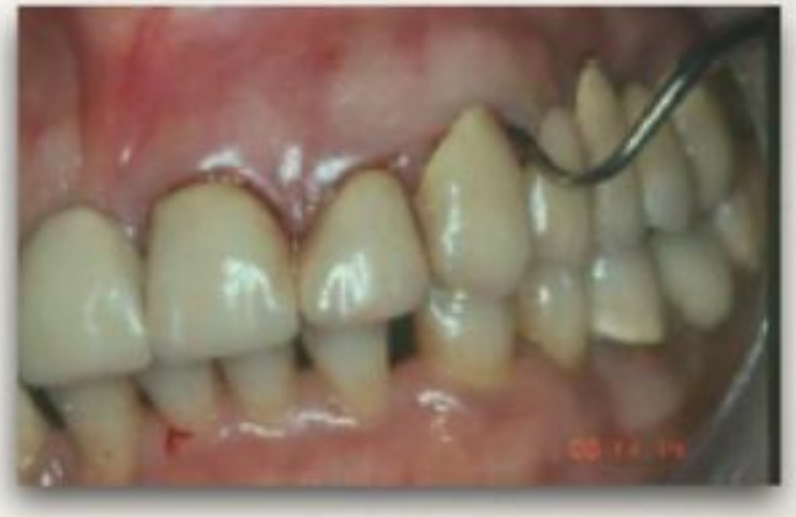
PERIODONTITIS

- Periodontitis is defined as “an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both

  Invasi bakteri ***: Phorpyromonas gingivalis, prevotela intermedia, actinobacilus actinomycetemcomitans***

Chronic periodontitis

- untreated plaque-induced gingivitis may develop into chronic periodontitis
- loss of attachment and bone
- irreversible
- BOP
- increased probing depth, pockets
- recession of the gingival margin
- root furcation exposure
- increased tooth mobility
- drifting and eventually exfoliation of teeth



- Aggressive periodontitis
- ✤ before 1999 - “early onset periodontitis” (EOP), juvenile periodontitis
- ✤ group of rare, often severe, rapidly progressive forms of periodontitis
- ✤ under the age of 35
- ✤ rapid attachment loss and bone destruction
- ✤ familial aggregation of cases
- ✤ elevated proportions of Aggregatibacter actinomycetemcommitans, Porphyromonas gingivalis
- ✤ phagocyte abnormalities
- ✤ hyper-responsive macrophage phenotype, including elevated production of prostaglandin E2 (PGE2)

Classification	Forms of Periodontitis	Deseases characterist
AAP World Workshop in cinical Periodontics, 1989	Adult periodontitis	Age of onset >35 years Slow rate of disease progressi No defects in host defenses
	Early-onset periodontitis (may be prepubertal, juvenile, or rapidly Progressive	Age of onset <35 years Rapid rate of disease progress Defects in host defenses Associated with specific micro
	Periodontitis associated with systemic disease	Systemic diseases that predisp to rapid rates of periodontitis Diseases: diabetes, Down syn HIV infection, Papillon-Lefèvre syndrome
	Necrotizing ulcerative periodontitis	Similar to acute necrotizing ulcerative gingivitis but with associated clinical attachment

European Workshop in Periodontology, 1993	Refractory periodontitis	Recurrent periodontitis that does not respond to treatment
	Adult periodontitis	Age of onset: fourth decade of life Slow rate of disease progression No defects in host response
	Early-onset periodontitis	Age of onset: before fourth decade of life Rapid rate of disease progression Defects in host defense
	Necrotizing periodontitis	Tissue necrosis with attachment and bone loss

AAP International Workshop for Classification of Periodontal Diseases, 1992

The disease periodontitis can be subclassified into the following three major types based on clinical, radiographic, historical, and laboratory characteristics.

CHRONIC PERIODONTITIS
AGGRESSIVE PERIODONTITIS
PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASES

Chronic Periodontitis

The following characteristics are common to patients with chronic periodontitis:

- Prevalent in adults but can occur in children.
- Amount of destruction consistent with local factors.
- Associated with a variable microbial pattern.
- Subgingival calculus frequently found.
- Slow-to-moderate rate of progression with possible periods of rapid progression.
- Possibly modified by or associated with the following:
- Systemic diseases such as diabetes mellitus and human immunodeficiency virus (HIV) infection.
- Local factors predisposing to periodontitis.
- Environmental factors such as cigarette smoking and emotional stress.

Chronic periodontitis may be further subclassified into localized and generalized forms and characterized as slight, moderate, or severe based on the common features described above and the following specific features:

- Localized form: <30% of sites involved.
- Generalized form: >30% of sites involved.
- Slight: 1 to 2 mm clinical attachment loss (CAL).
- Moderate: 3 to 4 mm CAL.
- Severe: ≥5 mm CAL.

Aggressive Periodontitis

The following characteristics are common to patients with aggressive periodontitis:

- Otherwise clinically healthy patient.
- Rapid attachment loss and bone destruction.
- Amount of microbial deposits inconsistent with disease severity.
- Familial aggregation of diseased Individuals.

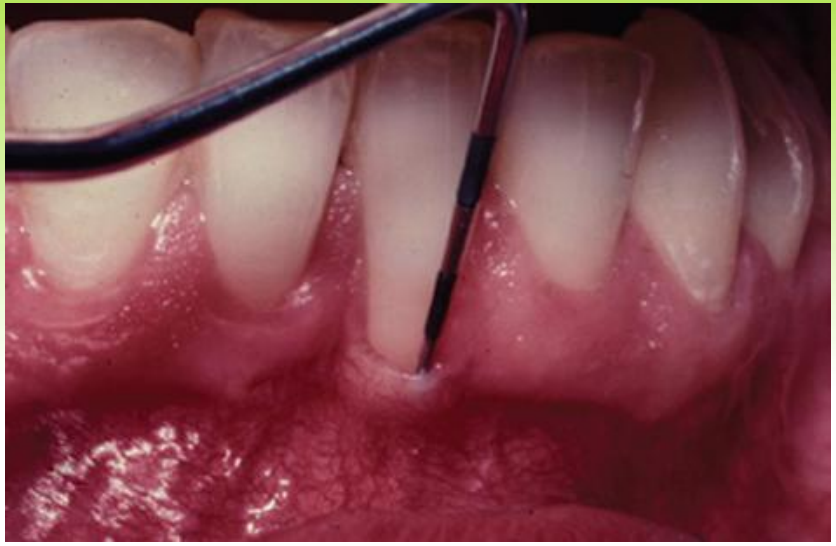
The following characteristics are common but not universal:

