Physical and Chemical Injuries of the Oral Mucosa

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Introduction

- The oral mucosa - the first part of the digestive tract, exposed to various exogenous noxes
- Exposition of longer duration → reactive changes, ! diff. dg. x malignancies

- Mucosal lesions with an increased chance of turning into a malignancy - premalignant lesions
- Frequently in areas of the oral cavity not obvious to the patient (sulcus glosso-alveolaris, dorsal and lateral tongue, oral vestibule, retromolar area)
- Therefore thorough intraoral examination twice a year is necessary

If mucosal lesions are evident:

- Try to remove local factors that could have contributed to the lesion
- Anti-inflammatory treatment for two weeks, if lesion remains: biopsy
- Diagnosis based on clinical appearance alone usually not sufficient to determine the histological nature of the tissue

Terminology

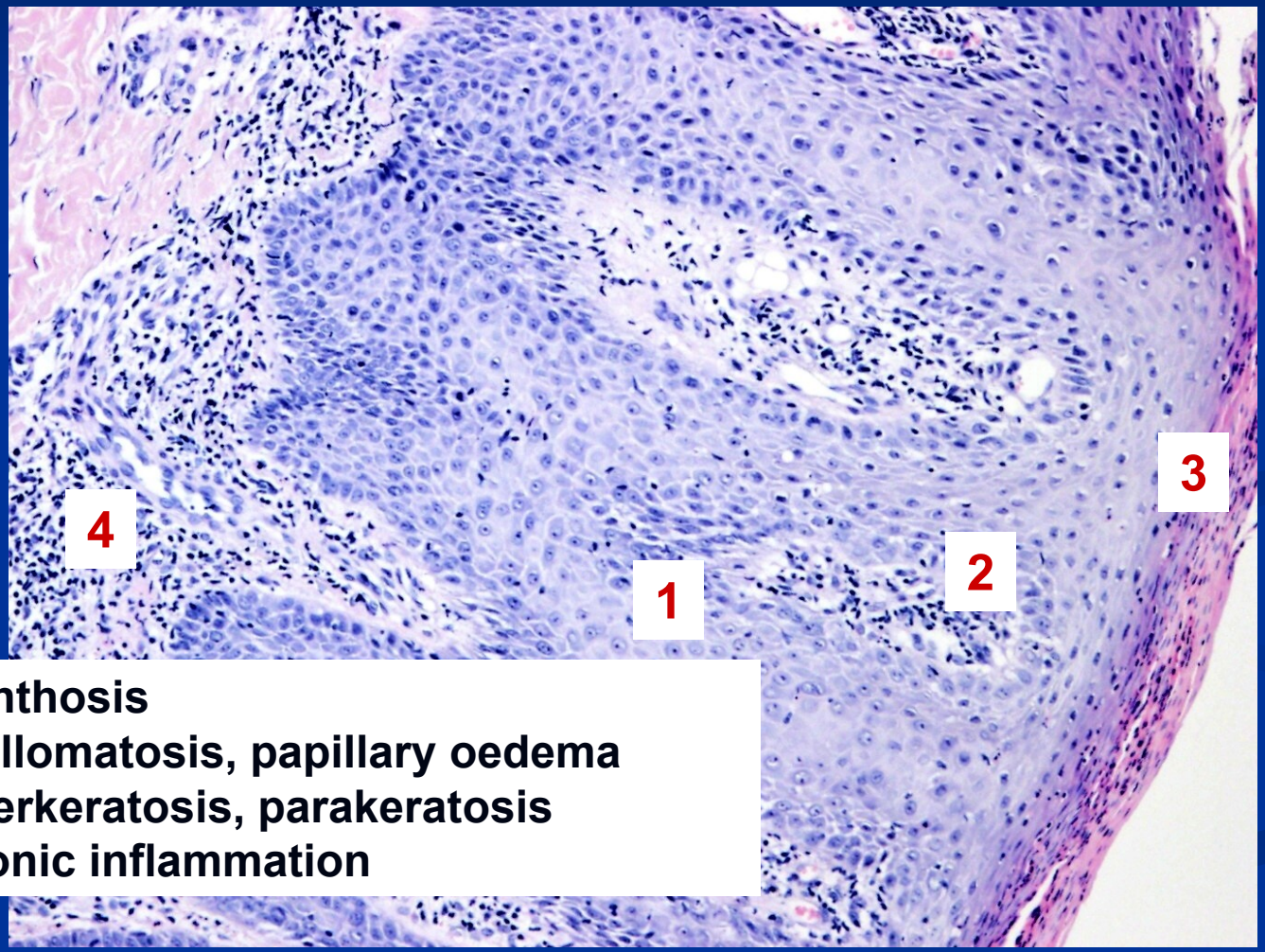
- **Lesion** – focus of abnormal finding (tissue) in the oral cavity: wounds, sores, any other tissue damage caused by injury or disease.
- Determining **the type of lesion** important for differential diagnosis.
- Appearance: **architecture**
  - *below* or *above* the surface.
  - *flat* or *even* with the surface.

  Color: white, red, white + red, pigmented

Epithelial changes: terms

- Acanthosis: excessively thickened intermediate cell layer with broad and long rete pegs
- Hyperkeratosis: excessively thickened keratin in stratum corneum
- Leucoplakia: a white patch on the oral mucosa that cannot be scraped off and cannot be classified as any other disease

Epithelial changes



1 Acanthosis
2 Papillomatosis, papillary oedema
3 Hyperkeratosis, parakeratosis
4 Chronic inflammation

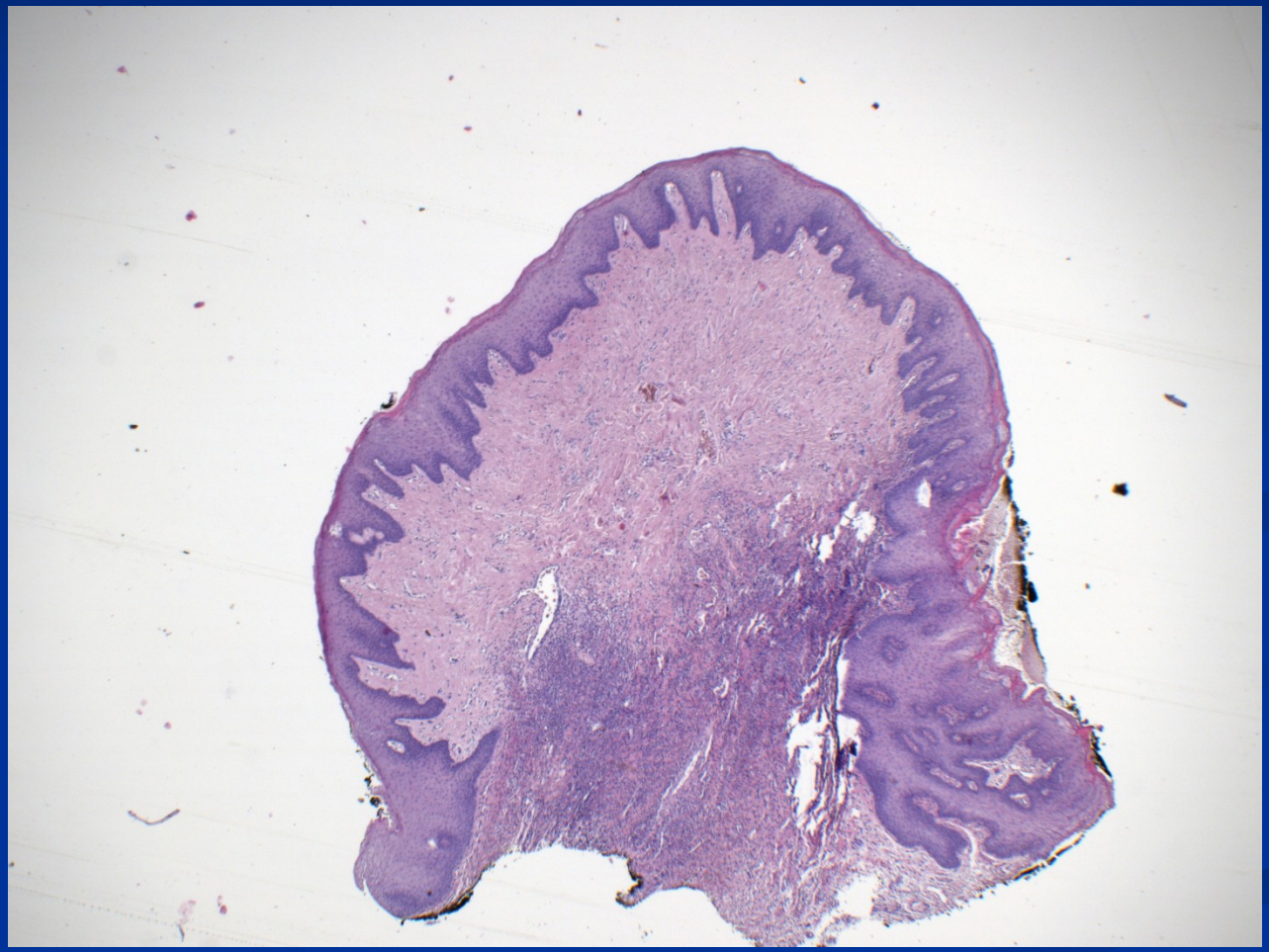
Lesions extending below the surface

- **Erosion:** shallow defect in the mucosa, commonly caused by mechanical trauma, healing by regeneration.
- **Ulcer:** crater-like defect of the mucosa, deeper than erosion, healing by repair
- **Abscess:** localized collection of pus in an area circumscribed by remaining tissue/granulation tissue.
- **Cyst:** pathological cavity lined with epithelium/endothelium/mesothelium; containing fluid or semisolid material.

Lesions extending above the surface

- **Blisters:** *vesicles*, *bullae*, lesions filled with watery fluid. Vesicobullous lesions.
- **Pustule:** similar in appearance to a blister, contains pus.
- **Hematoma:** similar to a blister, contains blood.
- **Plaque:** any solid patch or flat area slightly raised above the surface.
- **Polyp:** exophytic elevated lesion, superficial epithelium + fibrotic stroma

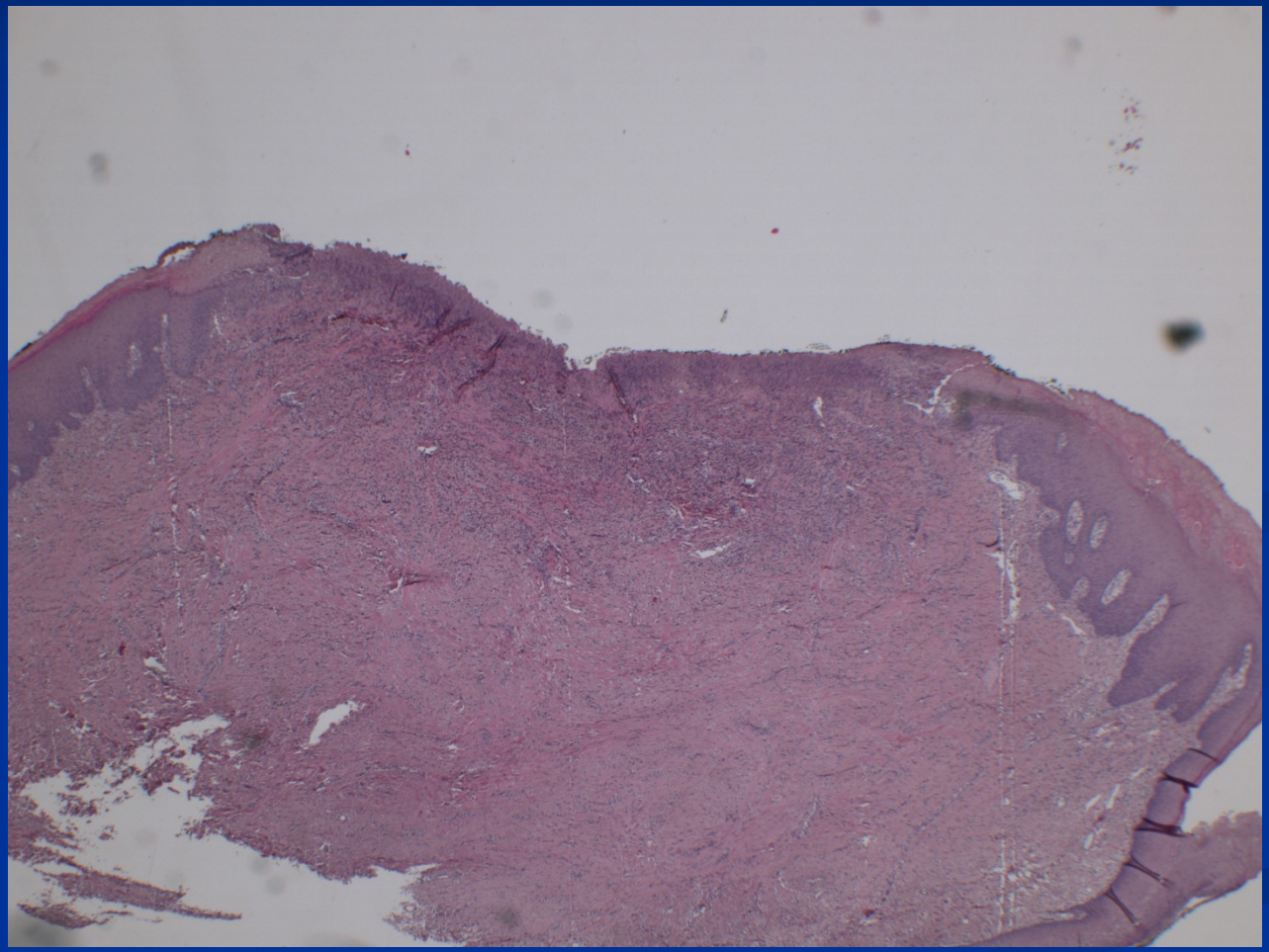
Reactive fibroepithelial polyp



Lesions above + below the surface

- **Nodules** – solid, commonly firm consistency, may be sharply demarcated, may be ulcerated

Reactive nodule + ulceration on the tongue



Etiology

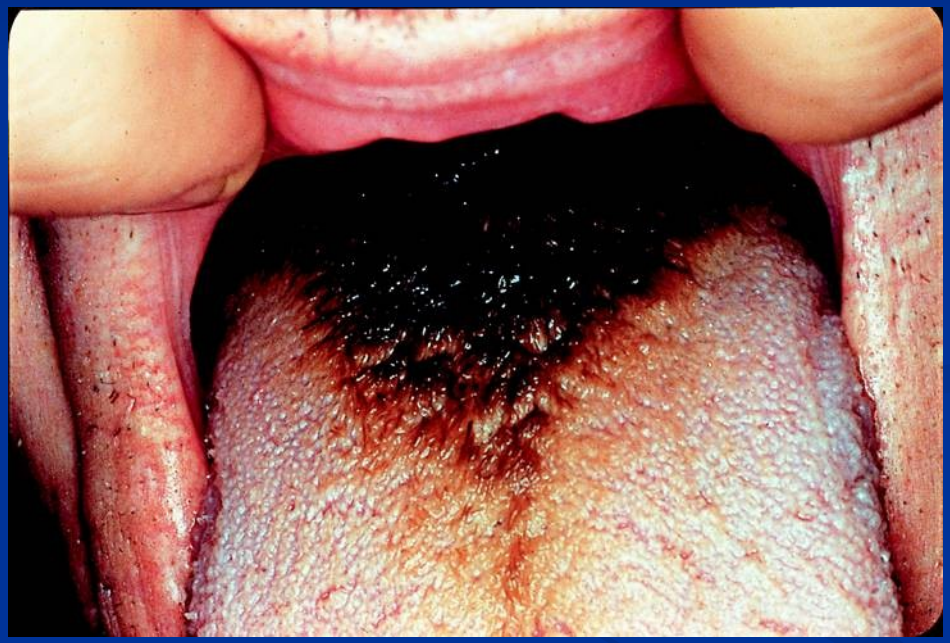
- Physical injuries
- Chemical injuries
- Infection
- Allergies, immunologic diseases
- Hyperplasia, neoplasia
- Inborn lesions
- Combination of multiple factors
- Idiopathic

Physical injuries

- trauma
- denture irritation leading to hyperplasia
- burn
- radiation injury
- amalgam tattoo

Diseases caused by chemical agents

- Aspirin burn
- Nicotine stomatitis
- Snuff lesion
- Hairy tongue
- Gingival hyperplasia



Oral lesions

White lesions:

- Leucoplakia
- Linea alba
- Lichen
- Leucoedema
- Morsicatio buccarum
- White sponge nevus
- Fordyce's granules

Red lesions:

- Erythroplakia
- Varicosity
- Hemangioma
- Purpura (petechiae, ecchymosis) - bleeding
- Hereditary hemorrhagic teleangiectasia

Oral lesions

Red-white lesions:

- Speckled erythroplakia
- Squamous cell carcinoma
- Lichen planus
- Lupus erythematodes
- Lichenoid drug reactions
- Candidiasis (candidal leucoplakia, antibiotic sore mouth, denture stomatitis)

Pigmented lesions:

- Melanoplakia
- Ephelis (freckle)
- Pigmented naevus
- Malignant melanoma
- Peutz-Jeghers syndrome
- Addisons's disease
- Tobacco associated pigmentation (smokers melanosis)
- Amalgam tattoo

Etiology of white oral lesions

- **Inborn**
  - White sponge nevus (AD)
  - Oral manifestation of other inborn/hereditary diseases
  - Fordyce‘s granules (sebaceous glands heterotopy)
- **Traumatic -reactive**
  - mechanical-friction keratosis - hyperplasia due to chronic irritation
  - other physical – thermal burn, nicotinic stomatitis
  - chemical - burns, snuff lesion aj.
- **Infections**
  - Candidiasis (acute pseudomembranous, chronic hyperplastic)
  - Diphteria (pseudomembranous inflammation)
  - Syphilitic leucoplakia (glossitis)
  - Oral hairy leucoplakia (EBV+HIV)

Etiology of white oral lesions

- **Idiopathic leucoplakias**
- **Dermatologic**
  - Lichen planus
  - Lupus erythematodes
- **Neoplastic**
  - Carcinoma *in situ*
  - Squamous cell carcinoma
  - others

Common oral lesions in the primary care office

- White lesions
  - Candida
  - Oral leucoplakia
  - Morsicatio buccarum
  - Hairy tongue
- Vesiculobullous lesions
  - Primary herpetic gingivostomatitis
  - Recurrent herpes
  - Herpangina
  - Hand-foot-and-mouth disease

Common oral lesions in the primary care office

- Ulcers
  - Aphthous ulcers
  - Behçet's syndrome (multiple/major oral aphthous ulcers + genital ulcers + ocular/skin lesions, multisystem immunologically mediated disorder)

White mucosal lesions

Appear white due to obscured visualization of the normal pink appearance (connective tissue vascularity).

