Periodontal diseases II

Department of oral pathology

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Learning Objectives-

At the end of lecture student should able to describe--

Definition,

Types,

Clinical features of-

Gingivitis, Necrotizing ulcerative gingivitis, Plasma cell gingivitis, Granulomtous gingivitis, Gingival fibromatosis.

- Difference between all types

Gingivitis

- Refers to the inflammation of the soft tissues that surround the teeth

Compounding Factors for Gingivitis

- Lack of proper hygiene
- Periods of susceptibility (puberty, pregnancy (progesterone level), menopause)
- Smoking
- Diet (poor nutrition)
- Medications
- Diabetes mellitus
- Metal poisoning
- Trauma/mastication injury
- Tooth crowding/overlap
- Mouth breathing

- Types include:
  - Plaque-related gingivitis
  - Necrotizing ulcerative gingivitis
  - Medication-influenced gingivitis
  - Allergic gingivitis (plasma cell gingivitis)
  - Specific infection-related gingivitis
  - Dermatosis-related gingivitis

Clinical Patterns of Gingivitis

- Localized/generalized
- Marginal/papillary

Necrotizing Ulcerative Gingivitis

The condition was studied by French physician Hyacinthe Vincent (1890s) and is sometimes referred to as Vincent infection

- Etiopathogenenically associated with *Fusobacterium nucleatum* and *Borrelia vincentii;* recent evidence that *Treponema ssp., Selenomonas ssp., & Prevotella intermedia* may play a role

- Common in solders during WWI where poor conditions prevailed (trench warfare) became known as trench mouth

Etiology

Fusiform bacillus & Borrelia vincentii a spirochete

Predisposing Factors(NUG)

- Psychologic stress
- Immunosuppression
- Smoking
- Poor nutritional status
- Poor oral hygiene
- Sleep deprivation
- Recent illness (e.g., URI or flu)
- Decreased resistance to infection

Clinical Features

- Young to middle aged adults
- Whites > blacks; males > females
- Smoker > non-smokers

- Interdental papillae are painful,inflamed, edematous and hemorrhagic
- Papillae are blunted and like “punched- out” craterlike erosions & covered by necrotic gray pseudomembrane;
- Fetid odor & pain; sometimes lymphadenopathy, fever and malaise
- Excessive salivation & metallic taste
- Incubation zones

Treatment

- Debridement, improved home care, rinses with chlorhexidine, diluted hydrogen peroxide and saline; scaling &polishing,sometimes antibiotics (metronidazole, tetracycline, penicillin); address cofactors such as poor nutrition & hygiene, smoking & stress