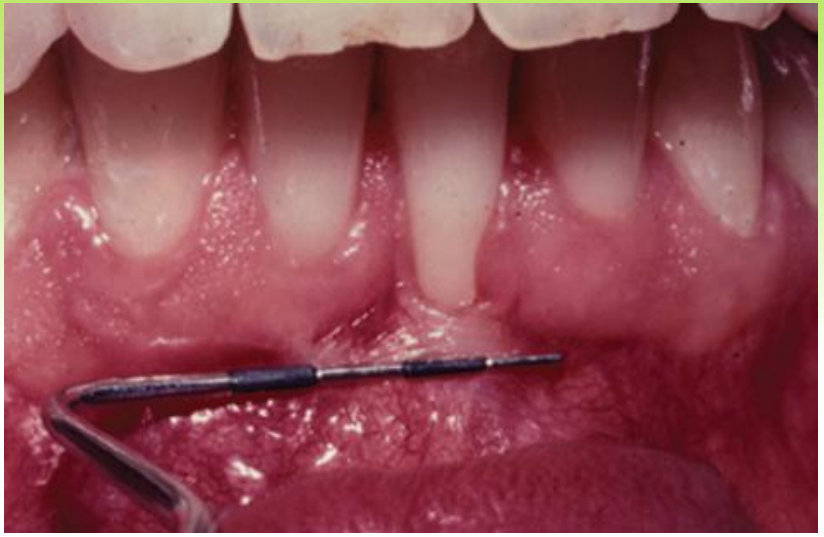
- Diseased sites infected with *Agregatibacter actinomycetemcomitans.*
- Abnormalities in phagocyte function.
- Hyperresponsive macrophages, producing increased prostaglandin E2 (PGE2) and interleukin-1β (IL-1β).

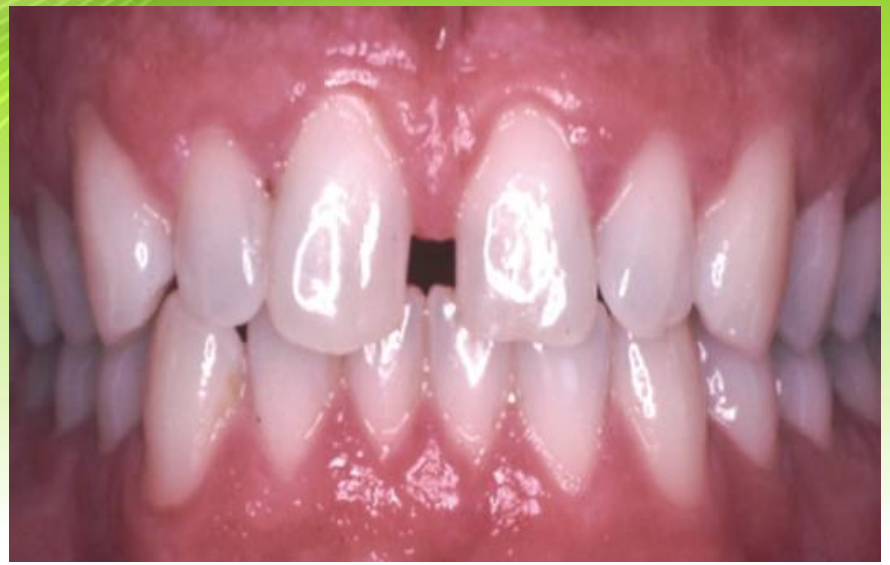
agressive periodontitis ;	
Localized form • Circumpubertal onset of disease. • Localized first molar or incisor disease with proximal attachment loss on at least two permanent teeth, one of which is a first molar. • Robust serum antibody response to infecting agents.	***Generalized form*** • Usually affecting persons under 30 years of age (however, may be older). • Generalized proximal attachment loss affecting at least three teeth other than first molars and incisors. • Poor serum antibody response to infecting agents.

Developmental or Acquired Deformities and Conditions

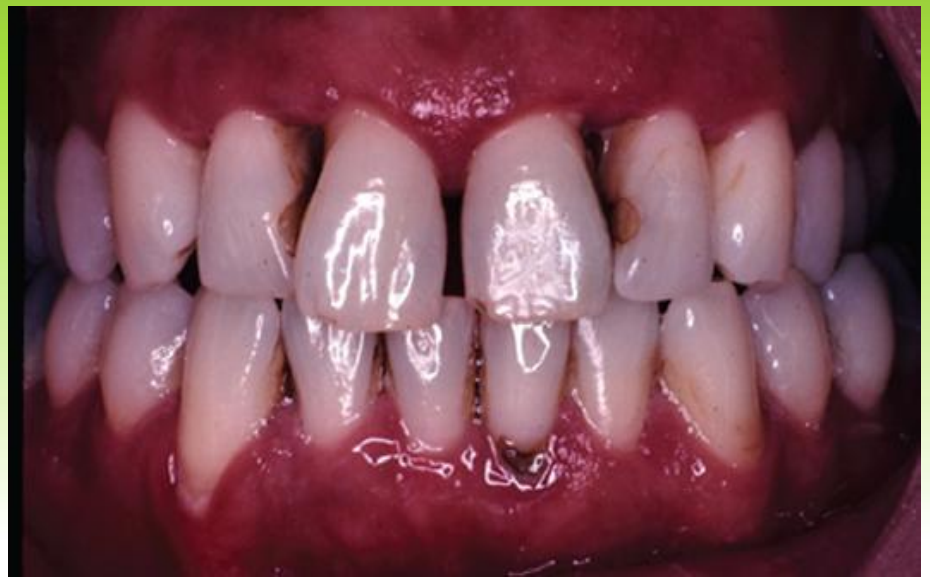
Localized Tooth-Related Factors That Modify or Predispose to Plaque-Induced Gingival Diseases or Periodontitis	Mucogingival Deformities and Conditions around Teeth	Mucogingival Deformities and Conditions on Edentulous Edges
1. Tooth anatomic factors 2. Dental restorations or appliances 3. Root fractures 4. Cervical root resorption and cemental tears	1. Gingival or soft tissue recession a. Facial or lingual surfaces b. Interproximal (papillary) 2. Lack of keratinized gingiva 3. Decreased vestibular depth 4. Aberrant frenum or muscle position 5. Gingival excess a. Pseudopocket b. Inconsistent gingival margin c. Excessive gingival display	1. Vertical and/or horizontal ridge deficiency 2. Lack of gingiva or keratinized tissue 3. Gingival or soft tissue enlargements 4. Aberrant frenum or muscle position 5. Decreased vestibular depth 6. Abnormal color Occlusal Trauma 1. Primary occlusal trauma 2. Secondary occlusal