Possible causes:

- the presence of a superficial material
- epithelial thickening
- submucosal alteration leading to a decrease in blood vessel density

White mucosal lesions

Focal

- physiologic hyperkeratosis - homogenous appearance, sharp borders
- idiopathic leucoplakia - heterogenous appearance, vague border

Large or diffuse

- nicotinic stomatitis, smokeless tobacco use, actinic keratosis, hairy tongue

White mucosal lesions

Multifocal

- Irregular, commonly tongue: diminished host resistance to infection

  Hyperplastic candidiasis – hairy leucoplakia

- Chronic bilateral buccal mucosa: may be inborn, acquired dermatoses

leucoedema, white sponge nevus, lichen planus

Submucosal change

- White or pale lesions
- Covered by normal epithelium
- Surface smooth and translucent.
- No pain or burning.
- Can not be rubbed off.
- Patient history or the distribution of the lesions important for diagnosis.

Clinical features of lesions caused by epithelial thickening

- Keratin: rough or grainy surface texture when dried with air or a cotton gauze.
- Additional keratin (hyperkeratosis) → opaque appearance
- Attached keratin only - wipe with gauze or scrape with a dull instrument.
- Epithelium intact: no pain, burning, or tenderness

Other superficial material

- ! food remnants, a dense accumulation of plaque: painless, mucosa appears normal.

- White material, soft or friable and rubbing → ! an ulcer or erythematous lesion
- Frequent burning + discomfort sensation.
- Diff. diagnosis: after removal of the white material (white surface coagulum) → defect - the ulcerative lesions category.

Physiological hyperkeratosis

- Thickening due to recurrent friction (callus formation).
- Focal keratosis, focal hyperkeratosis, frictional hyperkeratosis.

Reactive white hyperkeratotic lesions

They do *NOT* rub off

- Linea alba
- Denture acanthosis /papillary hyperplasia of the palate
- Nicotinic stomatitis
- Snuff (Dipper's lesion)
- Chemical burn
- Actinic cheilitis

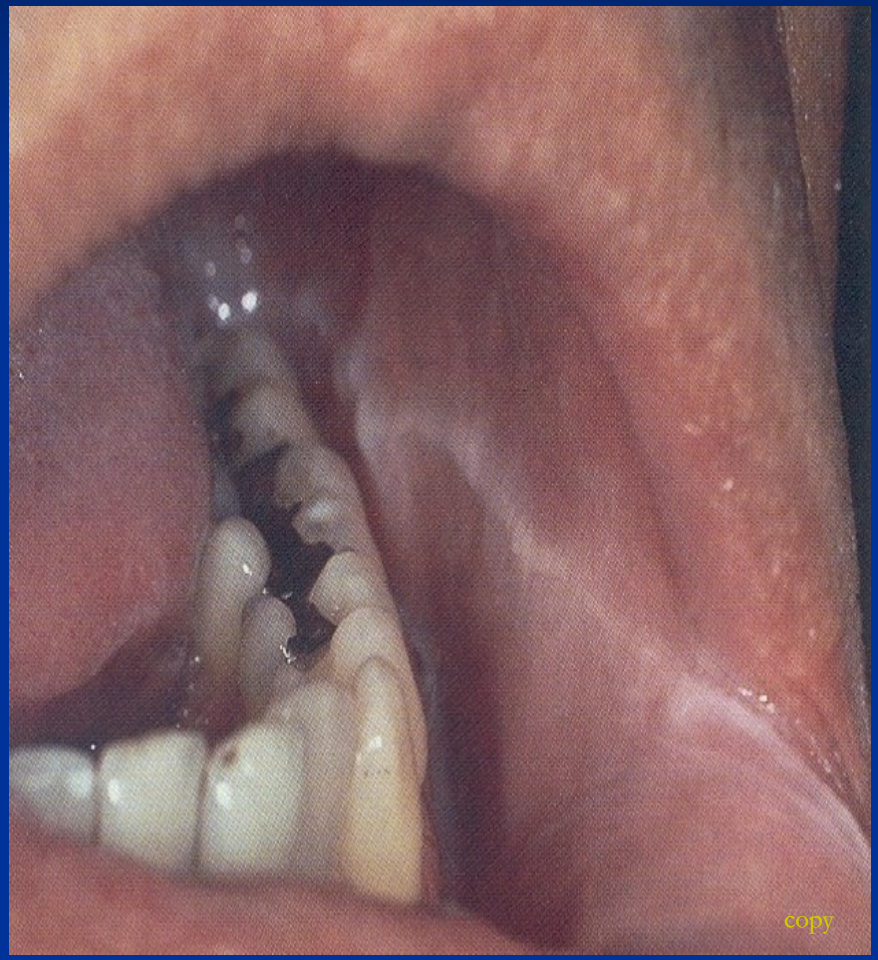
Linea alba

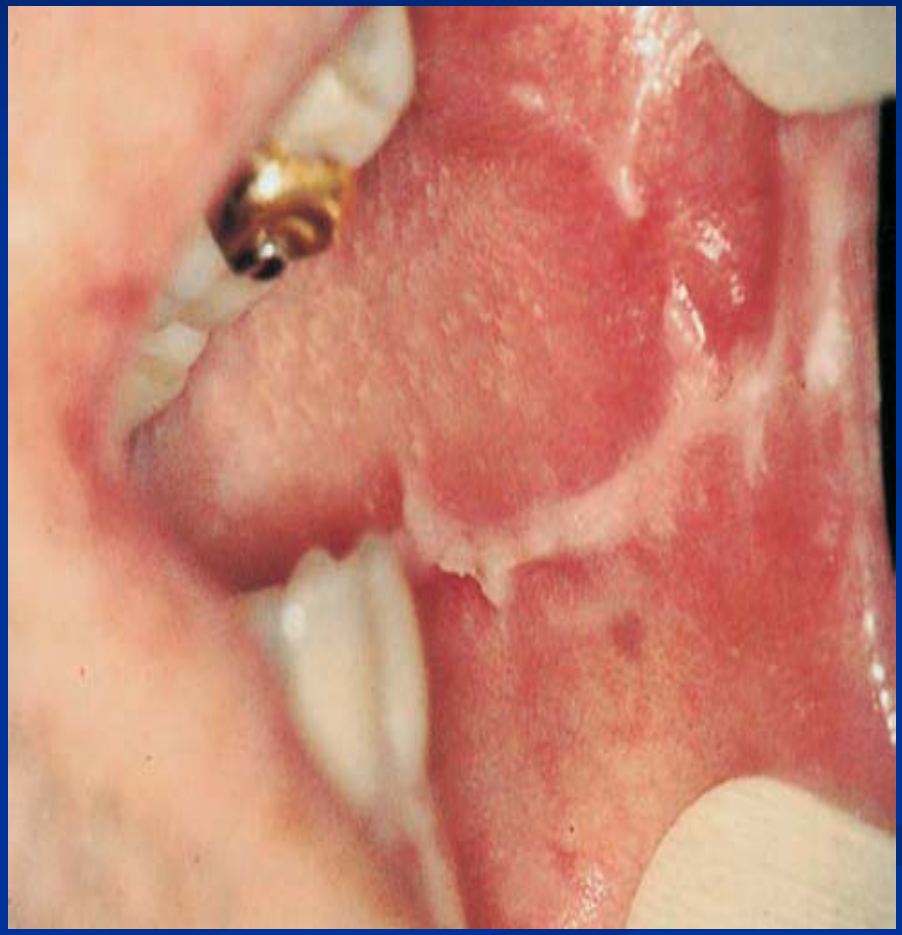
- A white line along the line of occlusion, usually bilateral
- Due to increased formation of keratin as a result of frictional irritation (chronic cheek chewing, grinding),
- Sharp tooth surfaces, appliances, and masticatory function of edentulous ridges
- galvanic irritation

Clinical features

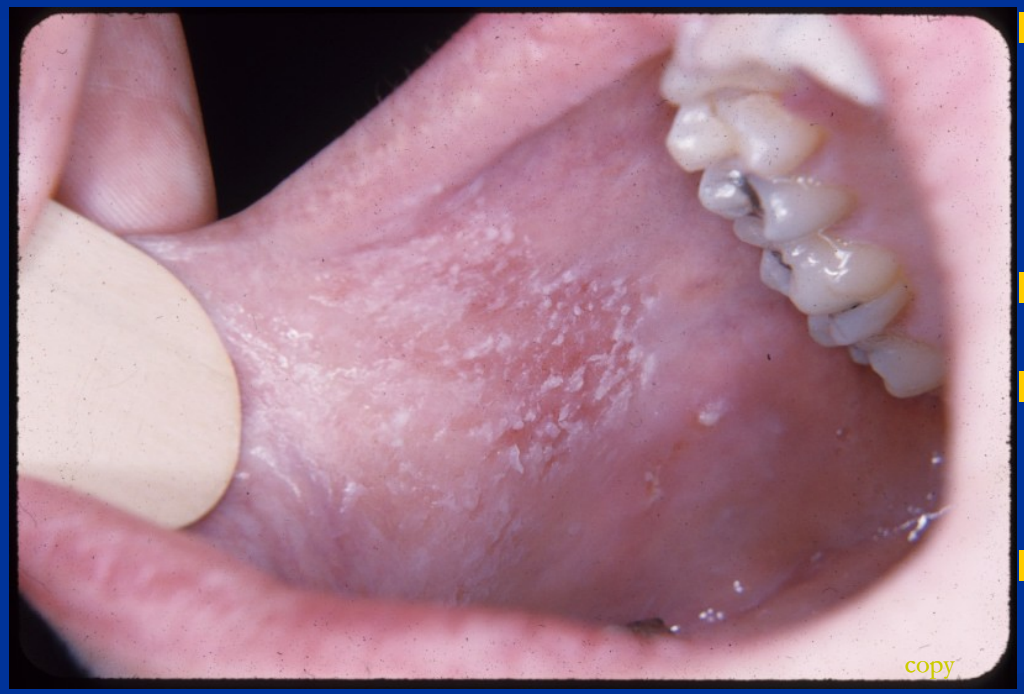
- Opaque, homogenous with sharply delineated borders.
- Usually focal
- Location against the source of friction.
- Asymptomatic, patient unaware.
- If irritation removed→ resolution within a few weeks, no other treatment needed
- Multiple or diffuse - multiple or large sites

Linea alba





Cheek biting (morsicatio buccarum)



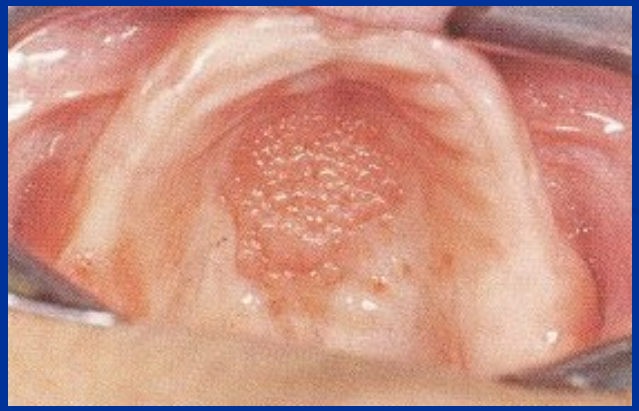
- Irregular whitish focus on the buccal mucosa in the line of occlusion
- May be ulcerated
- Due to chewing or biting the cheeks
- May also be seen on labial mucosa

Differential diagnosis

- Source of friction to the location.
- Resolution.
- Vague borders, focal ulceration, variation of thickening, or heterogeneous color of the white area are more suspicious
- Epithelial dysplasia and early carcinoma: unusual location for cheek biting (soft palate, floor of the mouth, facial vestibule)

Denture irritation

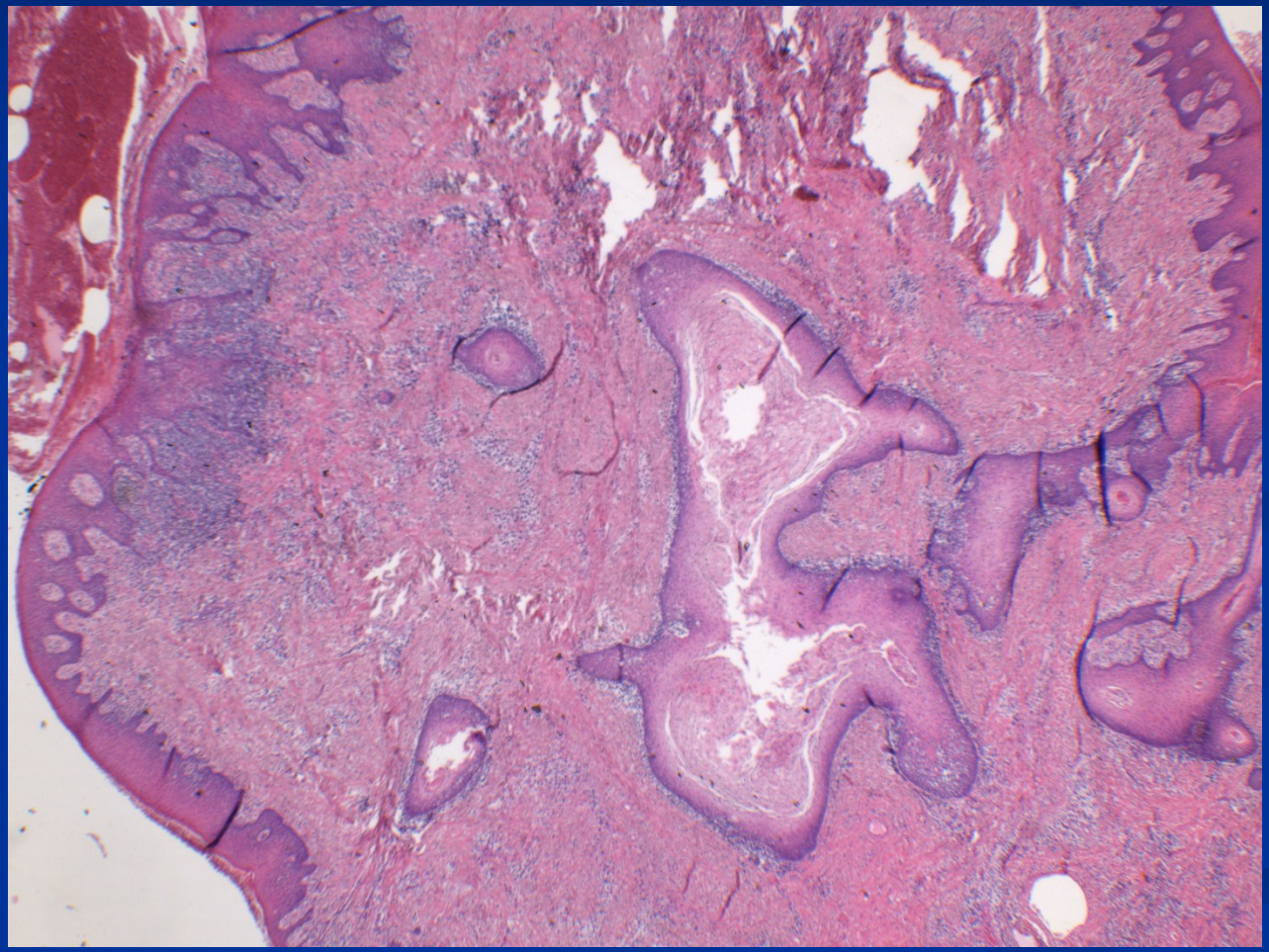
- An ill-fitting denture can cause small ulcers → continued irritation → mucosal hyperplasia in form of acanthosis



Denture acanthosis

- Caused by irritants
- Clinical appearance similar to hyperkeratosis
- Thickened intermediate cell layer
- Elongation of rete pegs
- Treatment: avoid irritants, ie. ill-fitting dentures
- In chronic lesions hyperplasia ev. papillary possible, commonly + chronic candidiasis
- Dif. dg. x true tumors (papilloma, carcinoma)