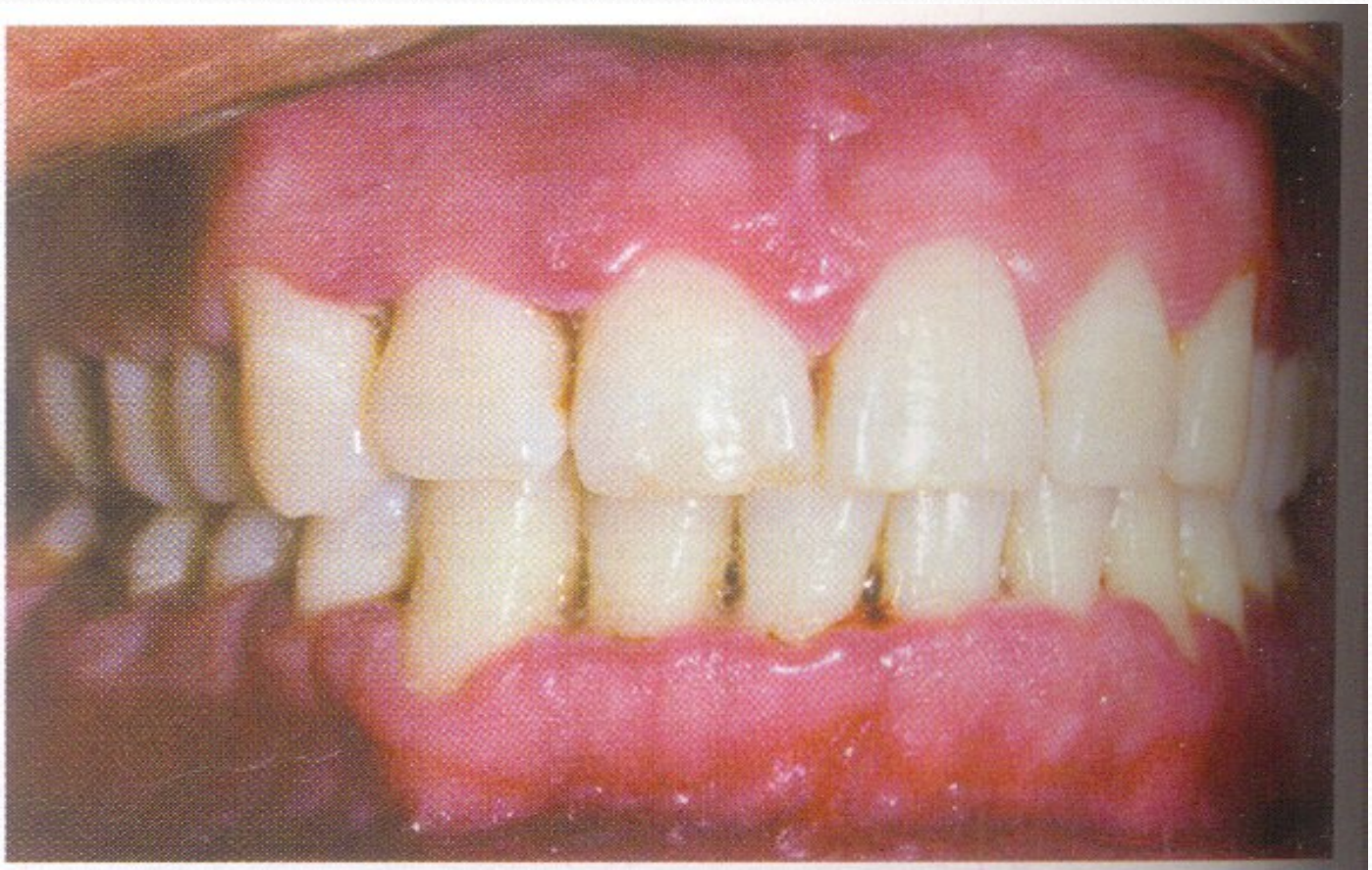


Figure 4-8 • Necrotizing ulcerative gingivitis. Gingiva demonstrates blunted interdental papillae that exhibit early mucosal necrosis.

Plasma Cell Gingivitis

- Usually a form of “allergic gingivitis” associated with a
  specific allergen

- First reported in persons that chewed Wrigley’s gum during

  the Vietnam war era (Wrigley Company changed formulation)