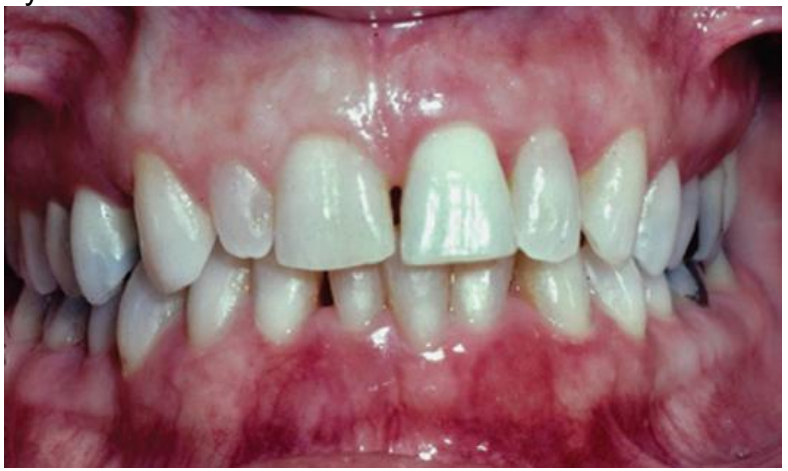


Clinical image of plaque-related slight/early chronic periodontitis with 1 to 2 mm clinical attachment loss in 40-year-old female



Clinical image of plaque-related moderate chronic periodontitis with 3 to 4 mm clinical attachment loss in 53-year-old male smoker

Clinical image of plaque-related severe/advanced chronic periodontitis with 5 mm clinical attachment loss in 47-year-old female

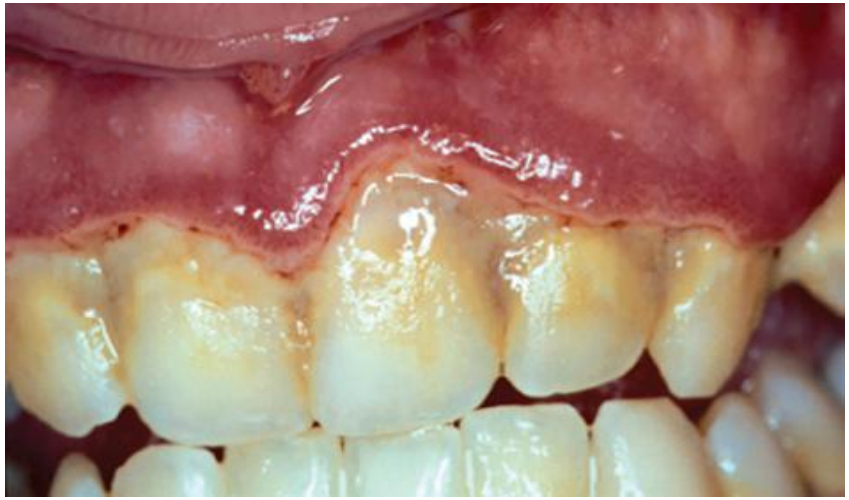


periodontitis as a manifestation of systemic disease is the diagnosis to be used when the systemic condition is the major predisposing factor and local factors, such as large quantities of plaque and calculus, are not clearly evident.

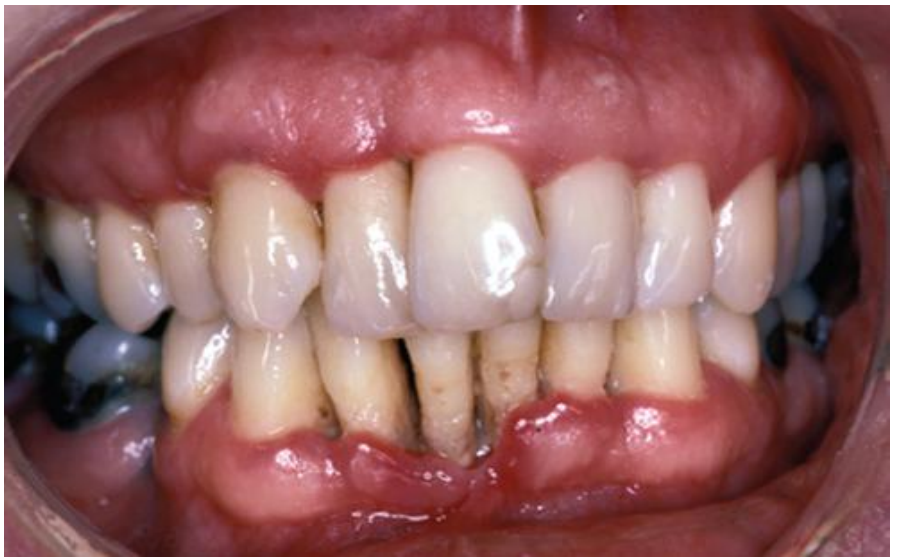
In the case in which periodontal destruction is clearly the result of local factors but has been exacerbated by the onset of such conditions as diabetes mellitus or HIV infection, the diagnosis should be *chronic periodontitis modified by the systemic condition.*

NECROTIZING PERIODONTAL DISEASES

The clinical characteristics of necrotizing periodontal diseases may include but are not limited to ulcerated and necrotic papillary and marginal gingiva covered by a yellowish white or grayish slough or pseudomembrane, blunting and cratering of papillae, bleeding on provocation or spontaneous bleeding, pain, and fetid breath. These diseases may be accompanied by fever, malaise, and lymphadenopathy,