Papillary hyperplasia of the palate

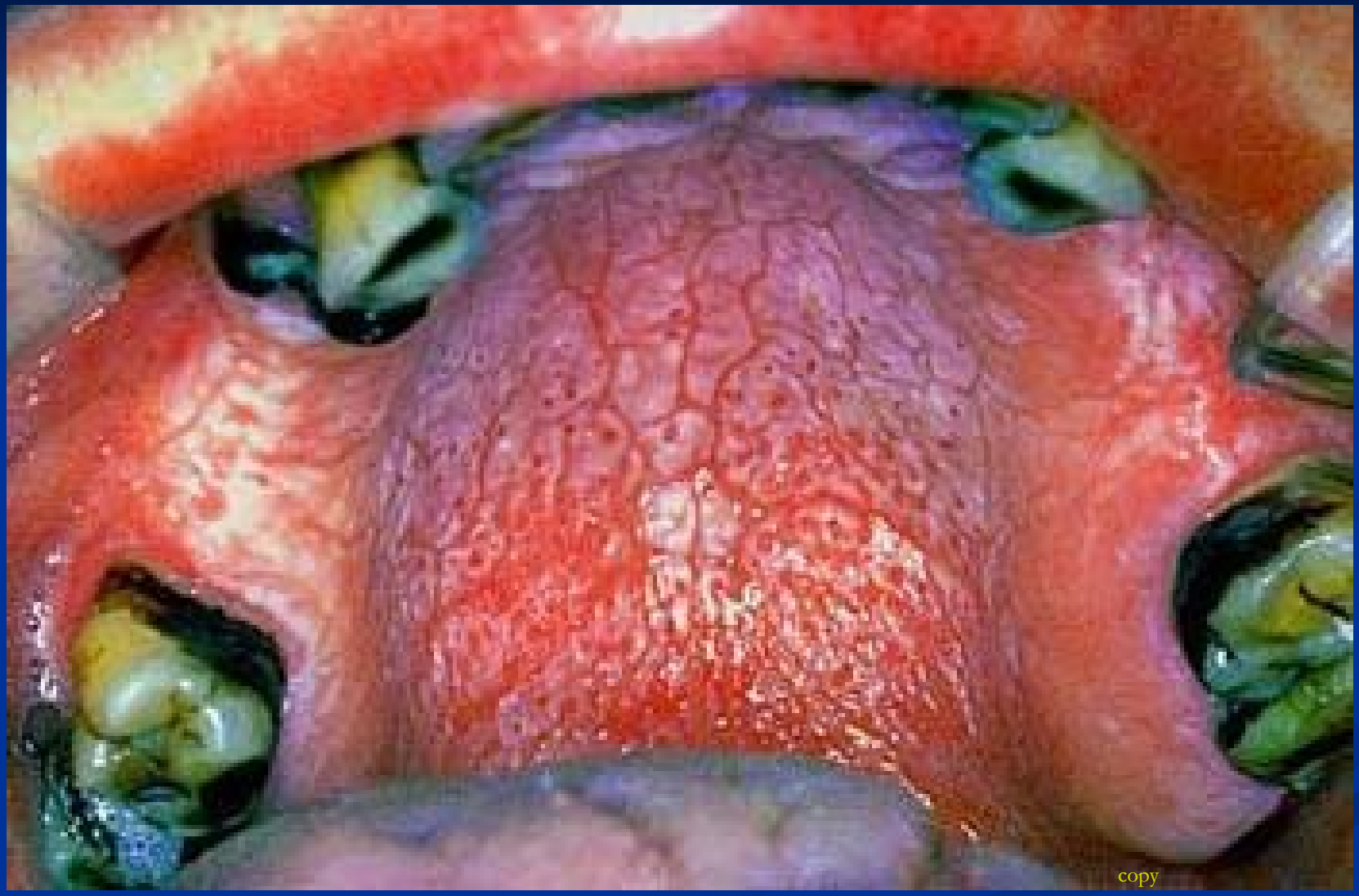


Nicotinic stomatitis

- Physical agent
- „Smokers keratosis" „Smokers patches"
- Response of the palatal tissues to the recurring irritation from tobacco smoke usually from a pipe or cigar habit (heat).
- Hotter smoke of cigars or pipes → more prominent lesions (x cigarettes).
- Possible influence of hot liquids

Nicotinic stomatitis

- Palate initially diffusely erythematous, later grayish white (hyperkeratosis)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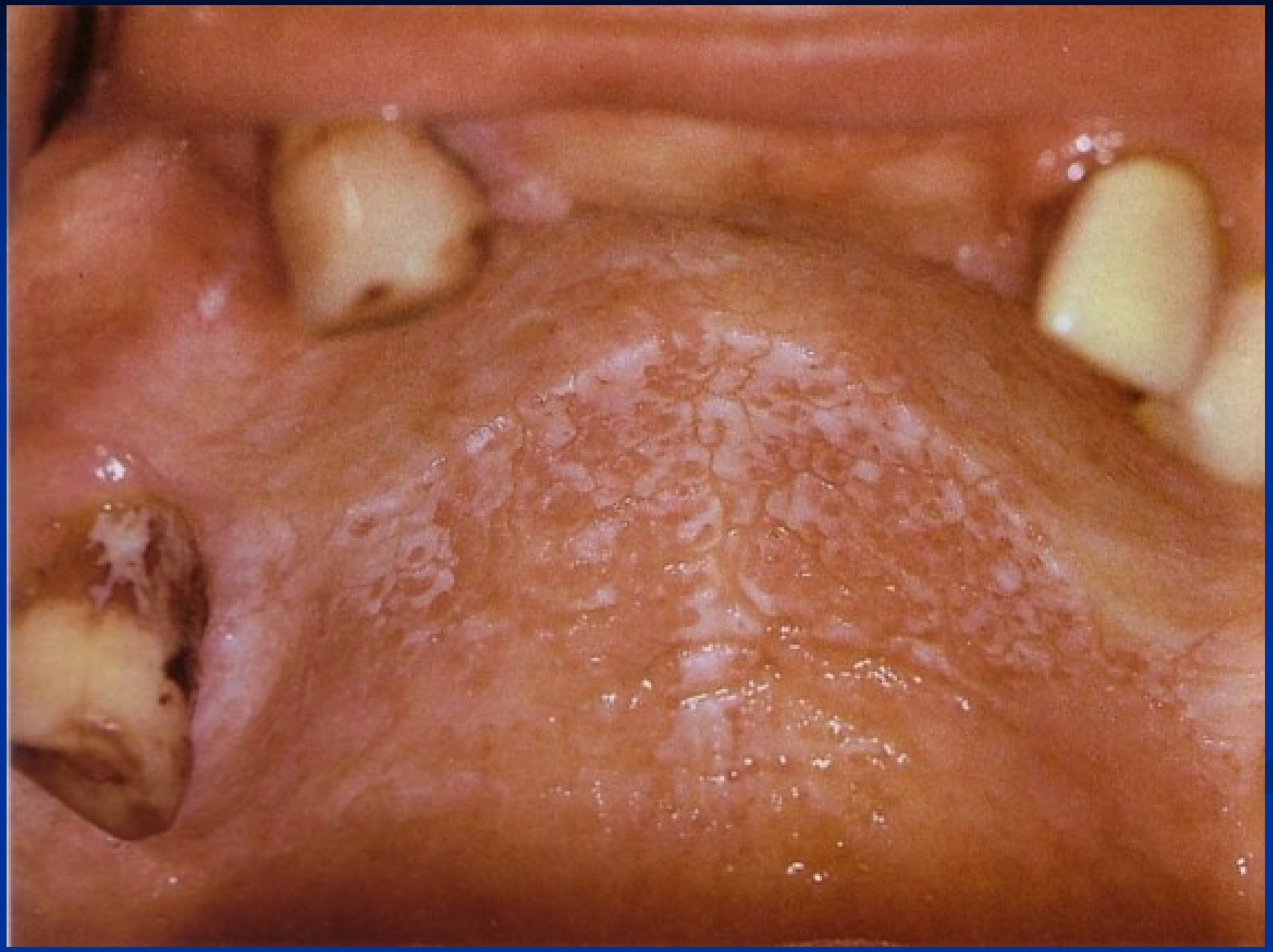
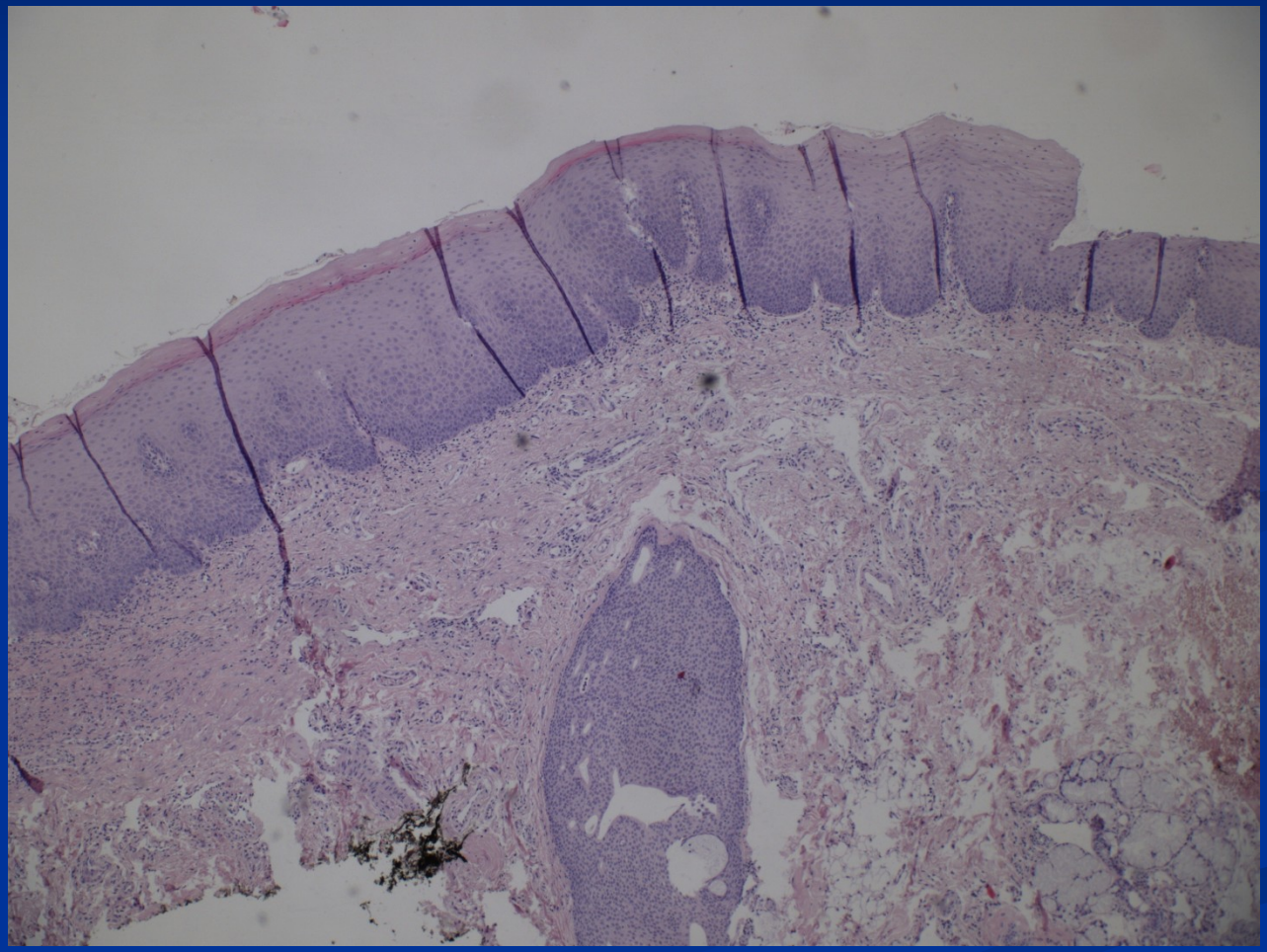
Nicotinic stomatitis

- Diffuse, dull greyish-white, opaque on hard palate
- Gradually fades to a normal pink on soft palate
- Severe - wrinkled or fissured surface texture
- Homogeneous with erythematous spots (inflamed minor salivary glands orifices)
- Maceration, ulceration and aphthae
- Tonsillar pillars are usually erythematous.
- Tobacco stains of the teeth, odor of tobacco, patient history confirms the cause of the lesion



Nicotinic stomatitis



Differential diagnosis

- Pipe or cigar + characteristic appearance.
- In long duration or non-healing ulceration, focal heterogeneous appearance and/or focal thickening:

CAVE CARCINOMA

Smokeless tobacco lesions (STL's)

- Changes in color and texture of the oral mucosa
- Common oral soft tissue lesions among young people.

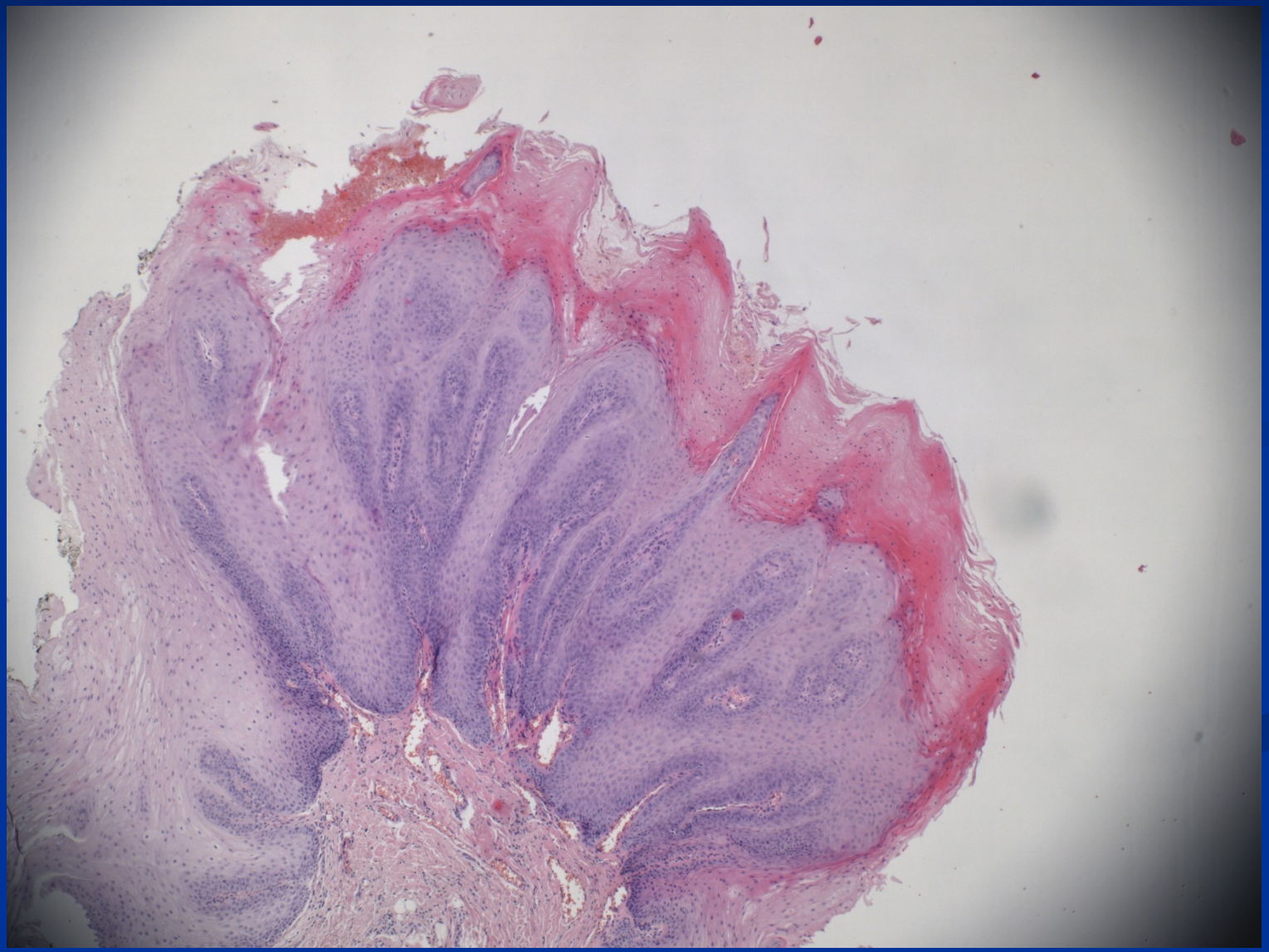
Hyperkeratosis caused by smokeless tobacco

- "snuff dipper's pouch."
- progression to carcinoma, (low grade squamous cell Ca, verrucous Ca).
- Western hemisphere (Sweden, US, Canada) lower carcinogenic rate
- Asia higher rate due to added carcinogens

Clinical features

- Hyperkeratotic lesion.
- Uniform, plaque-like thickening
- Homogeneous white, greyish-white, or dark color.
- Grainy rough surface with a uniform reticular pattern of wrinkles and folds (wrinkled, velvety).
- Labial, buccal, and facial alveolar mucosa.
- Large focal lesions, multiple sites (habit).
- Focal lesions - well delineated margins. Tobacco stains + residues.