H/P

Hyperplastic epithelium,with intracellular edema& microabscesses

Dense plasmacytic infiltration & vascular dilatation

T/t

Identification and removal of allergen; topical &systemic steroids-

good results

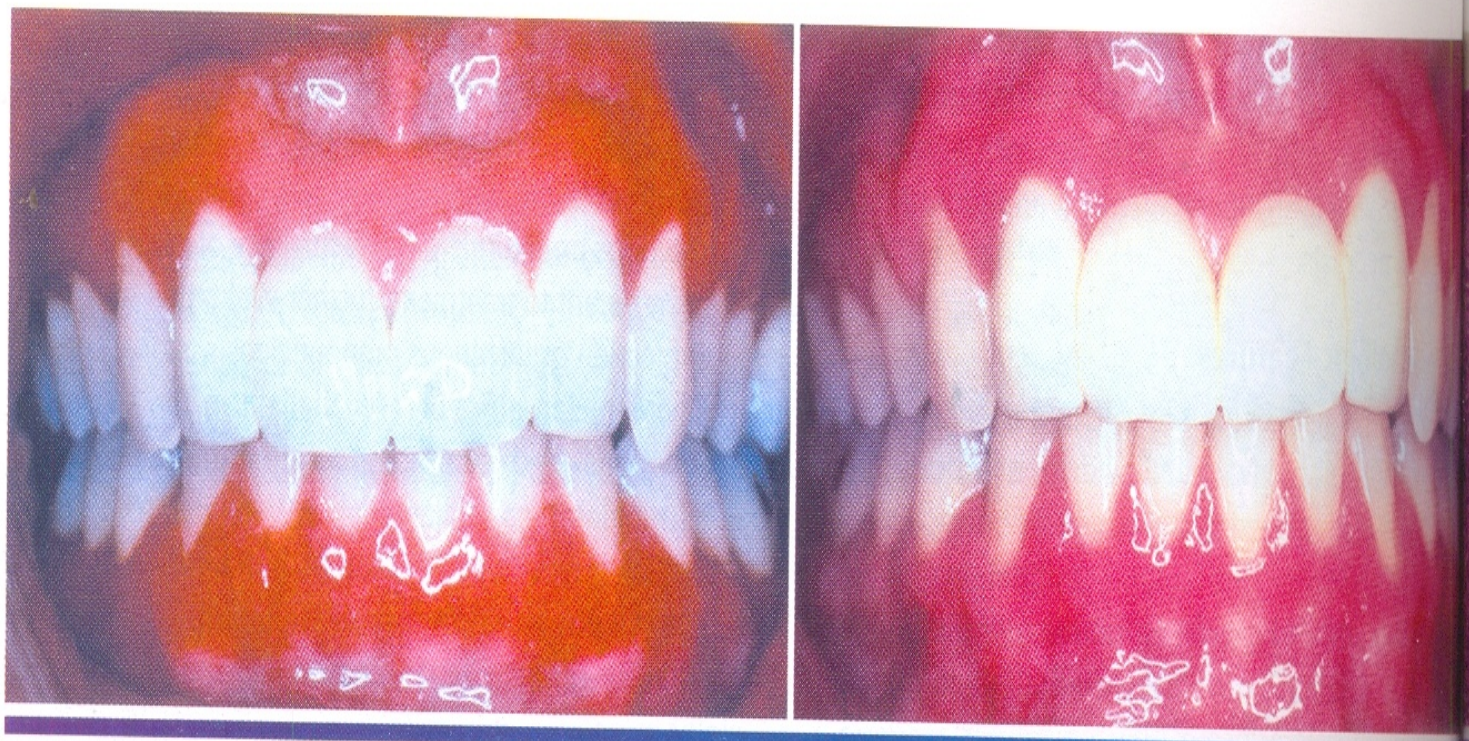


Figure 4-10 ♦ Plasma cell gingivitis. A, Diffuse, bright-red enlargement of the free and attached gingiva. **B,** Same patient as depicted in *A* after elimination of the inciting allergen.

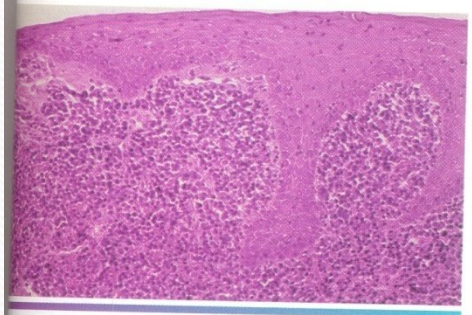
Granulomatous Gingivitis

- Defined by the presence of granulomatous inflammation in the gingiva; most often in adult patients; solitary or multifocal red or red/white macules in the interdental papillae < 2mm diameter.

- Must search for cause; foreign material, fungal infection, acid-fast bacterial infection, Crohn's disease, sarcoidosis, chronic granulomatous disease, Wegener's granulomatosis

- Most cases are related to foreign material (“foreign-body gingivitis”) but can produce lichenoid reaction rather or mixed with than foreign-body reaction

- Surgical removal of affected tissues



ure 4-11 • **Plasma cell gingivitis.** Hyperplastic squamous ithelium exhibiting exocytosis and dense plasmacytic infiltrate the underlying lamina propria.

Energy-dispersive radiographic microanalysis revealed e most frequently discovered materials were silicon, uminum, iron, and titanium. Less frequently discovered aterials included nickel, silver, chromium, zinc, copper, , manganese, zirconium, gold, and mercury. Rarely, ndental foreign material was detected.

linical Features

th foreign-body gingivitis and nonspecific granulo- atous gingivitis may occur at any age; however, they e most frequently encountered in adulthood. The sions may be solitary or multifocal, typically with a ameter less than 2 cm (Figure 4-12). The affected areas pear as red or red-and-white macules, which most fre- ently involve the interdental papillae. Extension may cur along the marginal gingiva or onto the attached giva (Figure 4-13). Pain or sensitivity is a common ding, and the lesions persist despite conventional rapy and rigorous oral hygiene. The process can be n adjacent to clinically normal teeth or next to teeth h restorations.

stopathologic Features

iopsy specimen of granulomatous gingivitis demon- ates focal collections of histiocytes intermixed with an nse lymphocytic infiltrate (Figure 4-14). On occa- n, well-formed histiocytic granulomas with multinu- ated giant cells are seen. Special stains for organisms uld be negative. If foreign material is detected, the cli- ian can render a diagnosis of a foreign-body reaction her than the more nonspecific term, granulomatous givitis). In some cases, however, the foreign material y be too fine to be detected.

n almost half of the reported cases of foreign-body givitis, granulomatous inflammation has not been sent. Instead, a lichen planus-like reaction has been

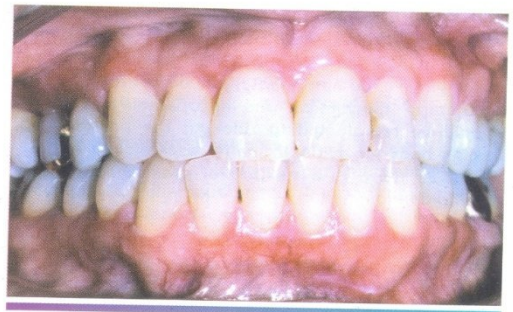


Figure 4-12 • **Foreign-body gingivitis.** Patient developed multifocal areas of tender and erythematous gingiva that arose shortly after periodontal curettage and coronal polishing.

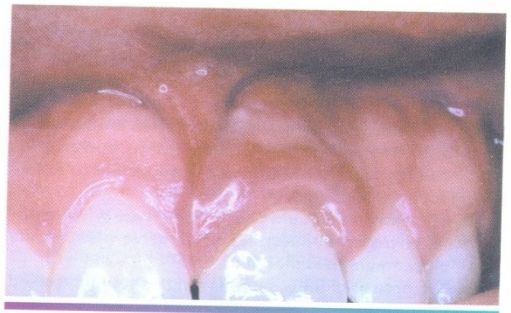


Figure 4-13 • **Granulomatous gingivitis.** Localized enlarged and erythematous gingiva associated with the maxillary left central incisor. The alterations developed shortly after placement of a porcelain-fused-to-metal full crown and were not responsive to conservative local therapy. (Courtesy of Dr. Timothy L. Gutierrez.)