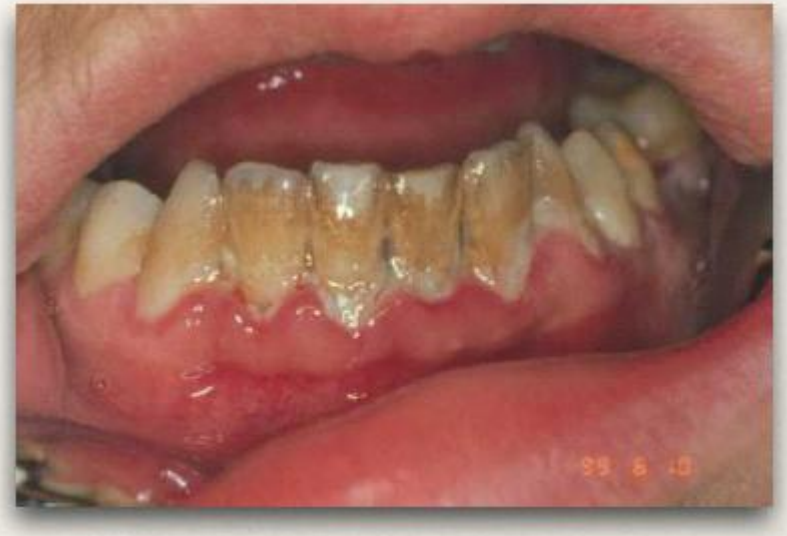
NUG



NUP

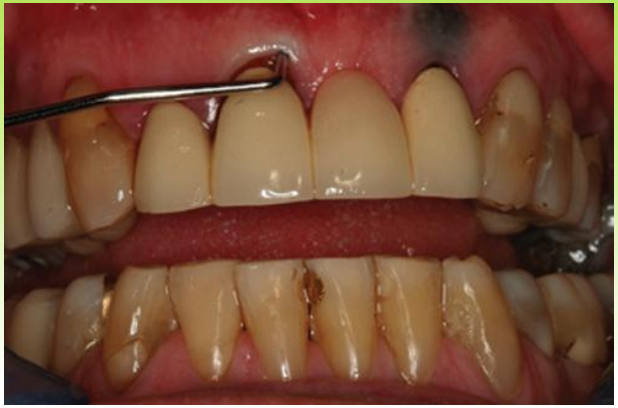
Necrotising periodontal diseases

- Predisposing factors for necrotizing ulcerative gingivitis (NUG) - ("trench mouth disease")
- abundent plaque
- pre-existing gingivitis
- smoking
- psychological stress
- immunsuppression



The defining characteristics of NUG are its bacterial etiology, its necrotic lesion, and predisposing factors such as psychologic stress, smoking, and immunosuppression. In addition, malnutrition may be a contributing factor in developing countries.

Endodontic–Periodontal Lesions



In endodontic–periodontal lesions, pulpal necrosis precedes periodontal changes. A periapical lesion originating in pulpal infection and necrosis may drain to the oral cavity through the periodontal ligament, resulting in destruction of the periodontal ligament and
adjacent alveolar bone.

Periodontal–Endodontic Lesions

In periodontal–endodontic lesions, bacterial infection from a periodontal pocket associated with loss of attachment and root exposure may spread through accessory canals to the pulp, resulting in pulpal necrosis.

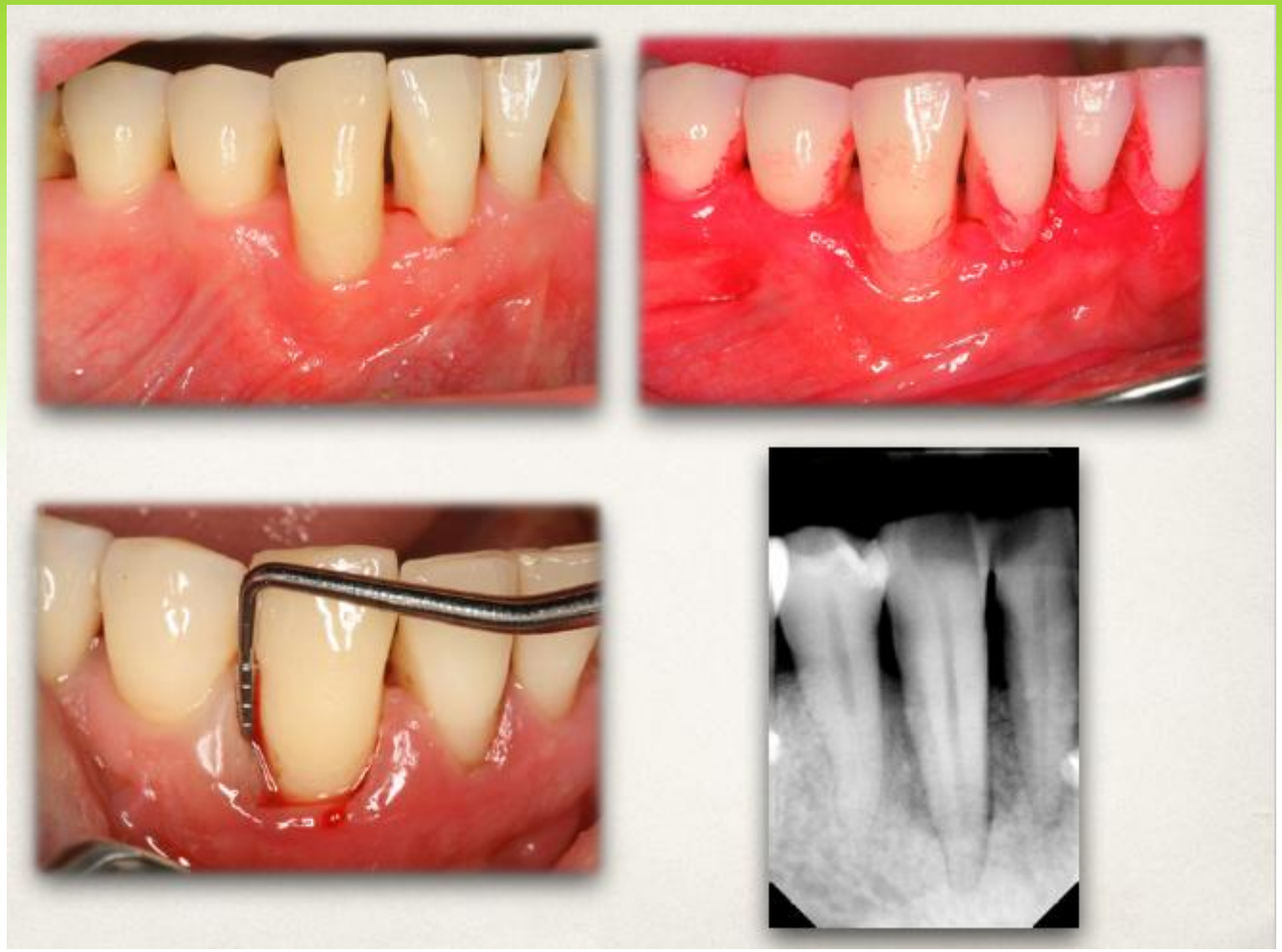
Combined

1). Pockets can be classified as follows:
Gingival pocket /pseudo pocket is formed by gingival enlargement without destruction of the underlying periodontal tissues. The sulcus is deepened because of the increased bulk of the gingiva .

Periodontal pocket /true pocket produces destruction of the supporting periodontal
tissues, leading to loosening and exfoliation of the teeth.
.
Two types of periodontal pockets exist, as follows:
Suprabony *(supracrestal or supraalveolar), in which the bottom of*
the pocket is coronal to the underlying alveolar bone

Intrabony *(infrabony, subcrestal, intraalveolar), in which the bottom*
of the pocket is apical to the level of the adjacent alveolar bone.