Oral verrucous hyperplasia – possible precursor lesion



Differential diagnosis

- Appearance + history of habit: pathognomonic
- Treatment: quit habit, switch site
- ! x CA
  - non- healing ulceration
  - excessive verrucous thickening

Actinic cheilitis

- Alteration of the lower lip caused by chronic exposure to sunlight (UV).
- UV portion of the spectrum → cellular damage of the epithelium + the underlying connective tissue .
- Premalignant → + carcinoma cofactors (smoking).
- Biopsy if thickened or ulcerated

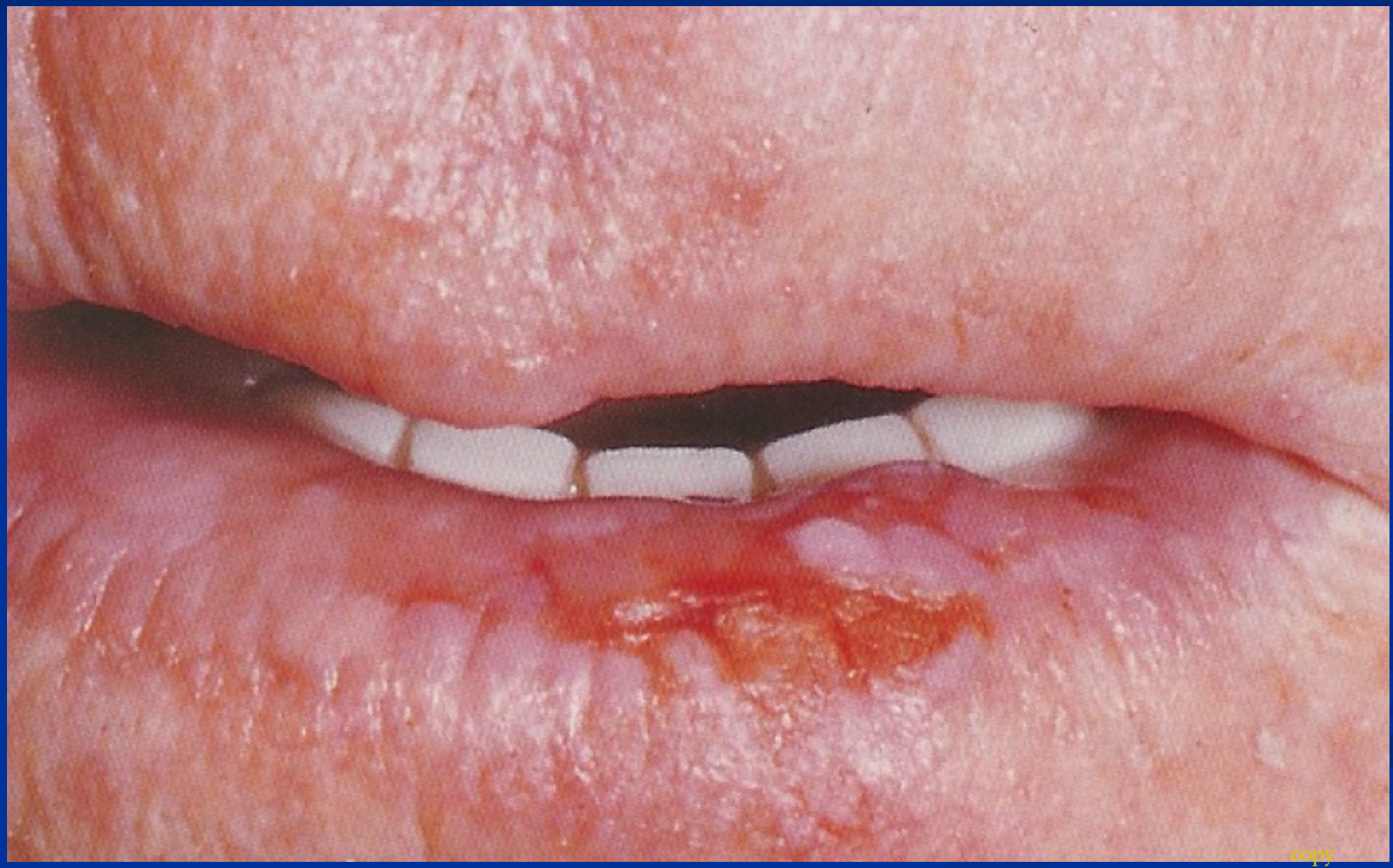
Clinical features.

- Fair skinned individuals.
- Usually over 60 yrs.
- Excessive sun exposure.
- Lower lip thinned + atrophic with indistinct demarcation of vermilion border.
- Persists for years, unchanged over an observation period of several months.

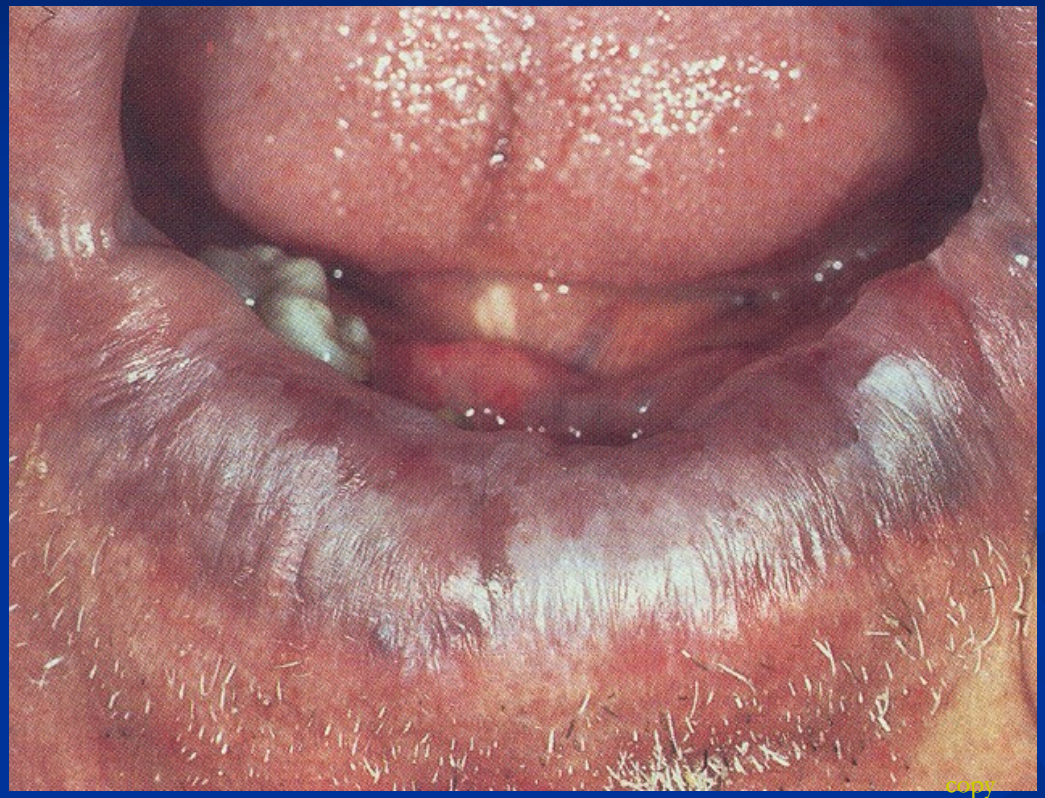
Actinic cheilitis

- Focal, homogeneous, milky-white, thickened patches.
- Ulceration is rare unless SCC is present
- Actinic keratosis → skin.
- Facial skin:
  - variation in pigmentation.
  - scaly atrophic patches.
  - seborrheic keratosis (thick, dark plaque).
- Possible skin cancers.

Actinic cheilitis

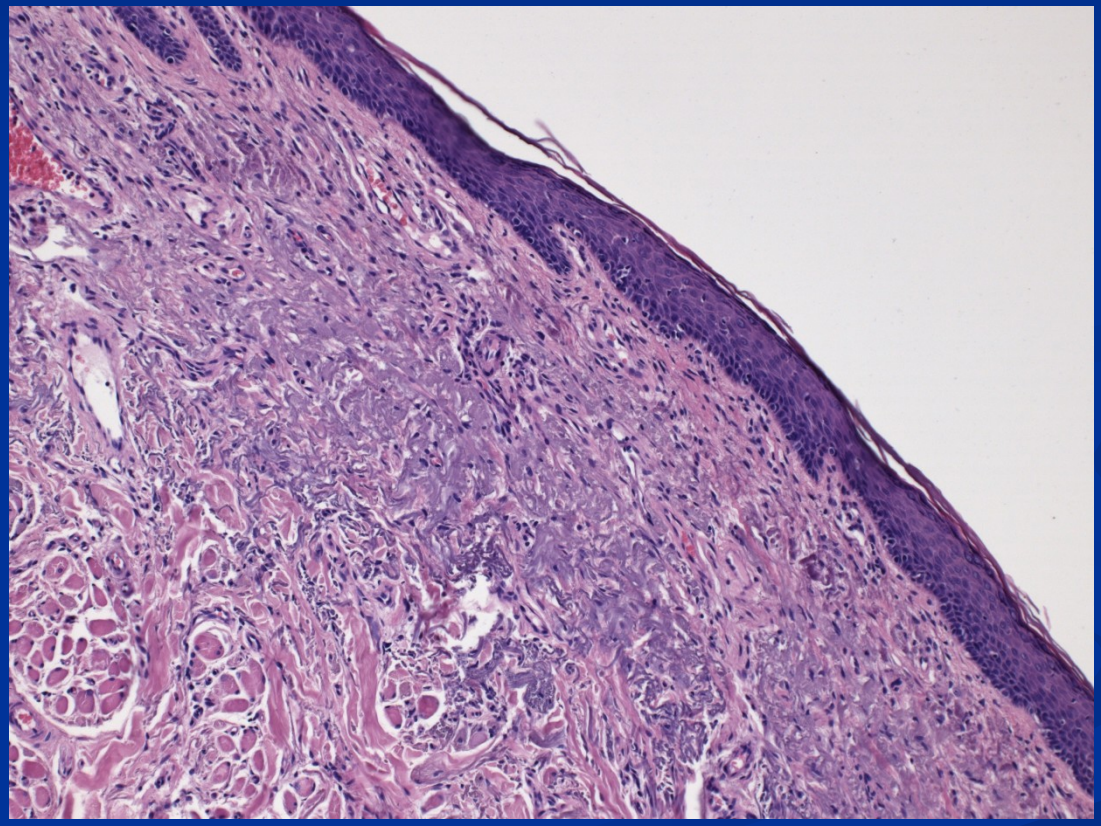


Actinic cheilitis

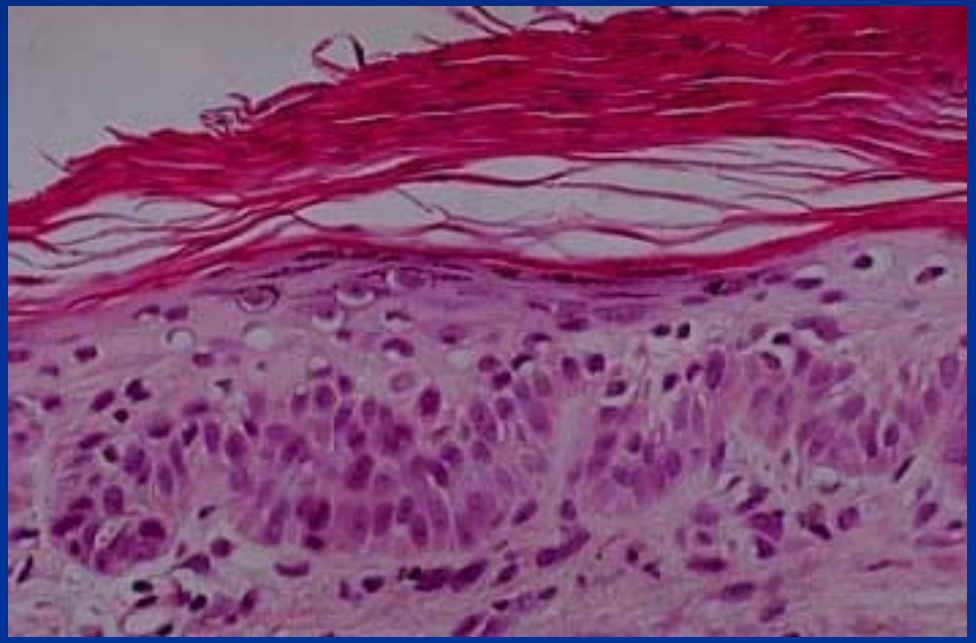


Lower lip thin + atrophic, indistinct demarcation of vermilion border.

Actinic cheilitis

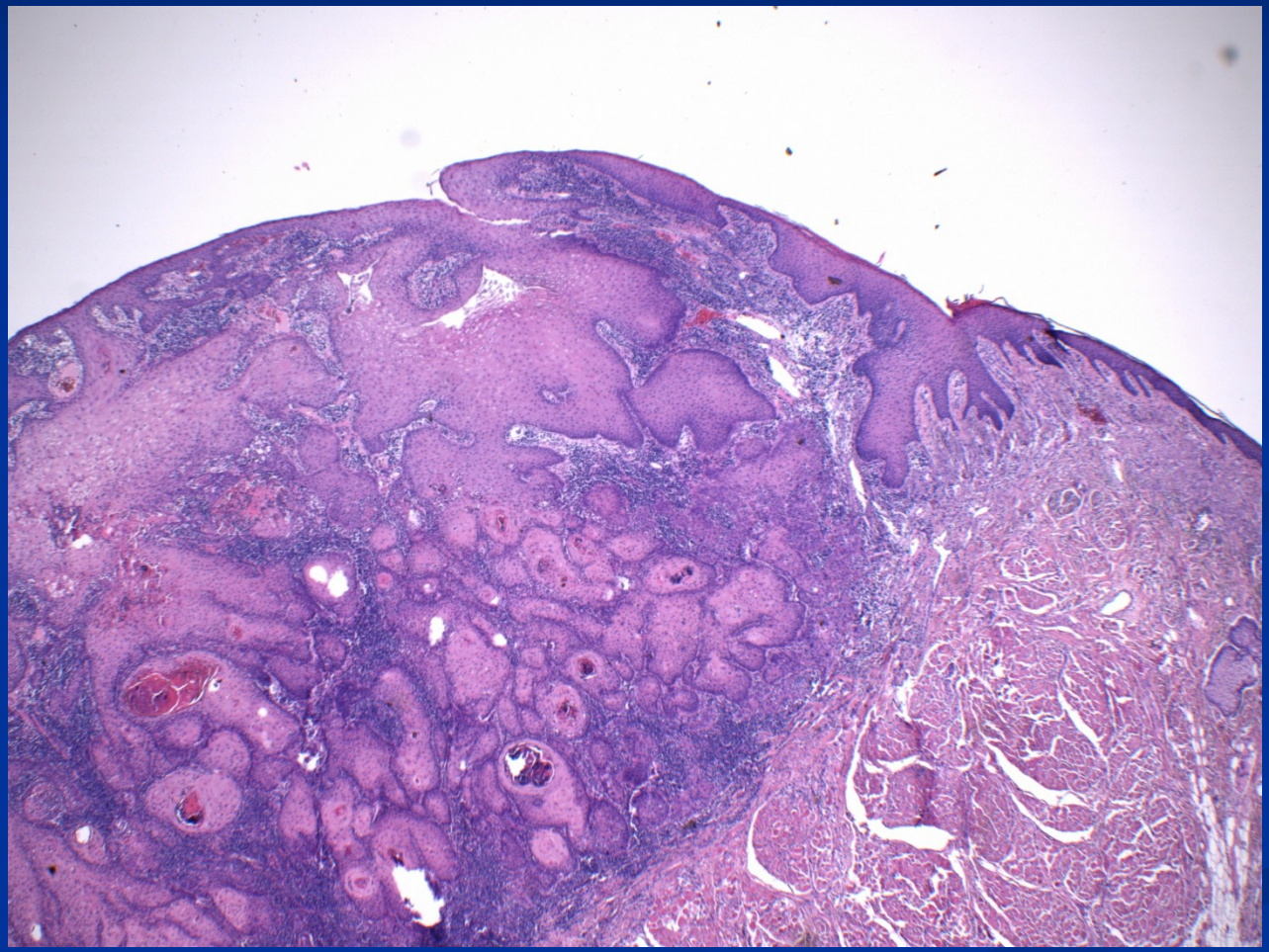


Actinic cheilitis



Superficial hyperkeratosis, dysplastic changes of squamous cell epithelium

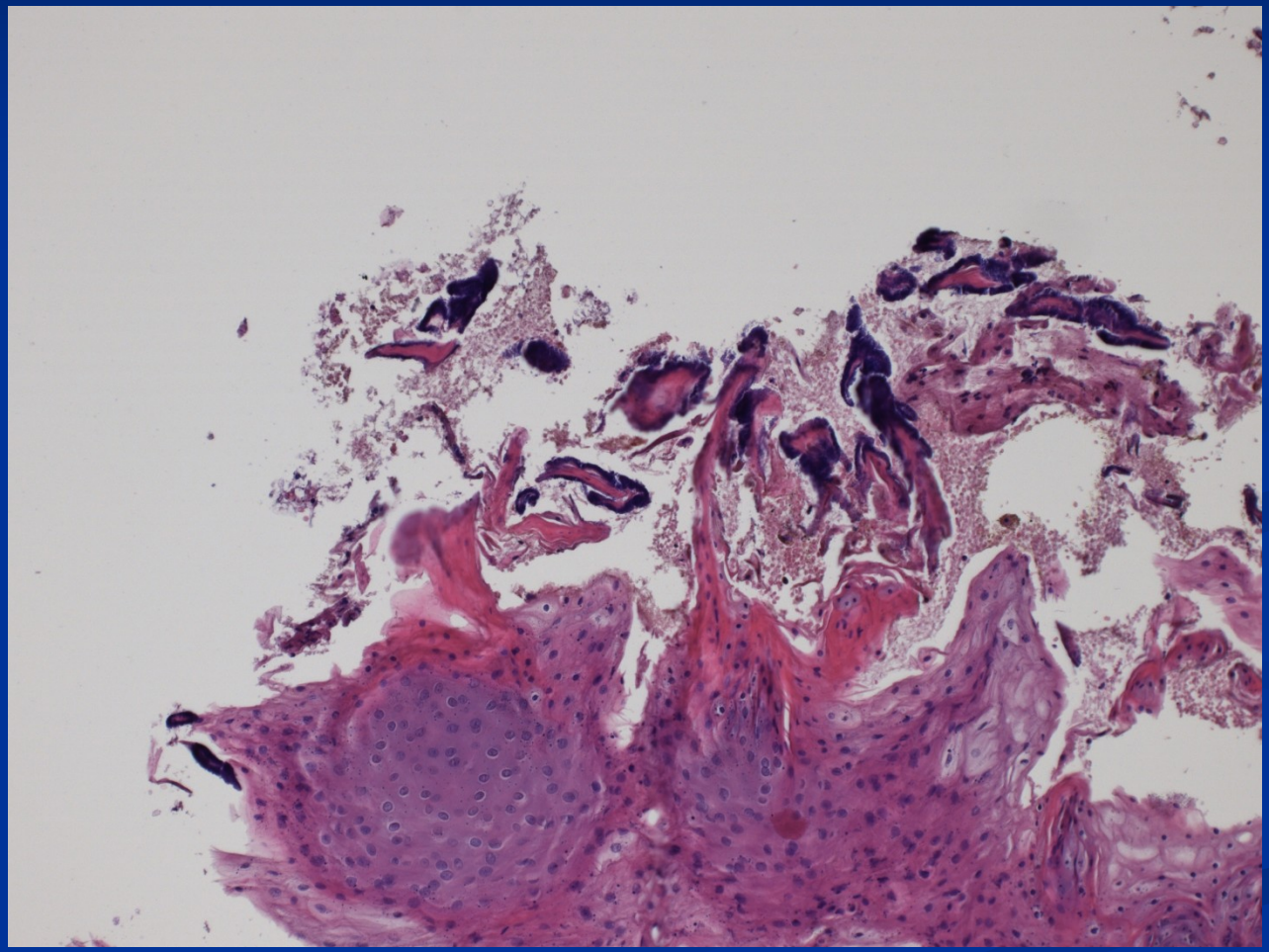
Squamous cell carcinoma - lip



Idiopathic or combined white hyperkeratotic lesions

- Geographic tongue (erythema migrans, benign migratory glossitis)
- Hairy tongue