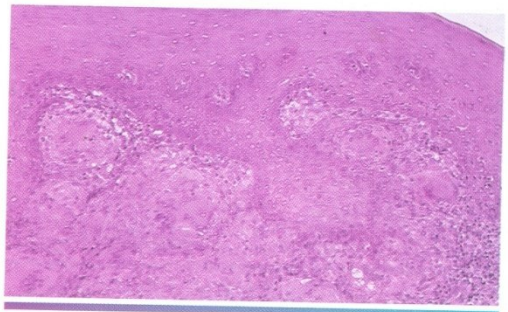


Figure 4-14 • **Granulomatous gingivitis.** Focal collection of histiocytes, lymphocytes, and multinucleated giant cells within the superficial lamina propria of the gingiva.

Desquamative Gingivitis

- Clinical term-intense redness & the epithelial cover of the gingiva sloughs away

Etiology

Based on etiology—

- Dermatoses
- Hormones
- Irritation
- Chronic infection
- Idiopathic

- Most often dermatoses--the result of cicatricial pemphigoid or lichen planus; occasionally pemphigus vulgaris, linear IgA disease, epidermolysis bullosa acquista, systemic lupus erythematosus, chronic ulcerative stomatitis or paraneoplastic pemphigus

- Some cases might be related to estrogen levels or abnormal immune response to plaque substances

- Biopsy, often supplemented by immunofluorescent testing for definitive diagnosis

Desquamative Gingivitis

- Most often in women (4x males) and usually after 40 year of age
- Gradual onset, limited area of gingiva that “spreads”; facial aspect more than lingual
- Smooth erythema, loss of stippling, blistering & desquamation, significant pain; Nikolsky sign
- Definitive therapy depends on histopathologic and immunologic diagnoses; often combination of scrupulous oral hygiene, antimicrobials and immunosuppressants

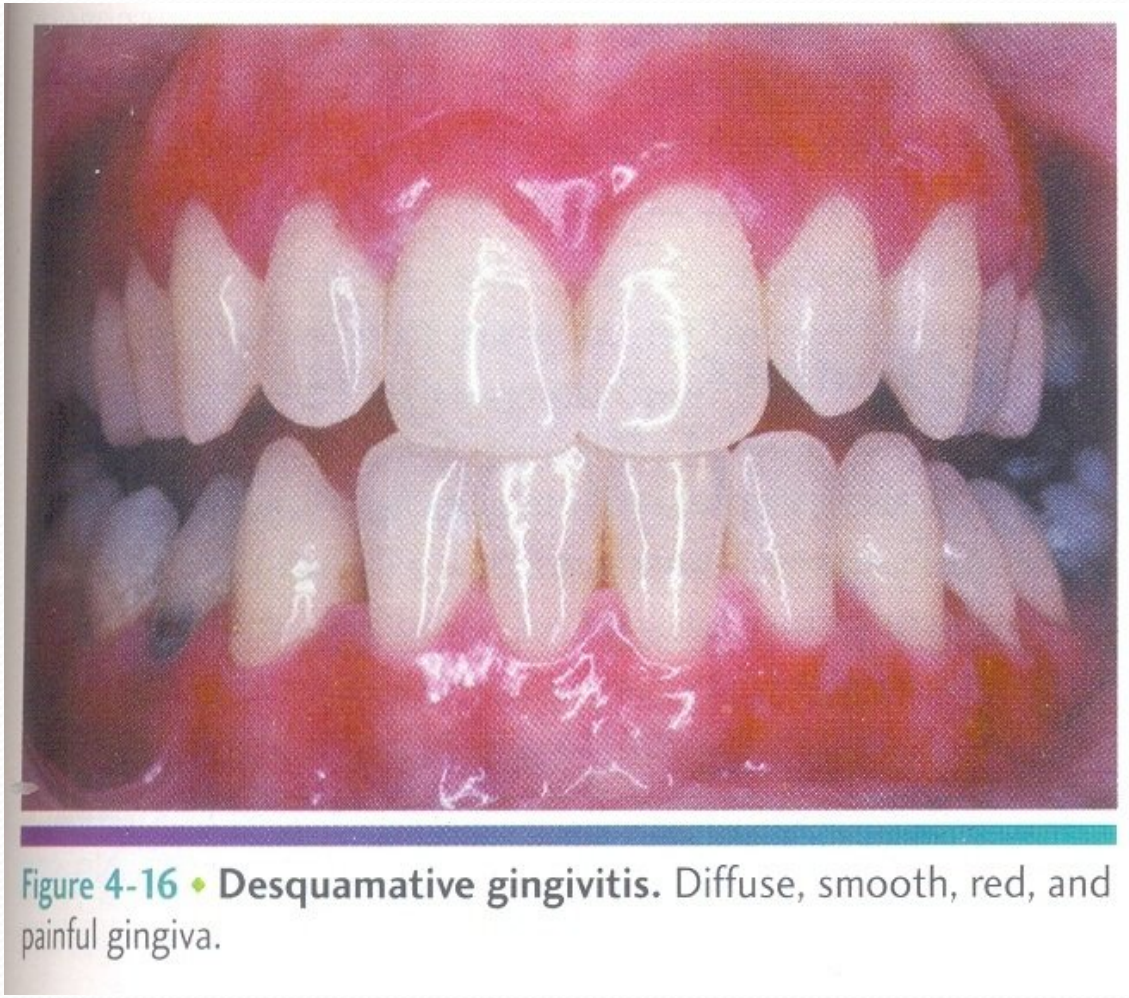


Figure 4-16 • **Desquamative gingivitis.** Diffuse, smooth, red, and painful gingiva.