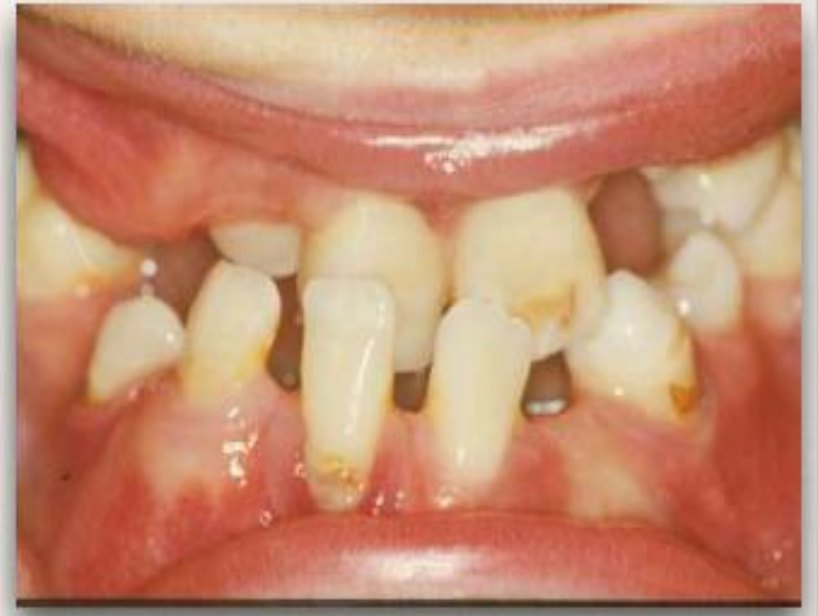
Clinical signs that suggest the presence of periodontal pockets include a bluish red, thickened marginal gingiva; a bluish red, vertical zone from the gingival margin to the alveolar mucosa; gingival bleeding and suppuration; tooth mobility; diastema formation; and symptoms such as localized pain or pain “deep in the bone.”



3. PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASES

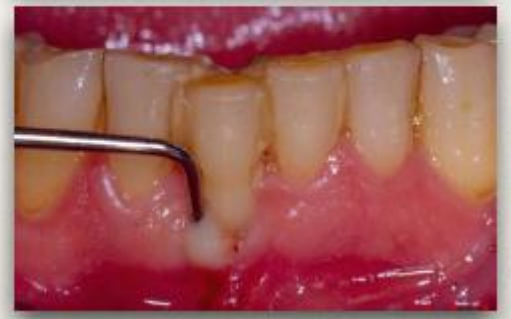
B. ASSOCIATED WITH GENETIC DISORDERS

1. Familial and cyclic neutropenia
2. Down syndrome
3. Leukocyte adhesion deficiency syndrome
4. Kostman's disease
5. Papillion-Lefévre syndrome
6. Chediak-Higashi syndrome
7. Hystiocytosis syndrome
8. Glycogen storage diseases
9. Infantile genetic agranulocytosis
10. Cohen syndrome
11 Ehlers-Danlos syndrome
12. Hypophosphatasia



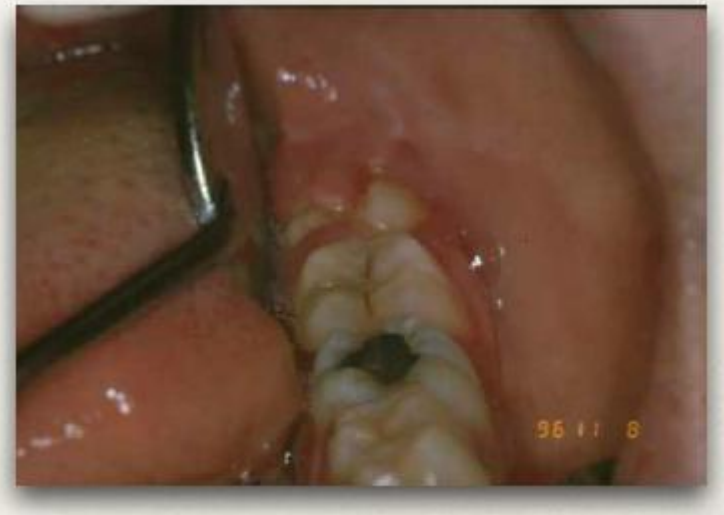
Abscesses of the periodontium

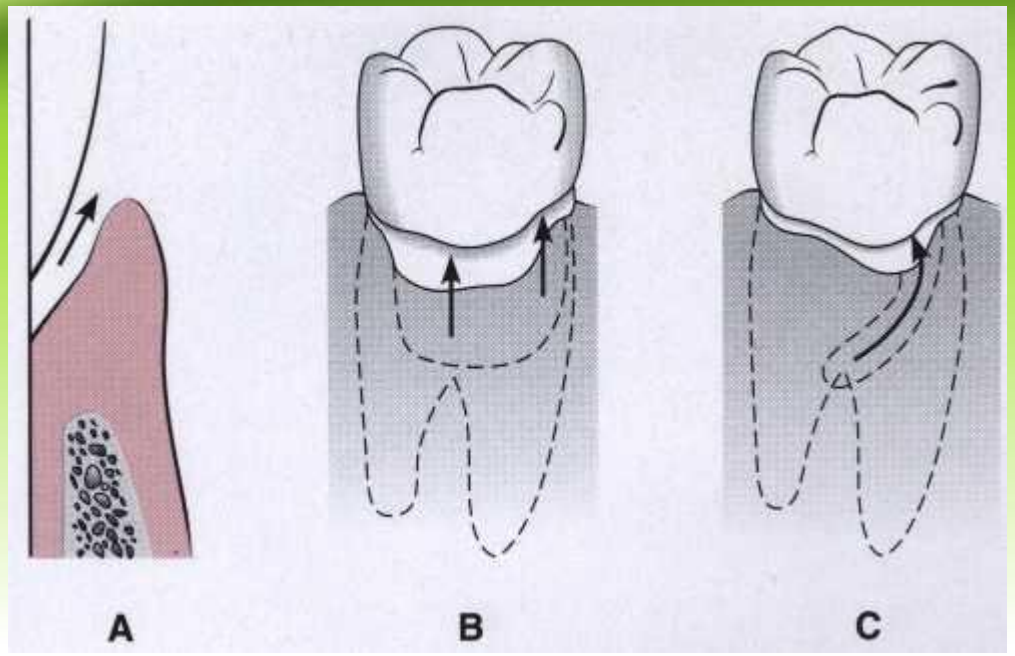
- periodontal abscess
- exacerbation of a chronic lesion
- post-therapy periodontal abscess
- non-periodontitis related abscess
- prominent symptom: presence of an ovoid elevation of the gingival tissues along the lateral side of the root
- suppuration
- pain/sensitivity to percussion, mobility



Abscesses of the periodontium

- pericoronal abscess - at incompletely erupted teeth





Classification of pockets according to involved tooth surfaces. A, Simple pocket. B, Compound pocket. C, Complex pocket.