Tongue papillae + bacteria



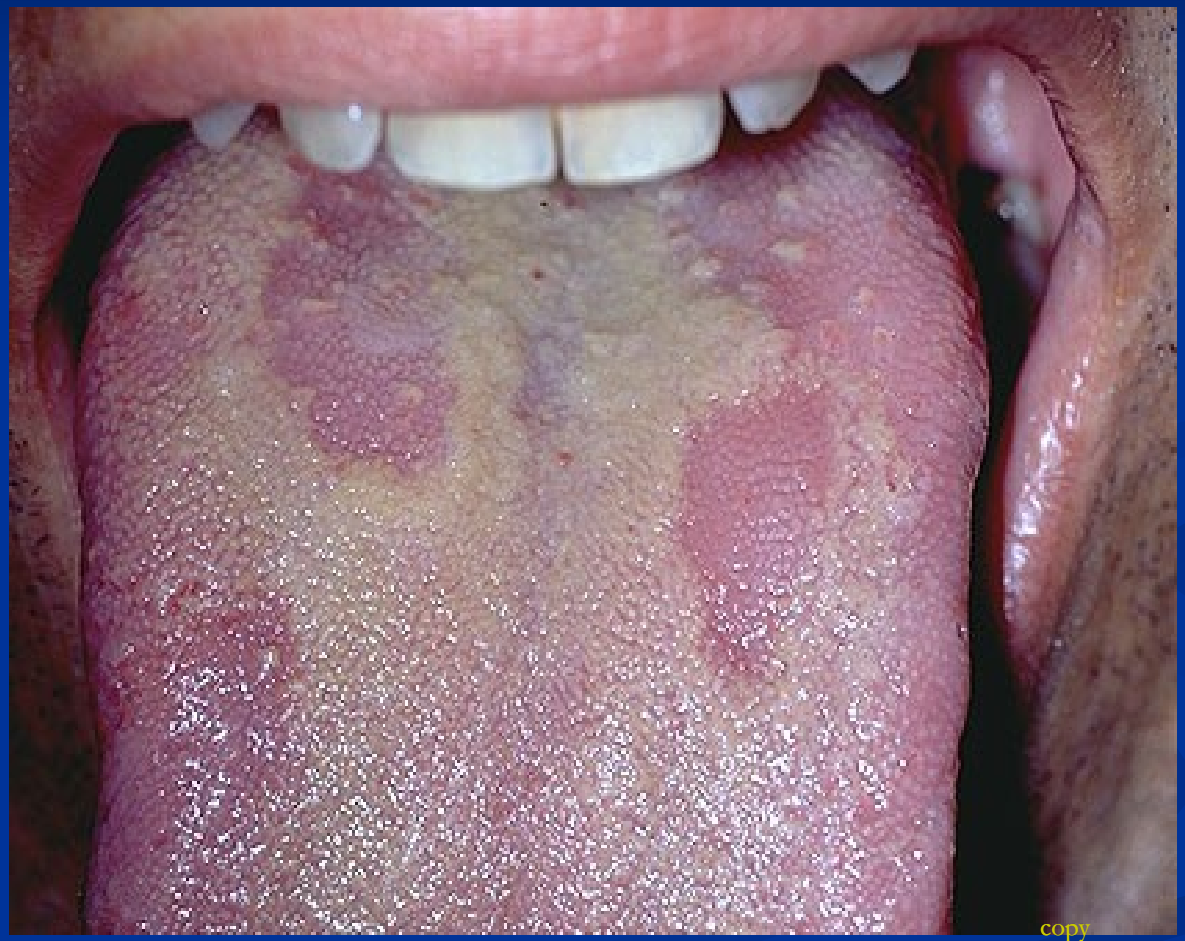
Geographic tongue

- Multiple areas of desquamation (*loss*) of the filiform papillae in several irregularly shaped but well-demarcated areas.
- May be on other parts of oral mucosa
- The smooth areas resemble a map → geographic tongue.
- Over a period of days or weeks, the smooth areas and the whitish margins „migrate“ across the surface of the tongue by healing on one border and extending on another.

Geographic tongue (Benign migratory glossitis)

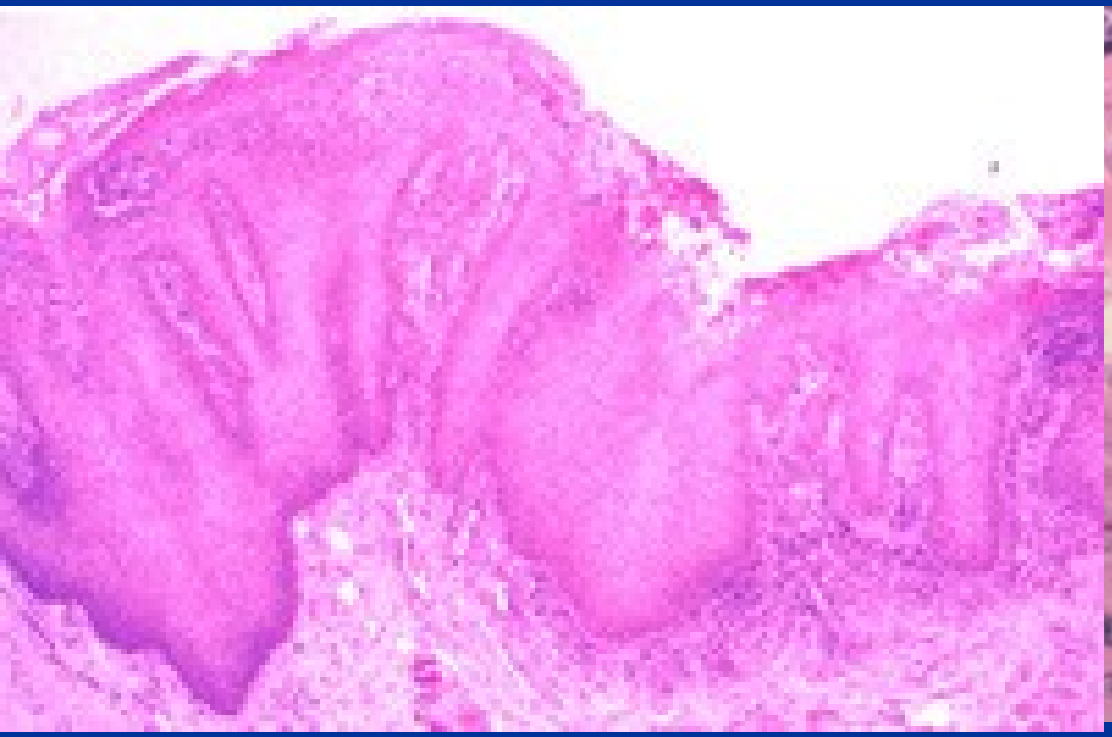
- Cause unknown, possible hypersensitivity to external factor + other factors (genetic, hormonal, …)
- White borders (+/-hyperkeratotic)
- Red patches of denuded filiform papillae
- Common disorder (1 - 2%), females, young adults
- Painfree usually
- Painful if inflammation present
- Treatment: none, or topical anesthetic

Geographic tongue



Geographic tongue

Histology: epidermal hyperkeratosis and marked transepidermal migration of neutrophils (Munro's microabcess-like). Cannot be differentiated histologically from pustular psoriasis or Reiter's syndrome.

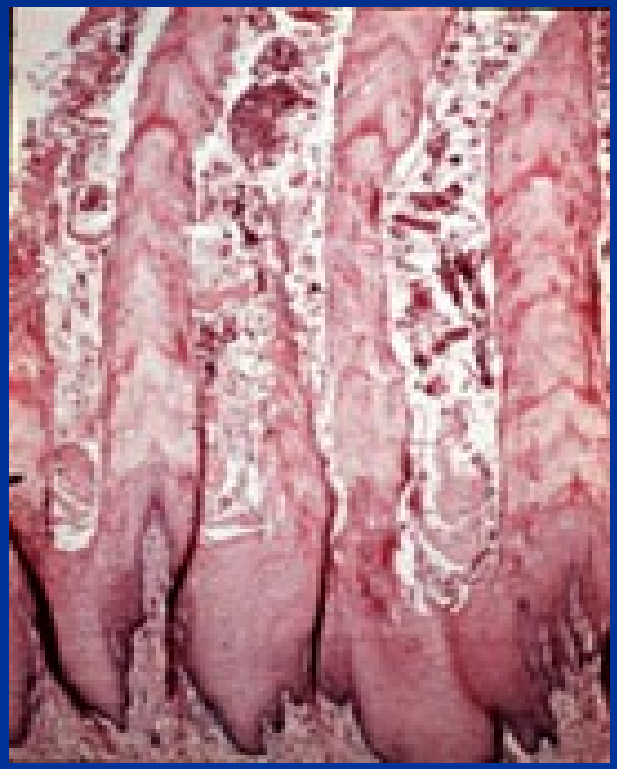


Hairy tongue

- Shaggy mat of filliform papillae
- In smokers, poor oral hygiene, antibiotics, …
- Hyperplasia may be stimulated by Candidiasis
- Coffee, tea, tobacco, bacteria → black discoloration
- Treatment: brush tongue, improve oral hygiene

Hairy tongue

Hairy tongue

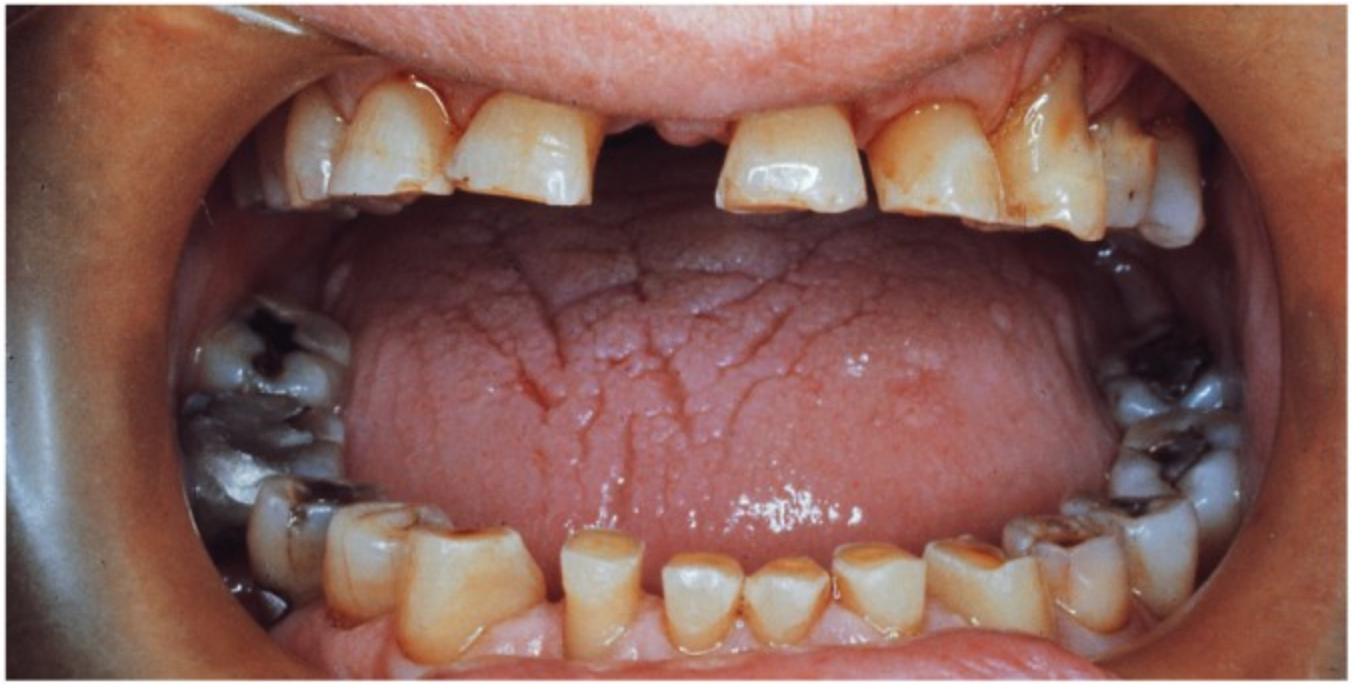


Elongation + hyperkeratosis of filiform papillae, superficial bacterial colonies
Dorsal localisation.
Differential diagnosis x oral hairy leukoplakia (EBV in immunodeficiency, on the lateral border of the tongue)

Fissured tongue

- A variant of norm, cause unknown.
- Some theories include a vitamin deficiency or chronic trauma over a long period.
- The dorsal surface (top) of the tongue appears to have deep fissures or grooves, irritation if food debris collects in them.

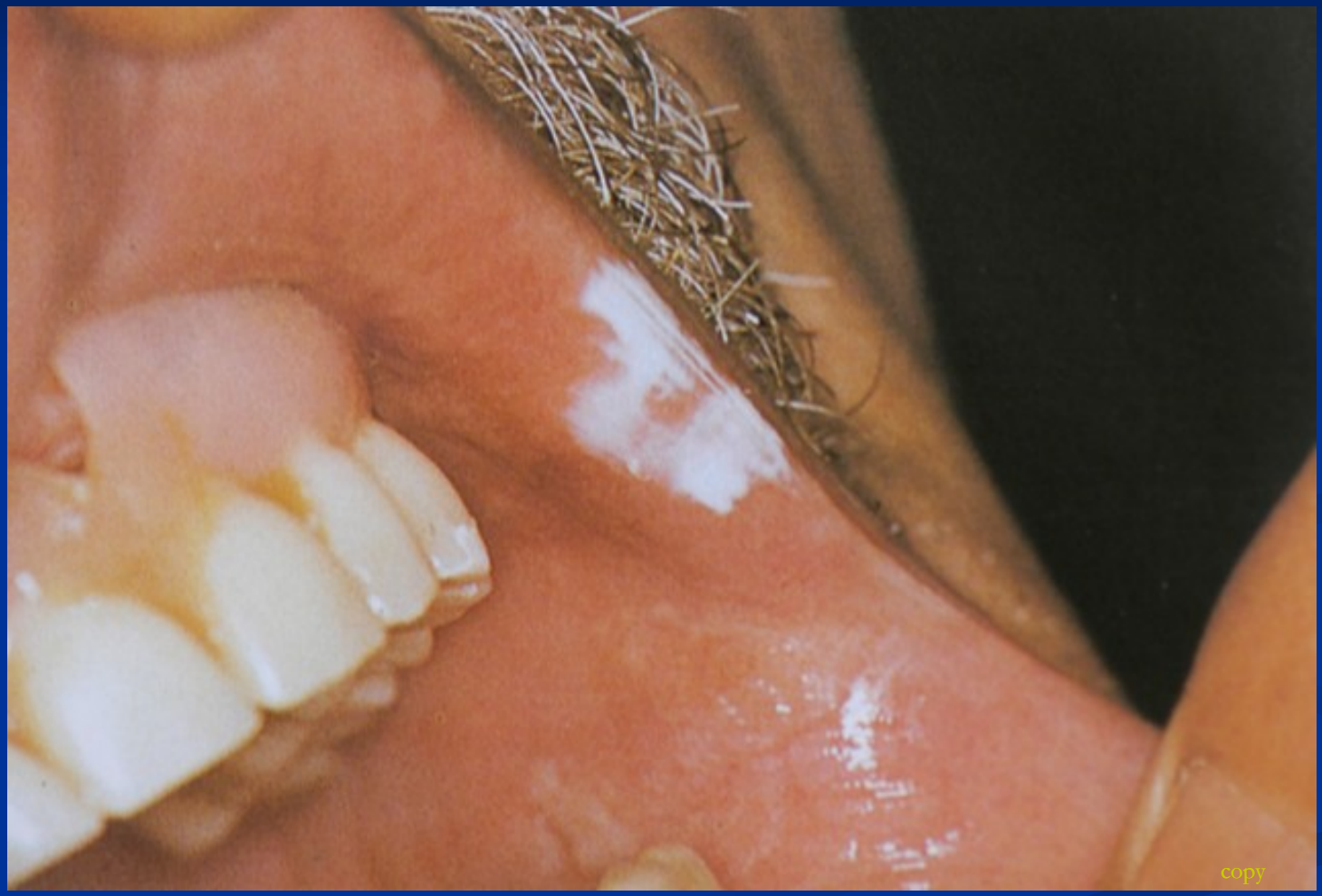
Fissured tongue



Chemical burn

- Different localization/size/appearance according to the type of chemical utilized, its concentration, the duration
- Whitish surface → desquamating → painful erosion or ulcer → bone damage
- Healing within 1-2 weeks