Drug-Related Gingival Hyperplasia

- Due to excess collagen formation in the gingiva (fibrous enlargement) secondary to the use of a systemic medication
- Strong association with cyclosporine (transplant rejection suppressant), Dilantin (phenytoin--anticonvulsant) and nefedipine (calcium channel blocker)
- Degree of enlargement related to patient's susceptibility and level of oral hygiene

- Enlargement begins 1-2 months of drug use, first in the anterior facial segment; the interdental papillae enlarges and spreads across facial surface; later lingual and posterior areas; pseudopockets leads to inflammatory changes

- Treatment may involve removal of offending medication (by the physician), substitution of drug of different class, use of folic acid therapy, metronidazole or azithromycin, and or gingivectomy & gingivoplasty

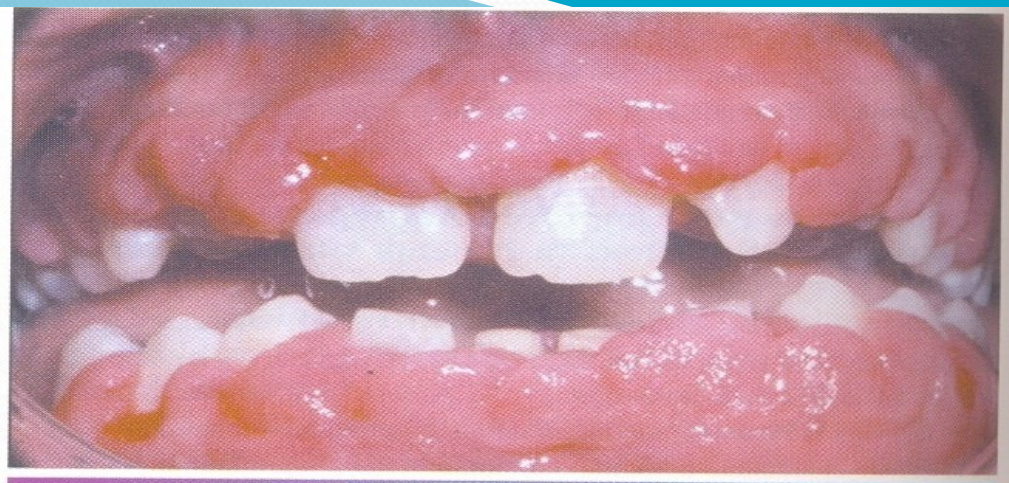


Figure 4-17 • Cyclosporine-related gingival hyperplasia. Diffuse, erythematous, and fibrotic gingival hyperplasia.

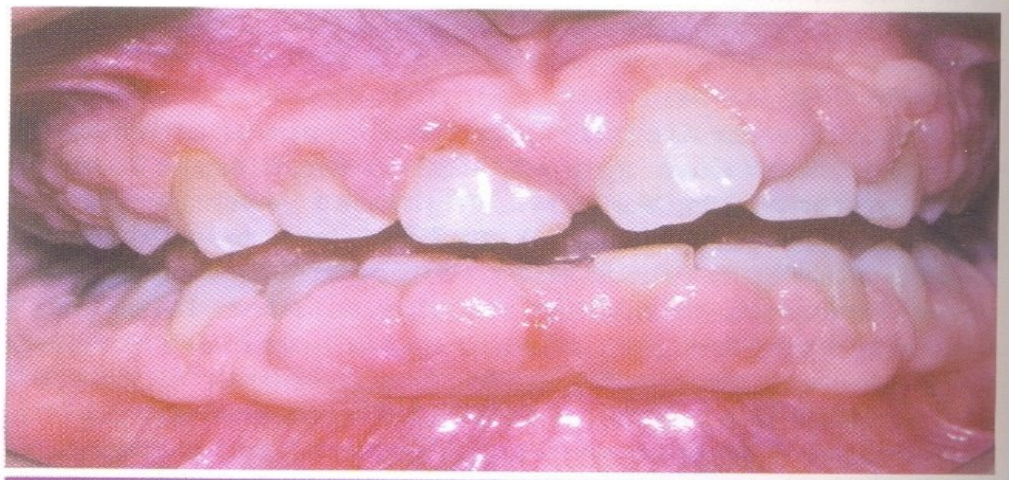


Figure 4-18 • Nifedipine-related gingival hyperplasia. Diffuse, fibrotic gingival hyperplasia after 1 month of intensive oral hygiene. Significant erythema, edema, and increased enlargement were present before intervention.