CLINICAL FEATURES

Clinical signs such as bluish-red, thickened marginal gingiva; a bluish-red vertical zone from the gingival margin to the alveolar mucosa; gingival bleeding, suppuration, or both; tooth mobility; and diastema formation and symptoms such as localized pain or pain "deep in the bone" are suggestive of the presence of periodontal pockets.

- *Klasifikasi poket berdasar bentuk mengelilingi gigi :*
- A.Poket simpel; bila yang terkena 1 permukaan
- B. Poket compound; bila terdapat 2 atau lebih permukaan dengan dasar poket berhubungan langsung dengan margin gingiva
- C. Poket kompleks : poket tipe spiral yaitu melibatkan 2 atau lebih permukaan tetapi sebagian yang berhubungan dengan margin gingiva.

Tanda klinis periodontitis kronis adalah:

1. Inflamasi gingiva dan perdarahan
2. Poket→ Pendlm sulcus scr patologi
3. Migrasi gigi→ muncul distema
4. Mobilitas gigi
5. Kerusakan tulang alveolar
6. Resesi gingiva
7. Kadang-kadang disertai nyeri
8. Halitosis

Dari tanda-tanda diatas, tanda yang paling penting adalah poket (true pocket) dan kerusakan tulang alveolar/reorbsi tulang alveolar, CAL (clinical attachment loss)

Hubungan timbal balik antara penyakit pulpa dg penyakit periodontal

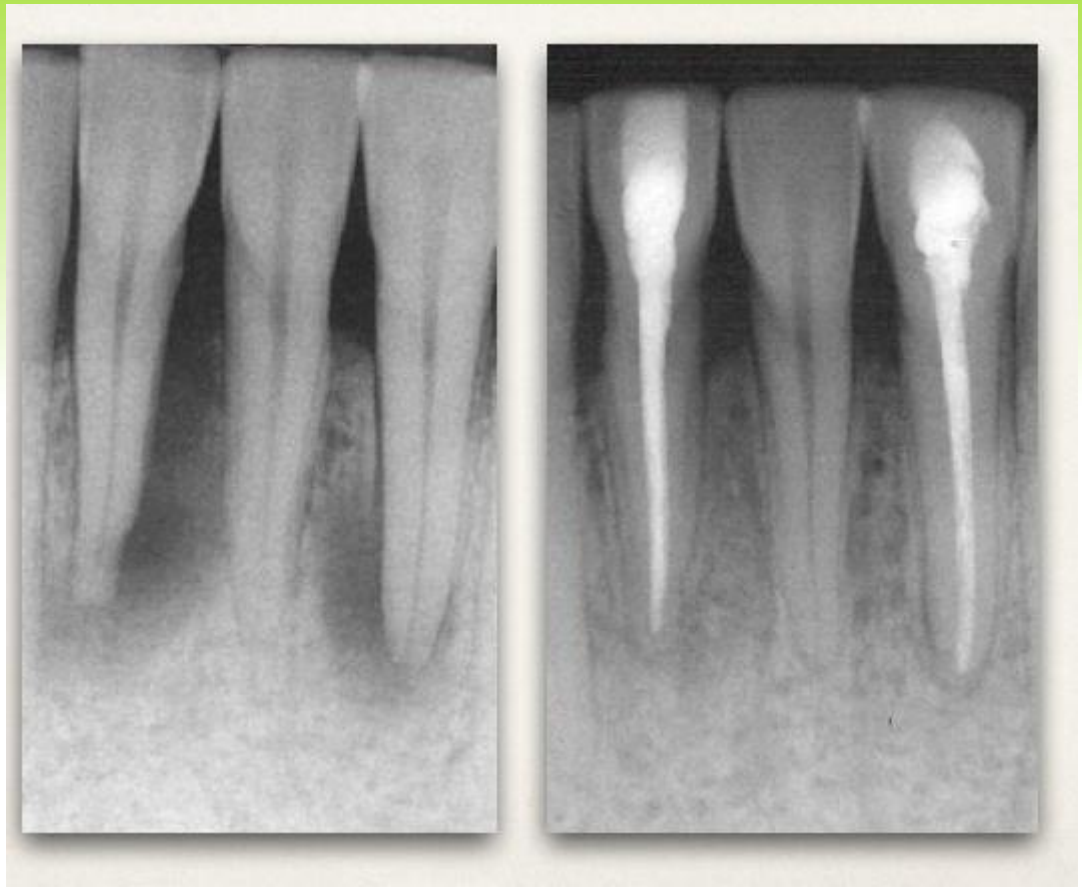
- Lesi pulpa / Pulpitis→ Nekrose→Lesi Periapikal→ Periodontal (periodontitis/Periodontal diseases)

- Periodontitis/Periodontal disease→kanal asesori / foramen periapikal → lesi Pulpa

Periodontitis associated with endodontic lesions

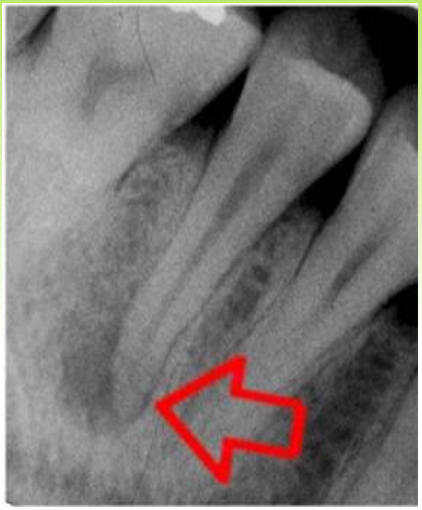
- Combined periodontic-endodontic lesions

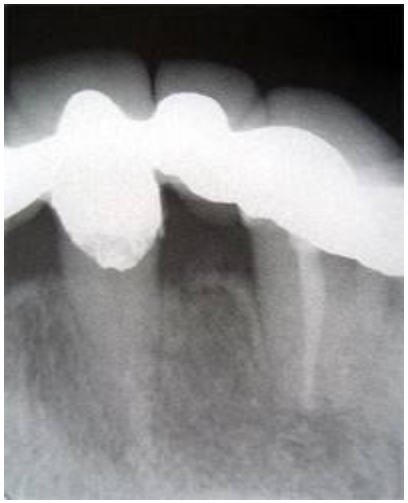
- inflammatory processes of the periodontium associated with necrotic dental pulps have an infectious etiology similar to periodontal diseases
- different source of infection
- role of accessory canals (molars - furcation involvement)
- the vital function of the pulp are rarely threatened by periodontal disease influences