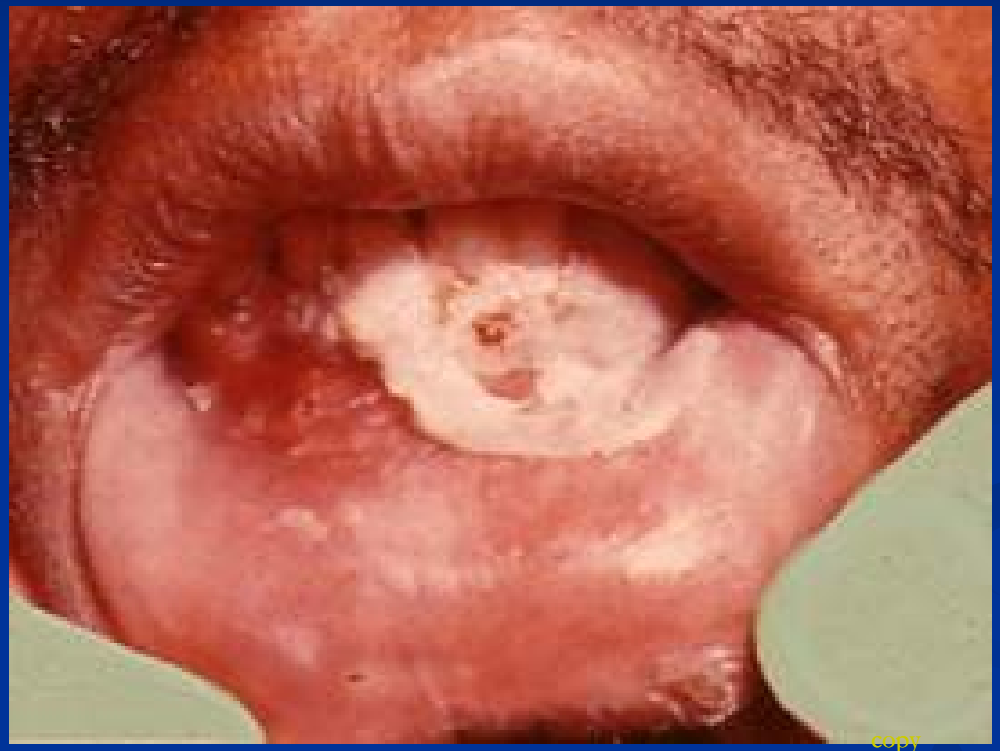
Chemical burn



Chemical burn

- Commonly caused by aspirin
- Painful
- Usually in molar region
- Treatment = discontinue aspirin use

Aspirin burn



Diff. dg. of white lesions Epithelial dysplasia and early squamous cell carcinoma

- Most important diff. dg.
- Dysplasia: premalignant alteration in the differentiation, development, and maturation of lining epithelial cells.
- Squamous cell carcinoma: malignant neoplastic proliferation of lining epithelial cells.
- Gross: focal epithelial thickening - leukoplakia

RED LESIONS

Focal erythematous lesions

Nonspecific mucositis

Mucosal burn

Macular hemangioma (benign tumor)

Erythroplakia (precancerosis)

RED LESIONS

Diffuse and/or multifocal red lesions

- Geographic tongue (erythema migrans)
- Vitamin deficiency induced glossitis
- Radiation mucositis
- Xerostomic mucositis
- Allergic mucositis
- Lupus erythematosus (immunologic)

Nonspecific mucositis (irritational):

- Age, sex (different causes – piercing, denture…).
- Clinical features:
  - Localized zones of redness correspond to the source of irritation.
  - Related to a physical agent.

Nonspecific mucositis (irritational):

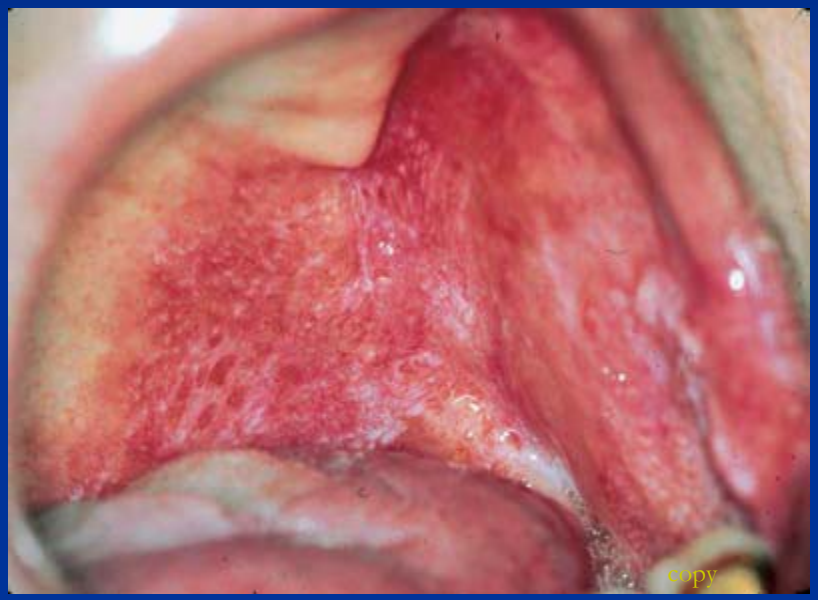
- Differential diagnosis:
  - ! precancerous erythroplakia
- May progress to ulcerative lesion
- Treatment:
  - Elimination of the irritating agent.
  - Analgesia

Erythroplakia (precancerous)

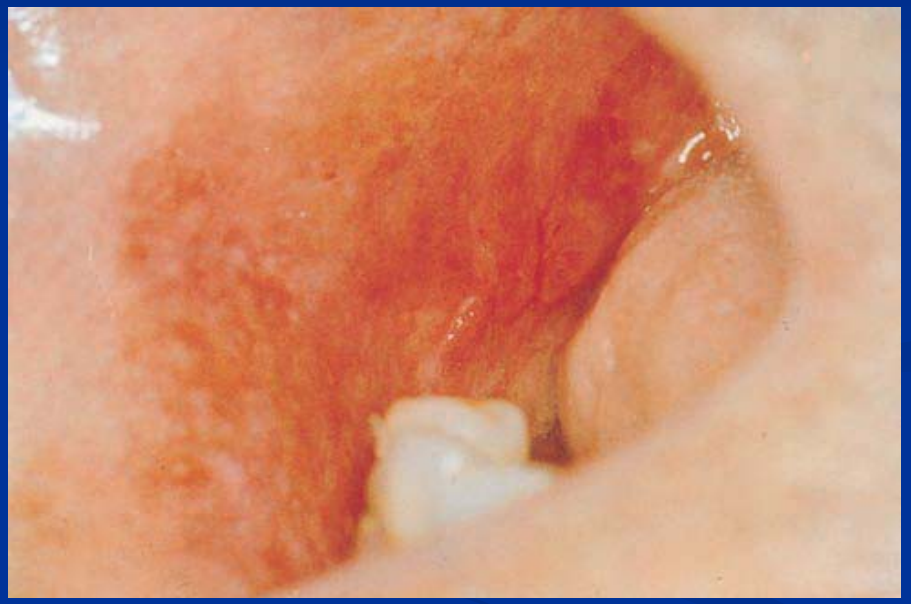
Bright red velvety plaque which cannot be characterized clinically or pathologically as being due to any other condition.

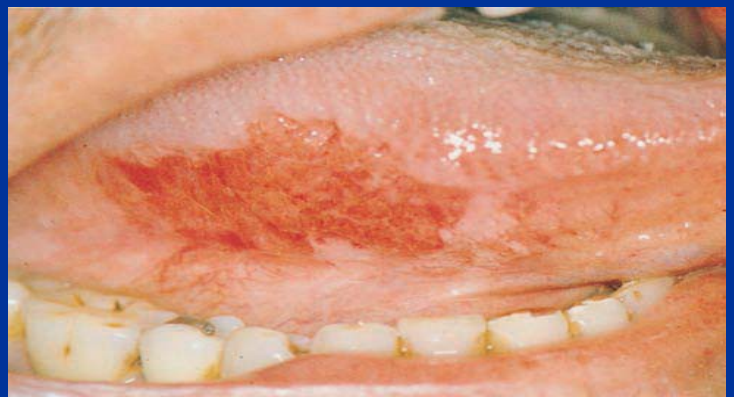
Erythroplakia

Erythroplakia of the buccal mucosa



Erythroplakia of the buccal mucosa.





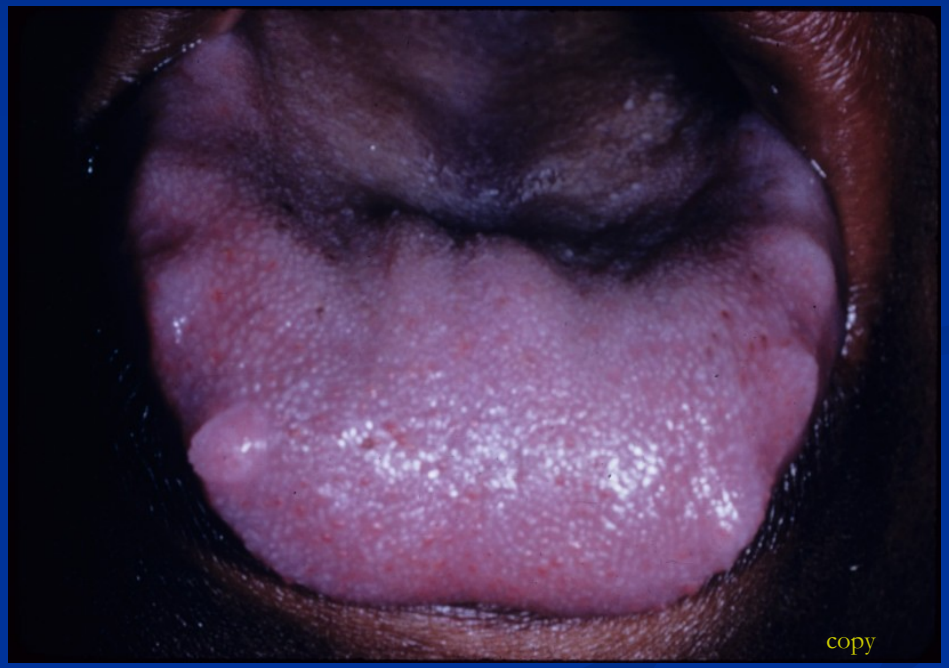
Erythroplakia of the lateral margin of the tongue.

Acute ulcerative lesion

- Combined mucosal damage – acute necrosis, commonly in form of burn (aspirin burn) →
- Erosion → healing by epithelial regeneration, may become hyperkeratotic
- Ulcer → healing by granulation tissue
- Physical factors – thermal, electrical burn
- Chemical – caustic drug reactions (aspirin, hydrogen peroxide, silver nitrate, cleaning substances – acids, etc.)

Scalloped tongue

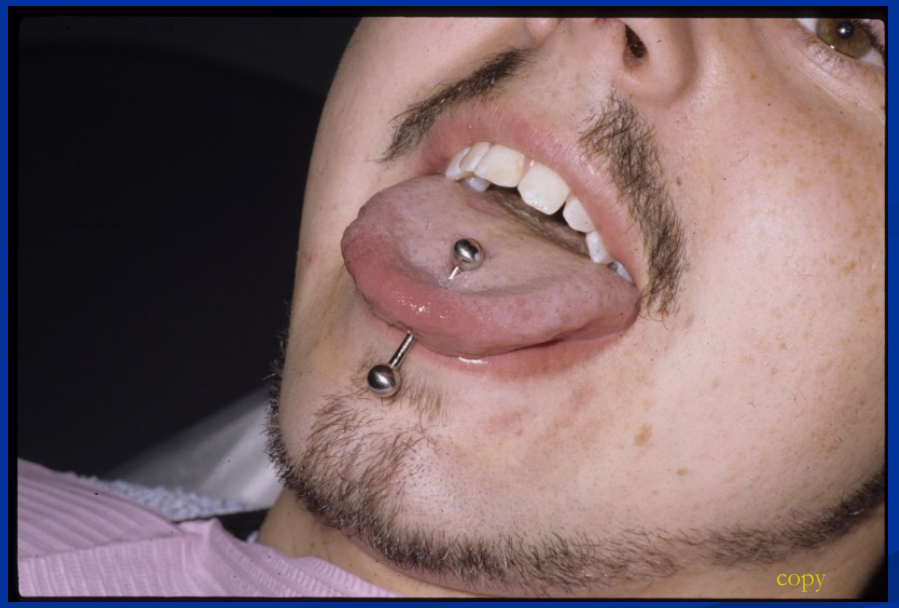
- Indentations along the lateral borders of the tongue
- Correspond to the teeth
- Thought to be habitual pushing of tongue against teeth
- Possible progression to ulcers



Tongue jewellery

- Pierced tongue

- Can affect the teeth and/or gingiva
- Multiple complications possible



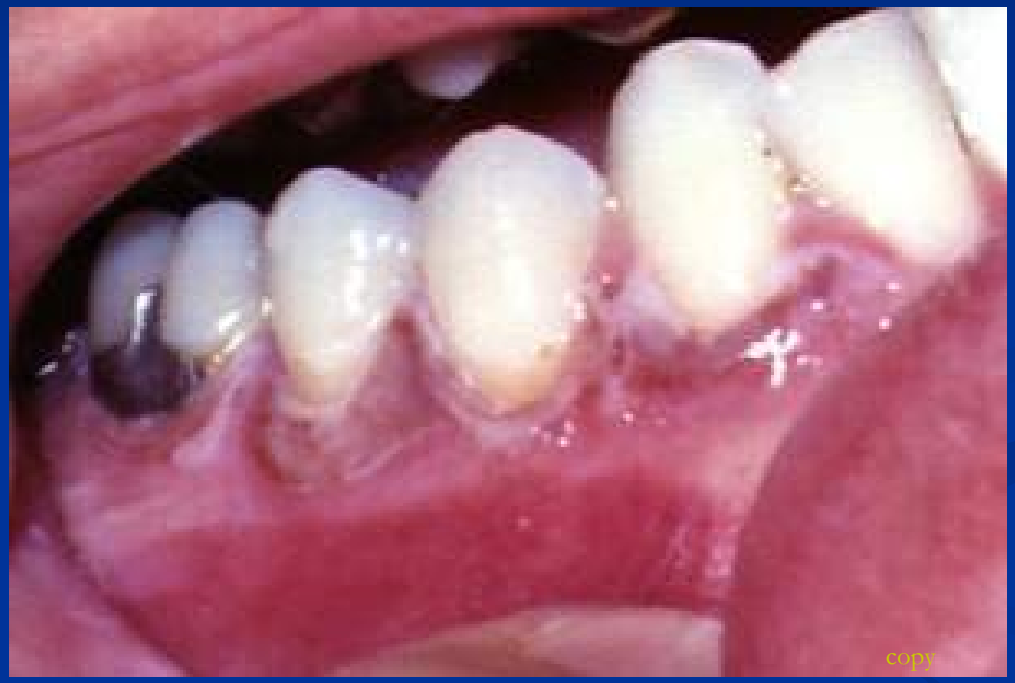
Mucosal burn

- **Clinical features:**
  - Chemical agents → caustic → coagulation necrosis of epithelium → whitish (can be scraped off)
  - Diluted chemicals → inflammation and redness without producing superficial necrosis (erythema of the superficial tissues)
  - Thermal burns: hot foods, caustic drug or beverages → palatal erythema → painful.