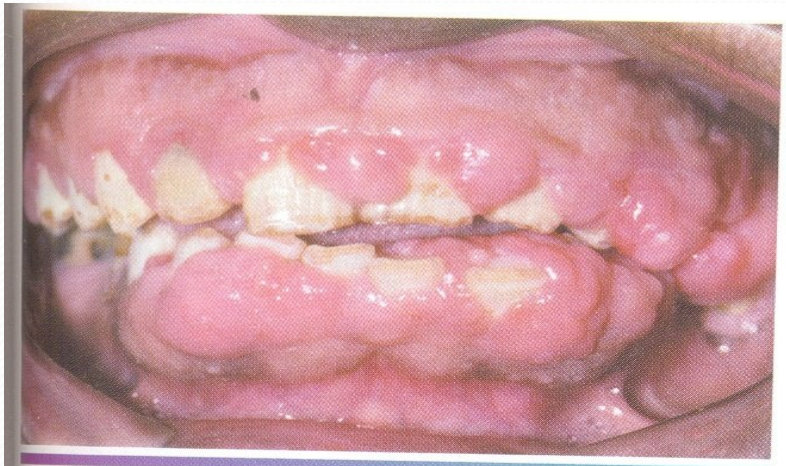


Figure 4-19 • **Cyclosporine- and nifedipine-related gingival hyperplasia.** Dramatic gingival hyperplasia in a patient using two drugs associated with gingival enlargement.

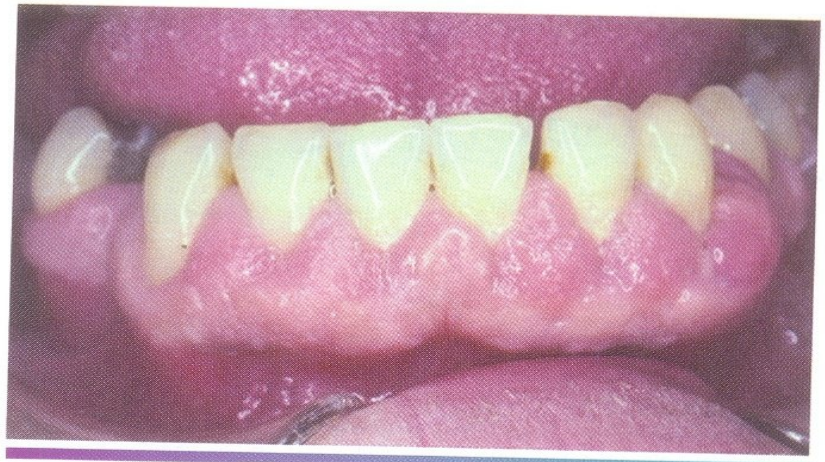


Figure 4-20 • **Mild phenytoin-related gingival hyperplasia.** Gingival enlargement present predominantly in the interdental papillae.

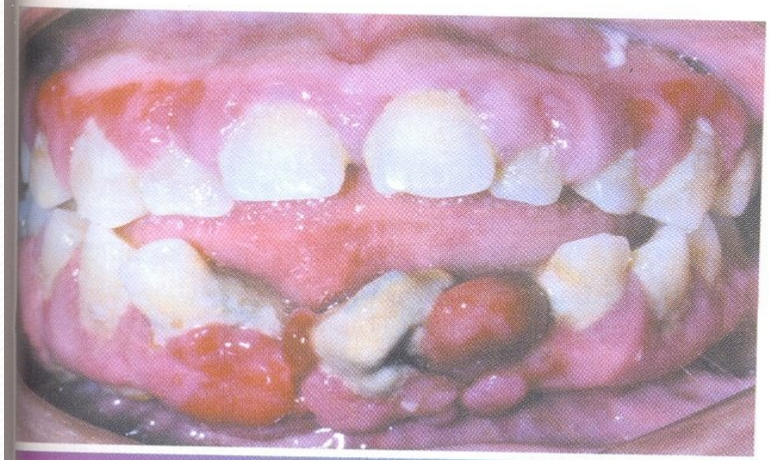


Figure 4-21 • **Phenytoin-related gingival hyperplasia.** Significant erythematous gingival hyperplasia is covering portions of the crowns of numerous teeth.