Proses Pathologik penyakit Periodontal

- Bersumber dari lesi periapikal
  - → retrograde Periodontitis
- Bersumber dari margin gingiva
  - → Marginal Periodontitis
- Kombinasi

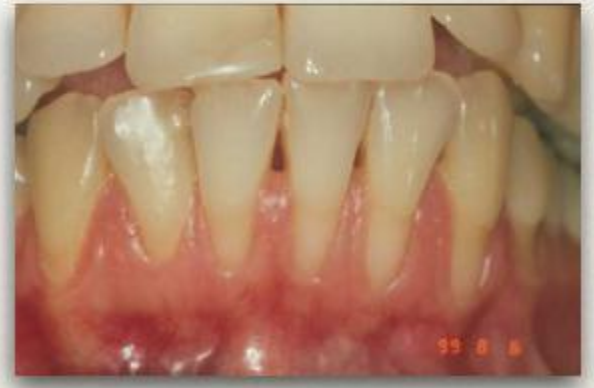




Developmental or acquired deformities and conditions

1. Localized tooth-related factors that modify or predispose to plaque-induced gingival/periodontal diseases

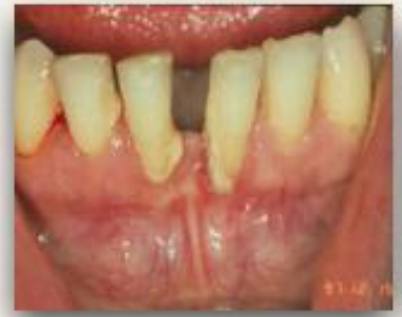
- tooth anatomic factors
- dental restorations/apliances
- root fractures
- cervical root resorption

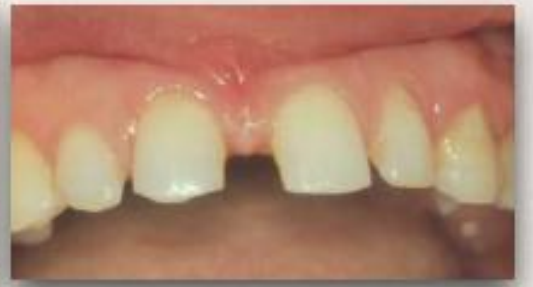


Developmental or acquired deformities and conditions

2. Mucogingival deformities and conditions around teeth

- gingival recession (buccal and lingual)
- gingival recession (interproximal)
- lack of keratinized gingiva
- decreased vestibular depth
- aberrant frenulum/muscle position
- gingival excess: pseudopocket, inconsistent gingival margin, gingival enlargement
- abnormal gingival color





Developmental or acquired deformities and conditions

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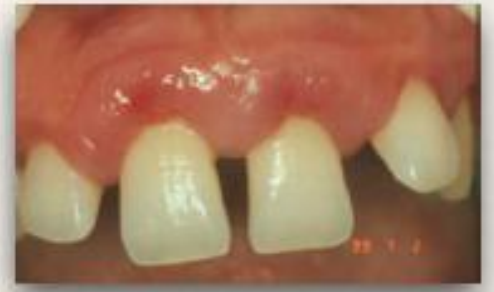

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- gingival recession (interproximal)
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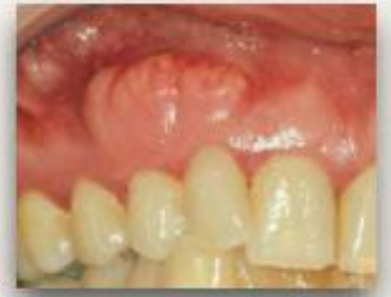
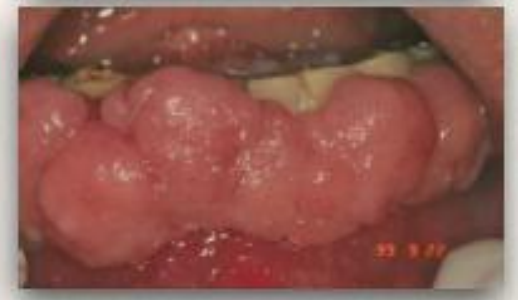




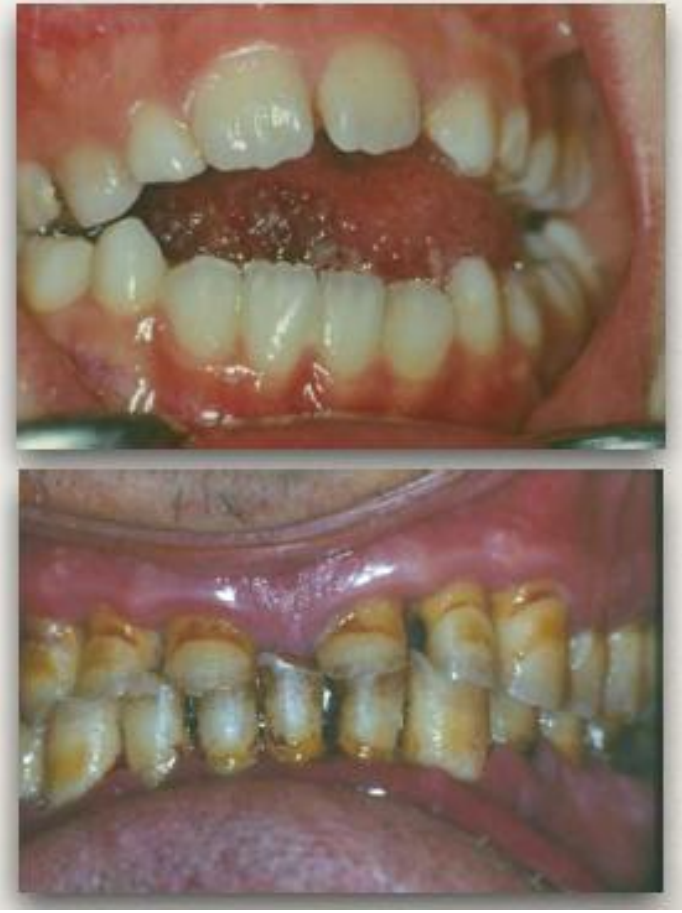
- gingival excess: pseudopocket, inconsistent gingival margin, gingival enlargement
- abnormal gingival color