Mucosal burn

- Differential diagnosis:
  - History.
  - Hypersensitivity reaction.
  - Biopsy (cytological atypia).
- Treatment:
  - Stop the irritant.
  - Topical analgetics
  - Avoid spicy foods

Hydrogen peroxide burn

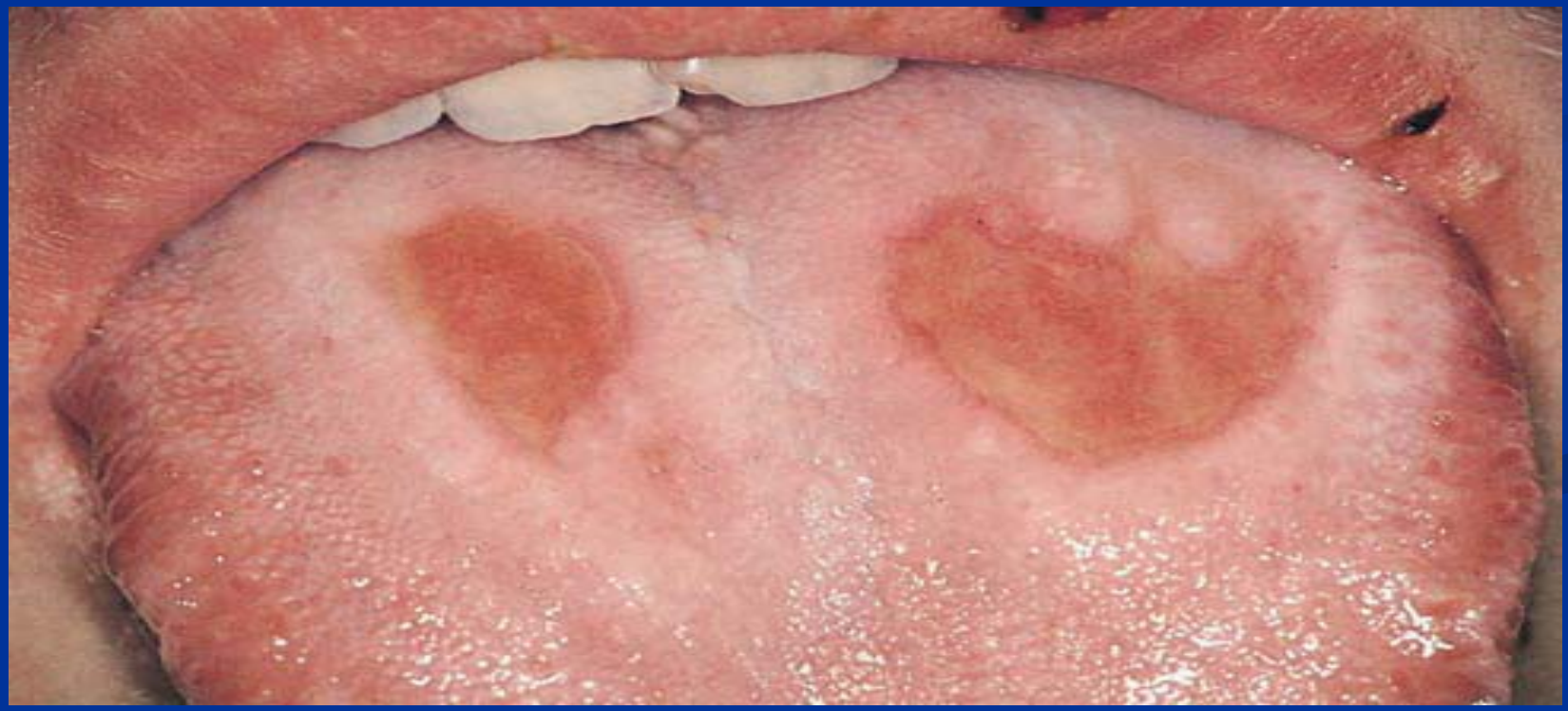


Thermal burn

- Acute lesion (x stomatitis nicotinica)
- very hot foods, liquid, or hot metal objects
- palate, lips, floor of the mouth, tongue
- initially painless, no bleeding; quick (hrs) edema evolution
- painful, red, necrosis undergoing desquamation, leaving erosions or ulcers, ! complications – bleeding, infection
- supportive treatment; self-healing in about a week

Thermal burn

- Erosions on the dorsum of the tongue, caused by very hot food (microwave oven, …)



Electrical burn

- Similar to thermal burn
- Commonly significant tissue destruction incl. bone

Physical injury

- **Traumatic ulceration**

mechanical

factitious injury

traumatic granuloma – eosinophilic ulcer

thermal

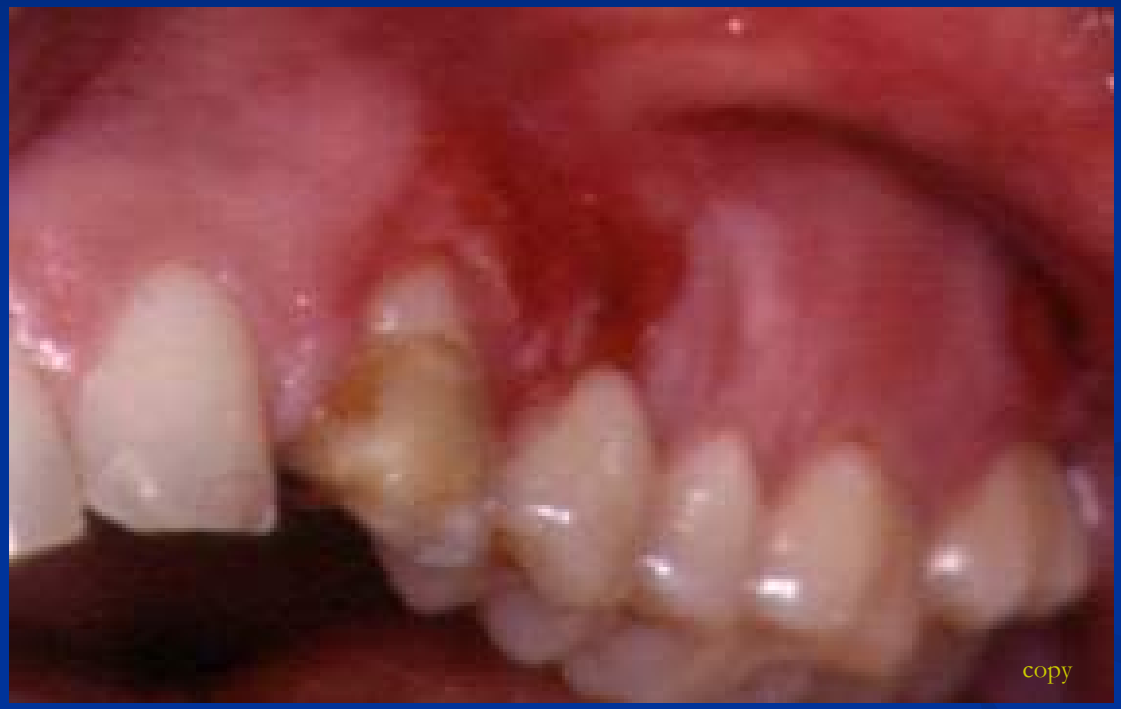
radiation

Factitious ulcer

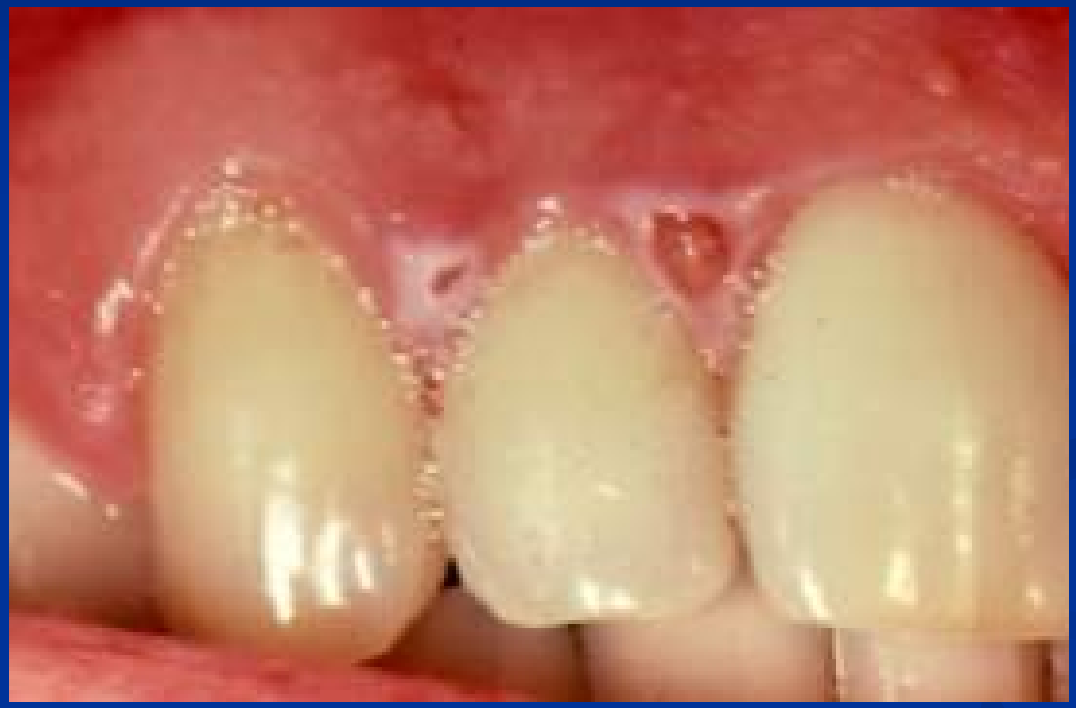
- Patients mentally handicapped, with serious emotional problems, incarcerated
- Oral self-inflicted trauma by biting, fingernails, or by the use of a sharp object
- Tongue, lower lip, gingiva
- Slow to heal due to perpetuation of the injury by the patient
- Local measures and psychiatric therapy

Traumatic injury – factitious

erosion → ulcer



Toothbrush trauma - friction



Etiology of ulcerations

Mechanical factors: a sharp or broken tooth, rough fillings, clumsy use of cutting dental instruments, hard foodstuffs, sharp foreign bodies, biting of the mucosa, denture irritation etc.

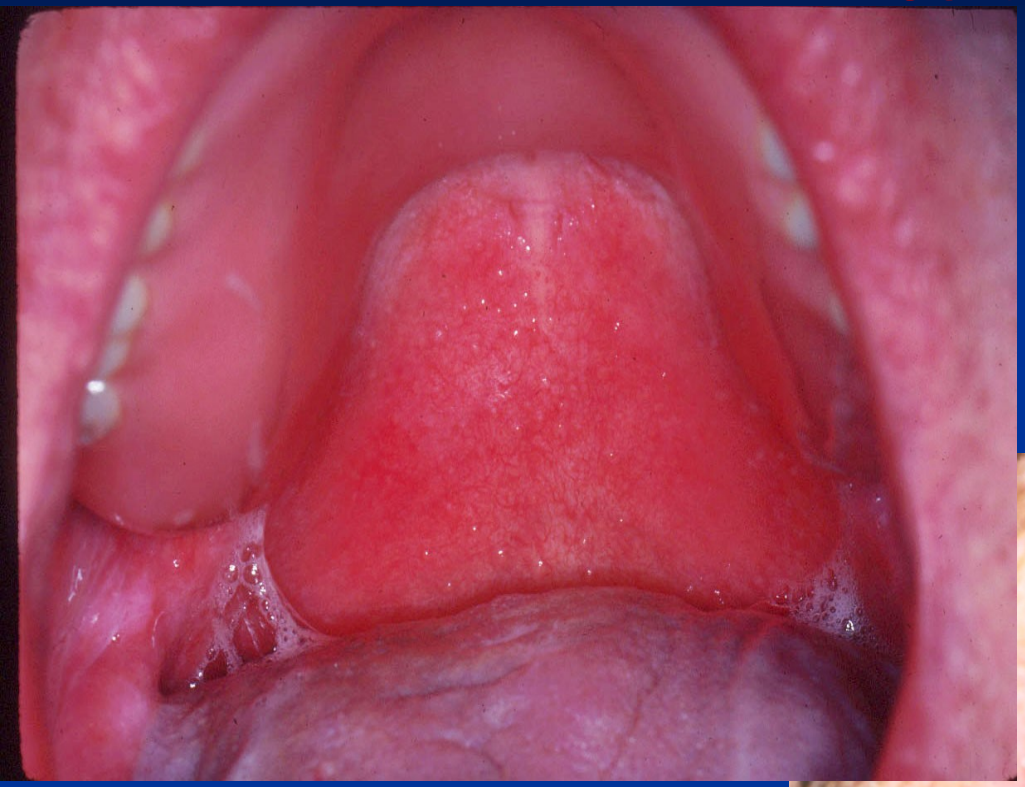
Physical factors: thermal burns etc.

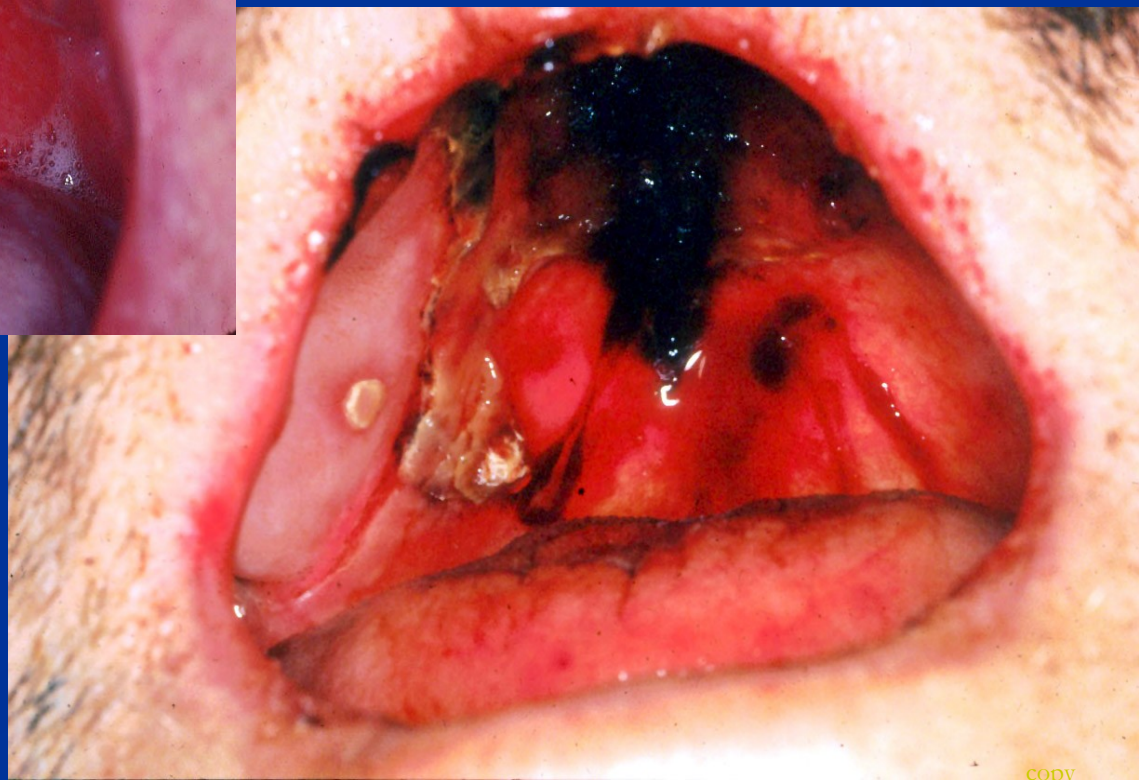
Chemical factors: strong acid, strong base, $Ag(NO)_3$, iodophenol

Decubital ulcer - clinical features

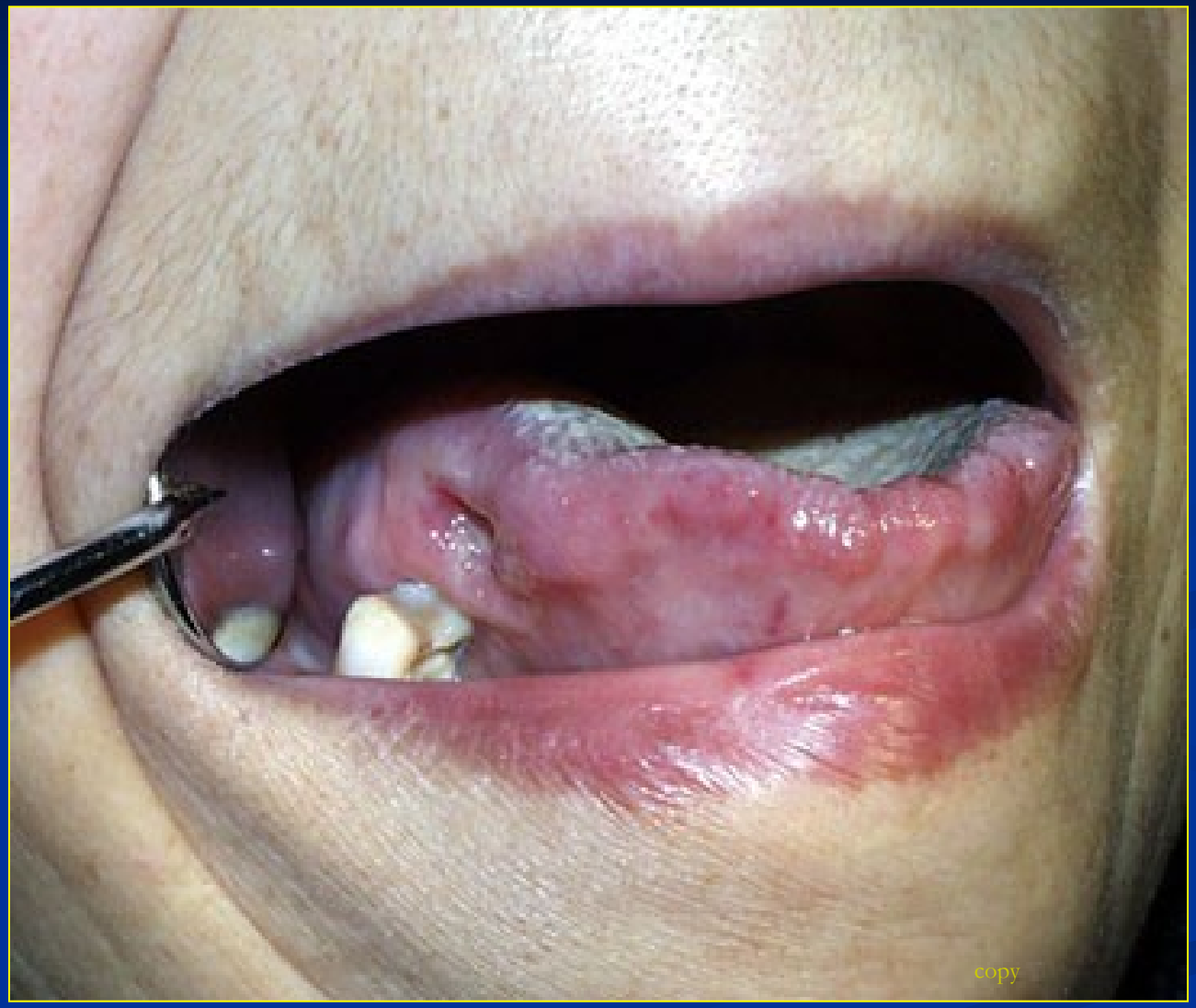
- mechanical irritating factors
- the ulcer conforms in area and linearity to the source of the irritating factors
- may affect mucosa, deep soft tissue, rarely progresses into the bone