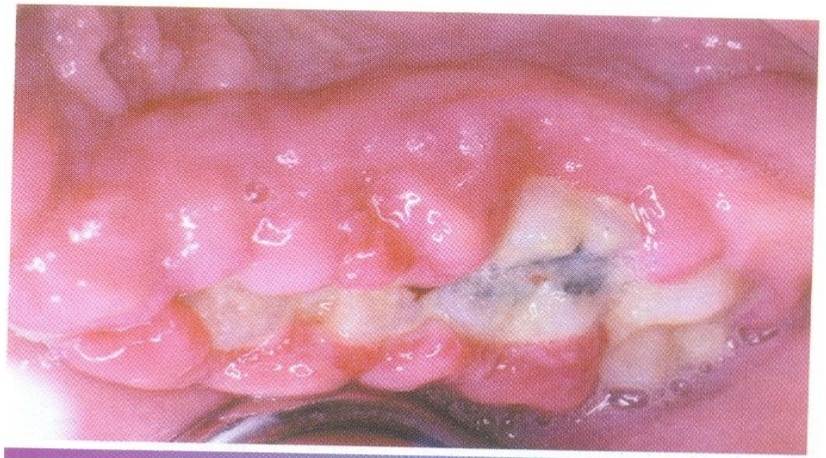


Figure 4-22 • **Phenytoin-related gingival hyperplasia.** Significant gingival hyperplasia almost totally covers the crowns of the posterior maxillary dentition. (Courtesy of Dr. Ann Drummond and Dr. Timothy Johnson.)

Conditioned enlargement

1.Hormonal

2.Nuritional

3.Allergic

Enlargement due to systemic diseases

Leukemia

Granulomatous diseases

Regional enteritis

Gingival Fibromatosis

- A familial or idiopathic, slowly progressive gingival enlargement caused by an overproduction of collagen produced by the fibrous connective tissue of the gingiva
- Sometimes seen in conjunction with hypertrichosis, epilepsy, mental retardation, sensorineural deafness, hypothyroidism, chondrodysthropia and GH deficiency (or a few rare syndromes)

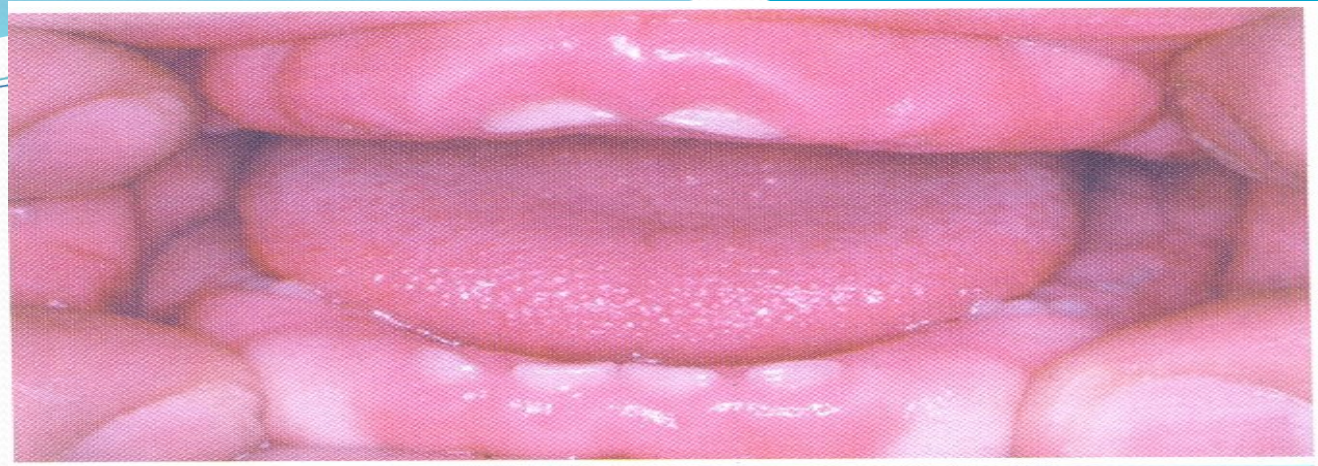


Figure 4-26 • Gingival fibromatosis. A young child with cheeks retracted by the parent. Note erythematous gingival hyperplasia arising in association with erupting deciduous dentition. (Courtesy of Dr. George Blozis.)