Developmental or acquired deformities and conditions

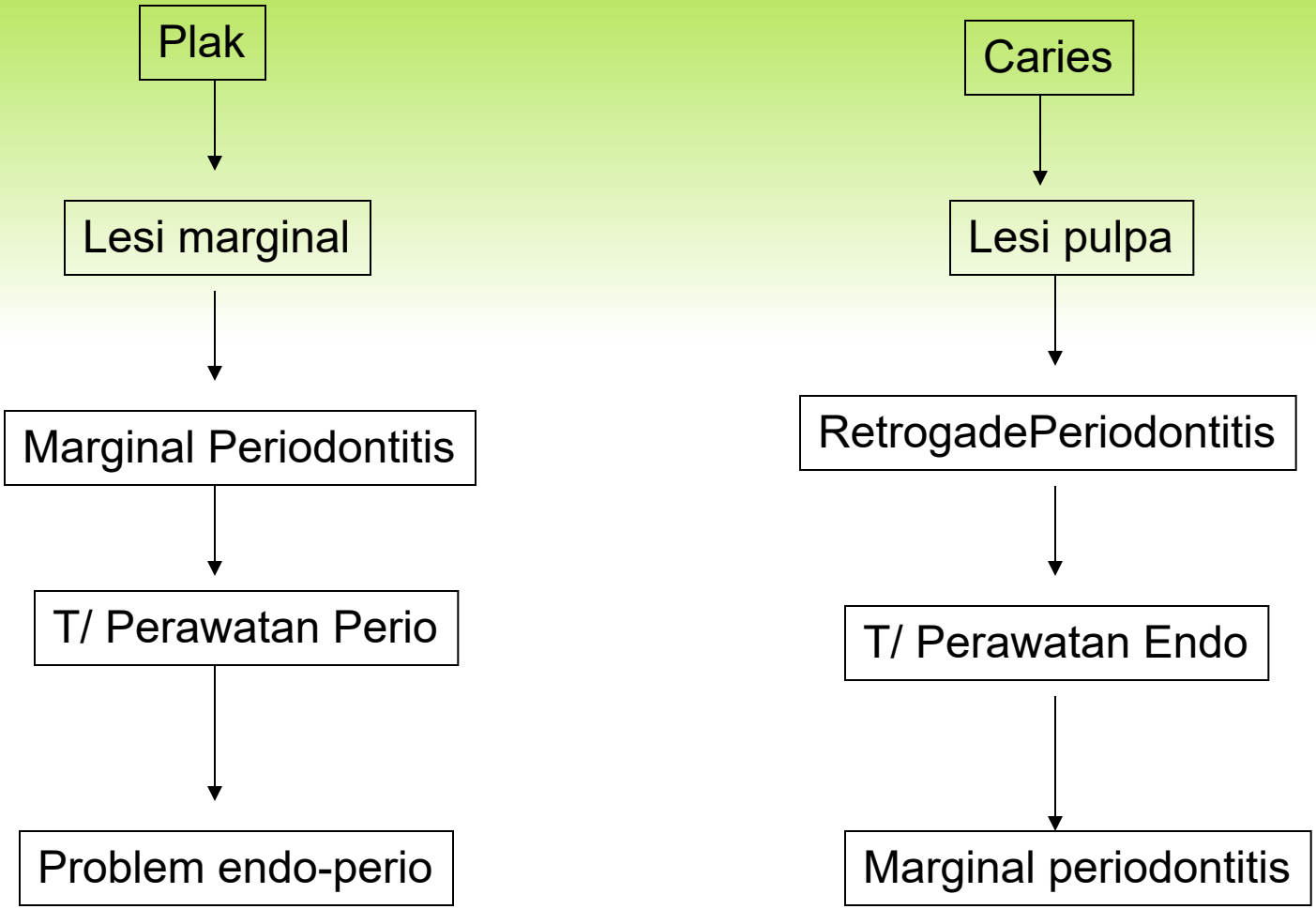
- Occlusal trauma

- primary occlusal trauma - a tissue reaction around a tooth with normal height of the periodontium

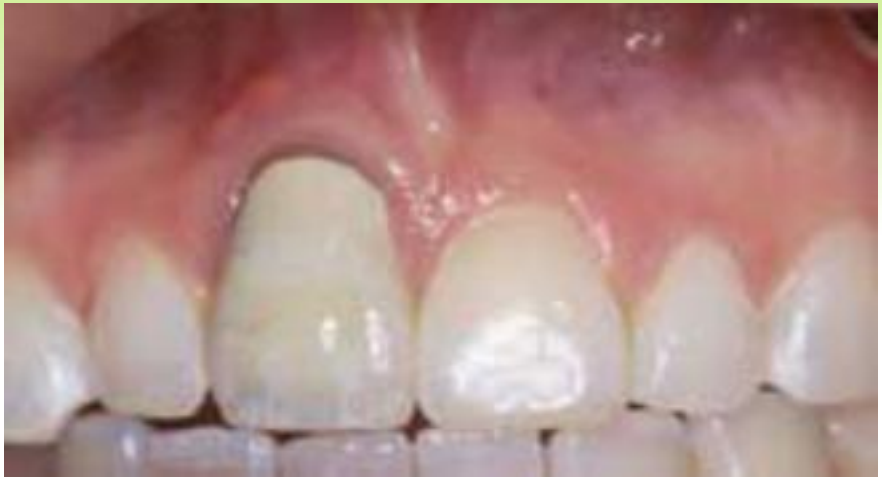
- secondary occlusal trauma - occlusal forces cause injury in a periodontium of reduced height



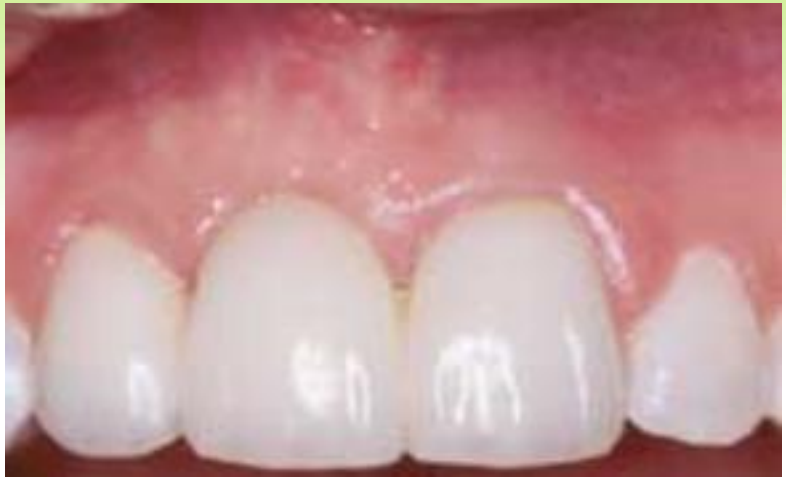
PATHOGENESIS PENYAKIT PERIODONTAL



Perawatan Konser-Perio



Sebelum perawatan



Sesudah perawatan