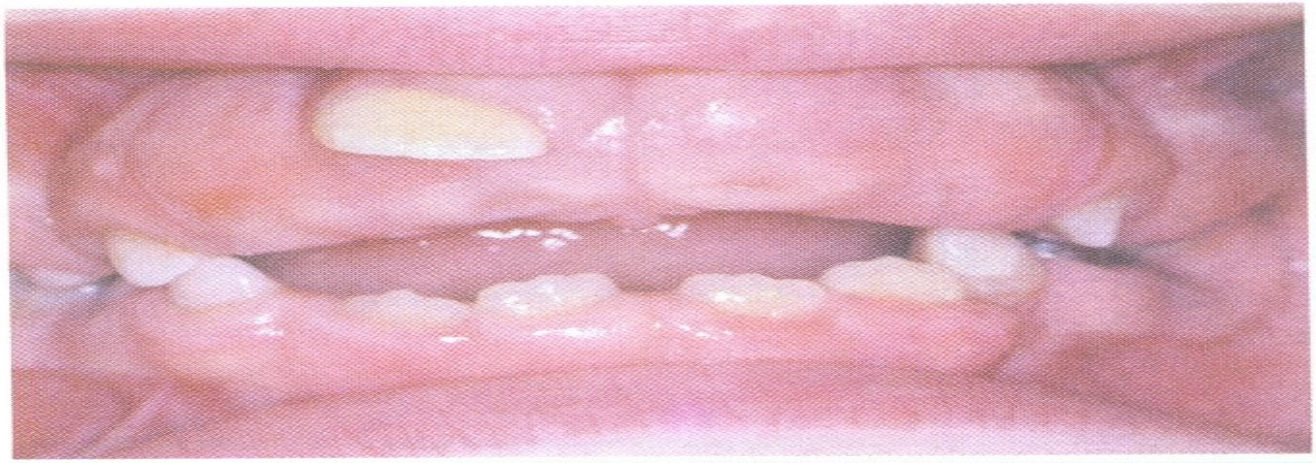


Figure 4-27 • Gingival fibromatosis. Significant fibrotic gingival hyperplasia with resultant delayed eruption of numerous teeth. (From Neville BW, Damm DD, White DK, Waldron CA: *Color atlas of clinical oral pathology,* Philadelphia, 1991, Lea & Febiger.)

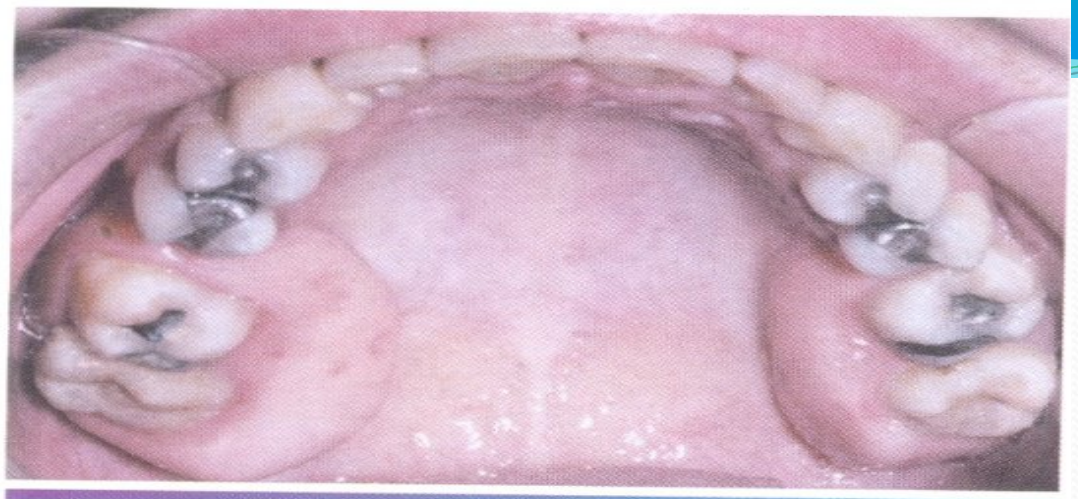


Figure 4-28 • **Localized gingival fibromatosis.** Bilateral and symmetric fibrotic enlargements of the palatal surfaces of the posterior maxillary alveolar ridges.

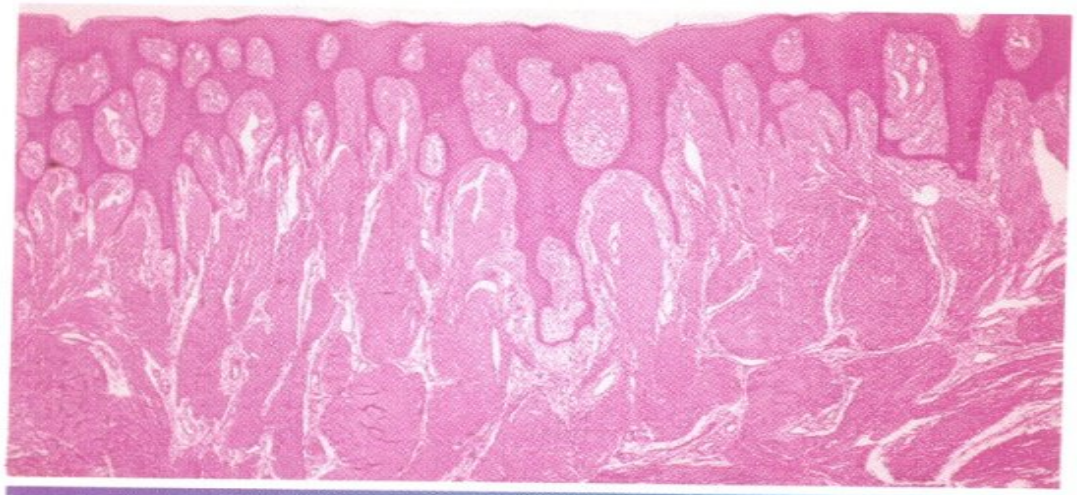


Figure 4-29 • **Gingival fibromatosis.** Surface stratified squamous epithelium exhibiting long, thin rete ridges and underlying dense, fibrous connective tissue.

The frenular attachments may appear to divide the

Summary-

Definition, Types, Clinical features of-

Gingivitis, Necrotizing ulcerative gingivitis, Plasma cell gingivitis, Granulomtous gingivitis, Gingival fibromatosis.

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Thank you