Trauma – dentures – decubital ulceration

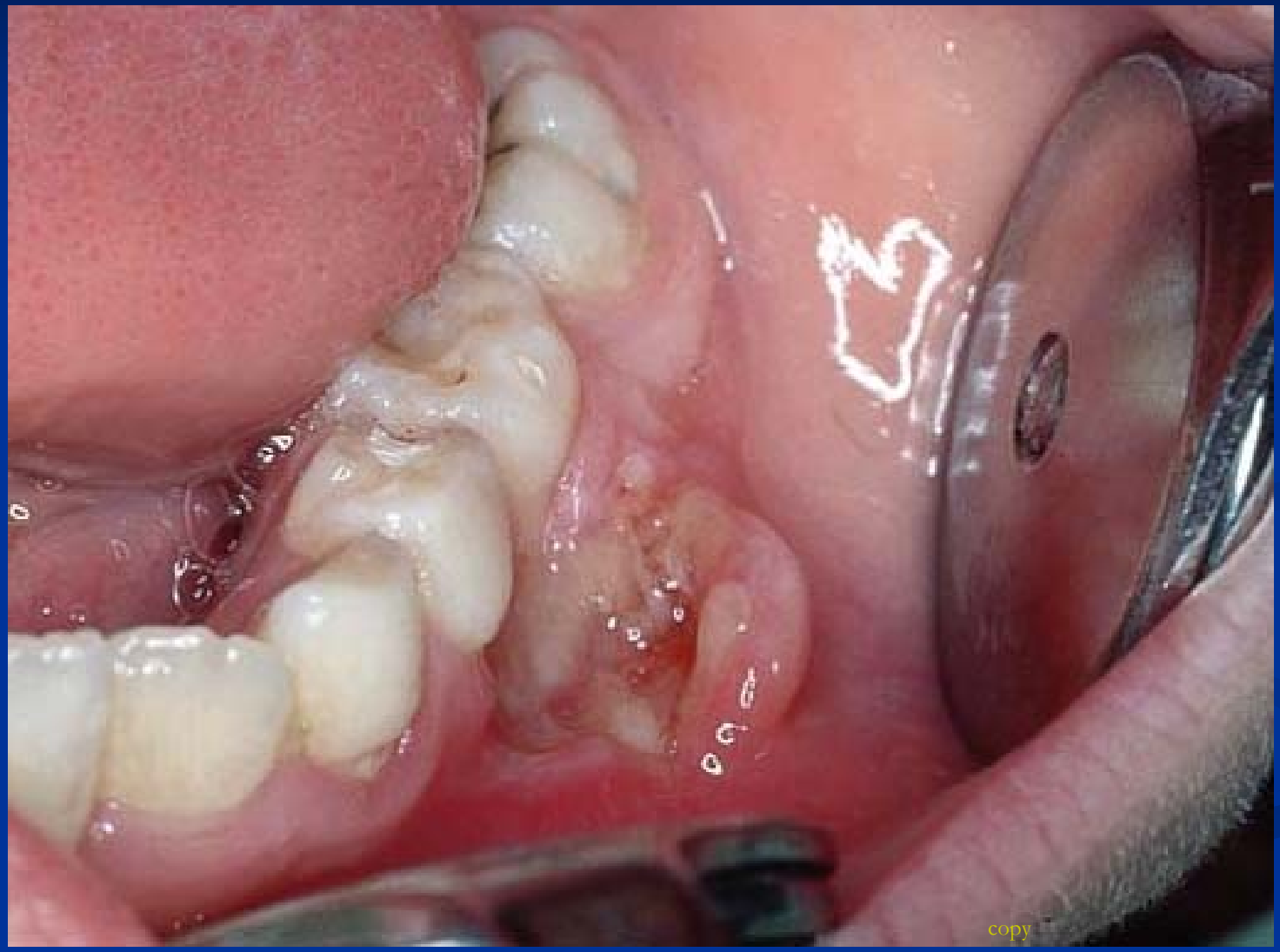




Decubital ulcer



Traumatic ulcer

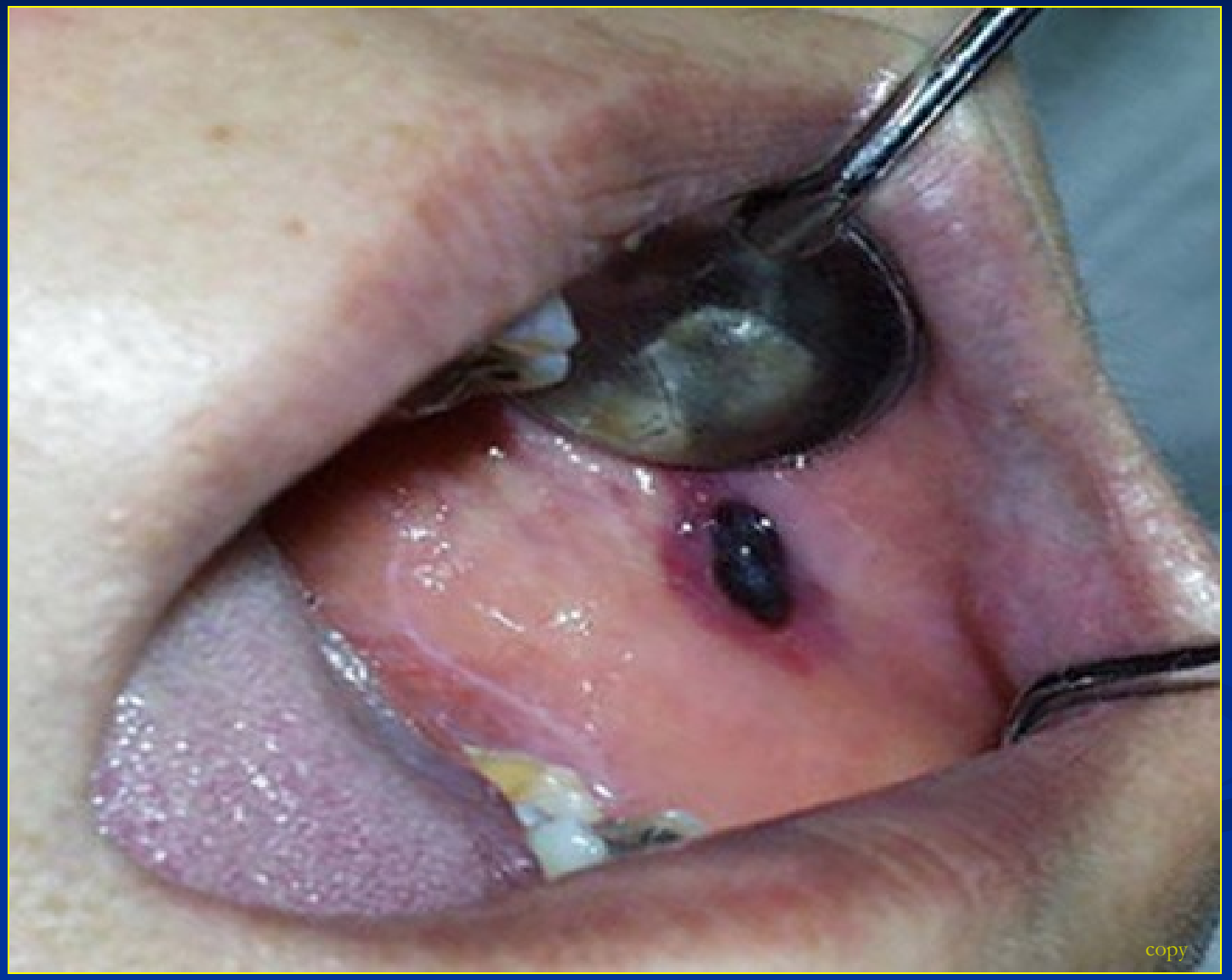


Traumatic ulcer
Traumatic bulla

Frequent, due to the constant motion of the masticatory mucosa over the teeth or the introduction of hard objects into the oral cavity.

- buccal mucosa, soft palate, lips, tongue
- self-healing in 4-6 days

Traumatic bulla



Traumatic bulla

Diagnosis

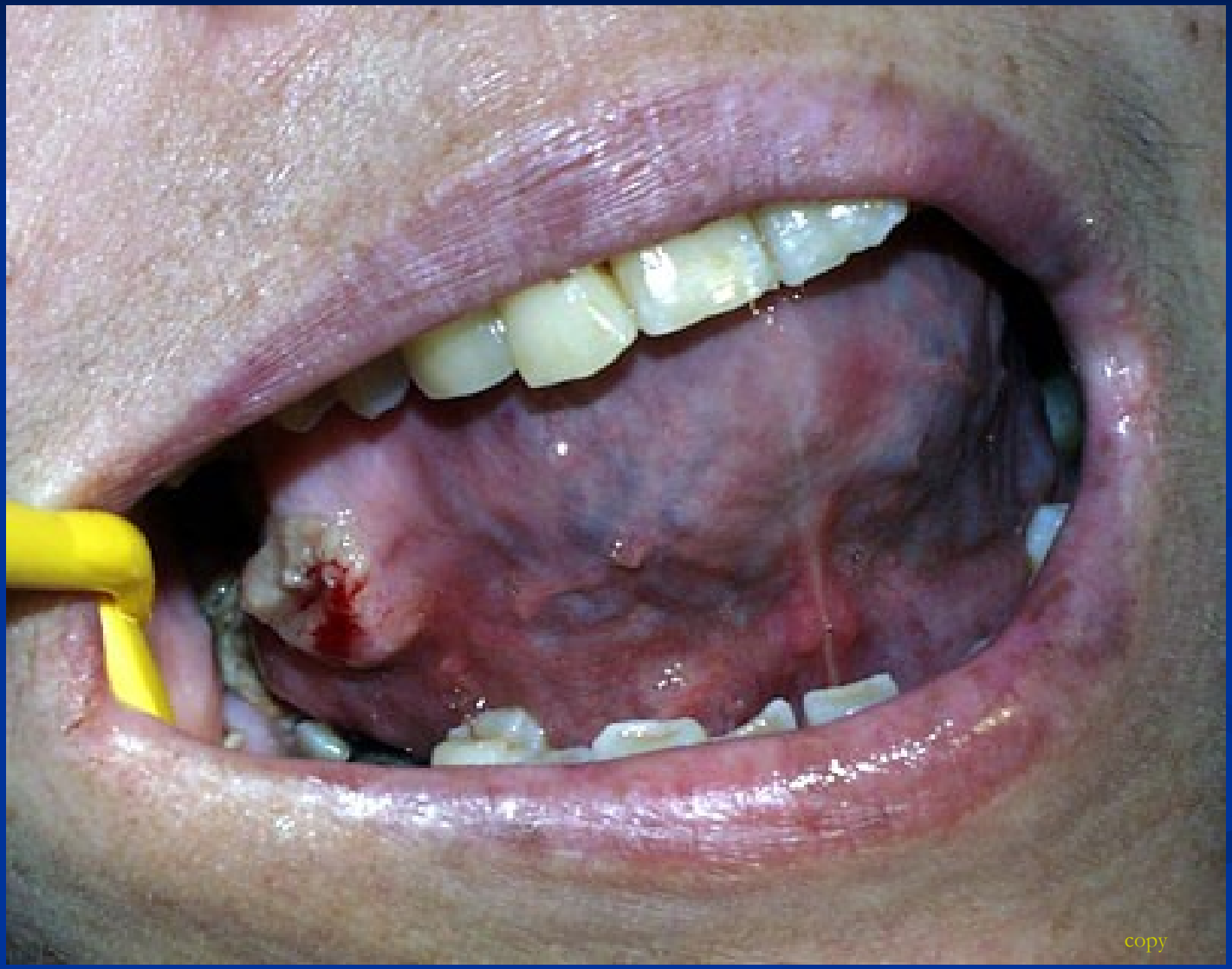
History

Clinical features

Differential diagnosis

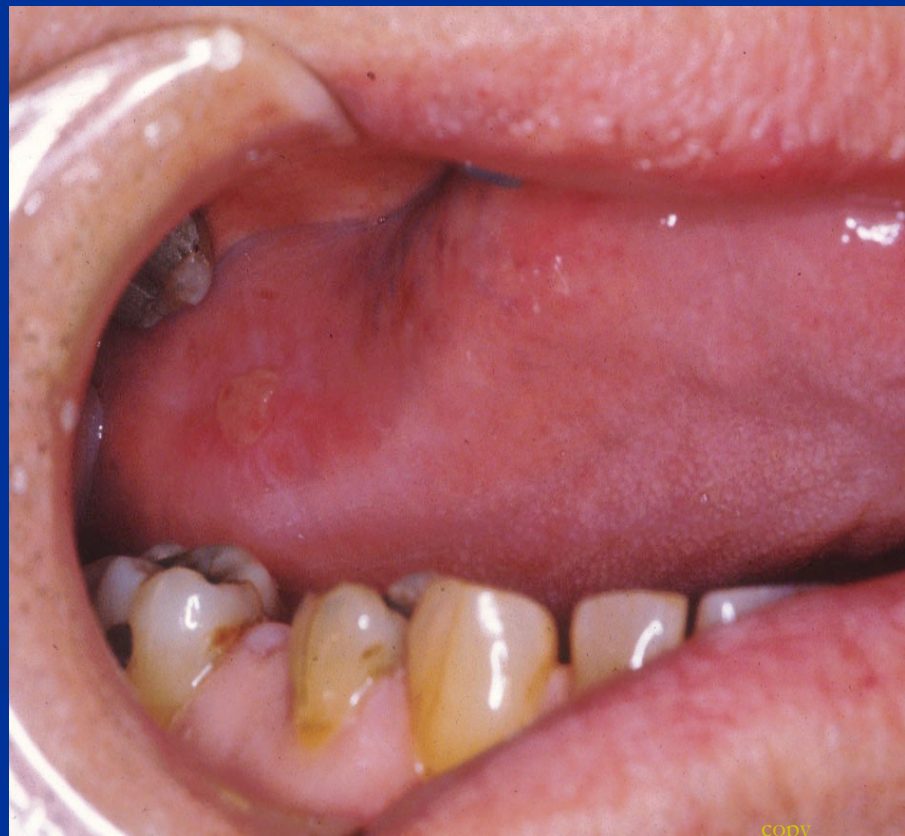
carcinoma, syphilis, tubercular ulcer, major aphthous ulcer
thrombocytopenia, thrombasthenia, pemphigus, cicatricial pemphigoid

Malignant ulcer

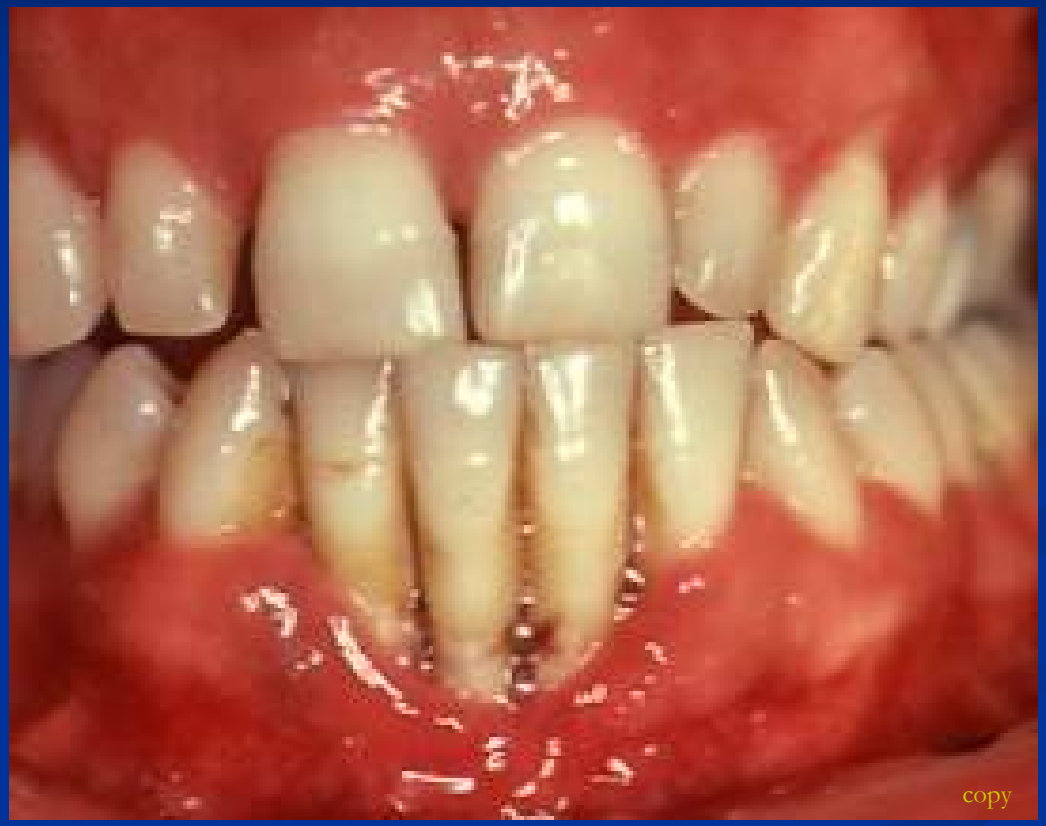


Soft tissue necrosis

- Traumatic vs. spontaneous
- Intraoral source RT
- Possible late occurence
- Prolonged duration

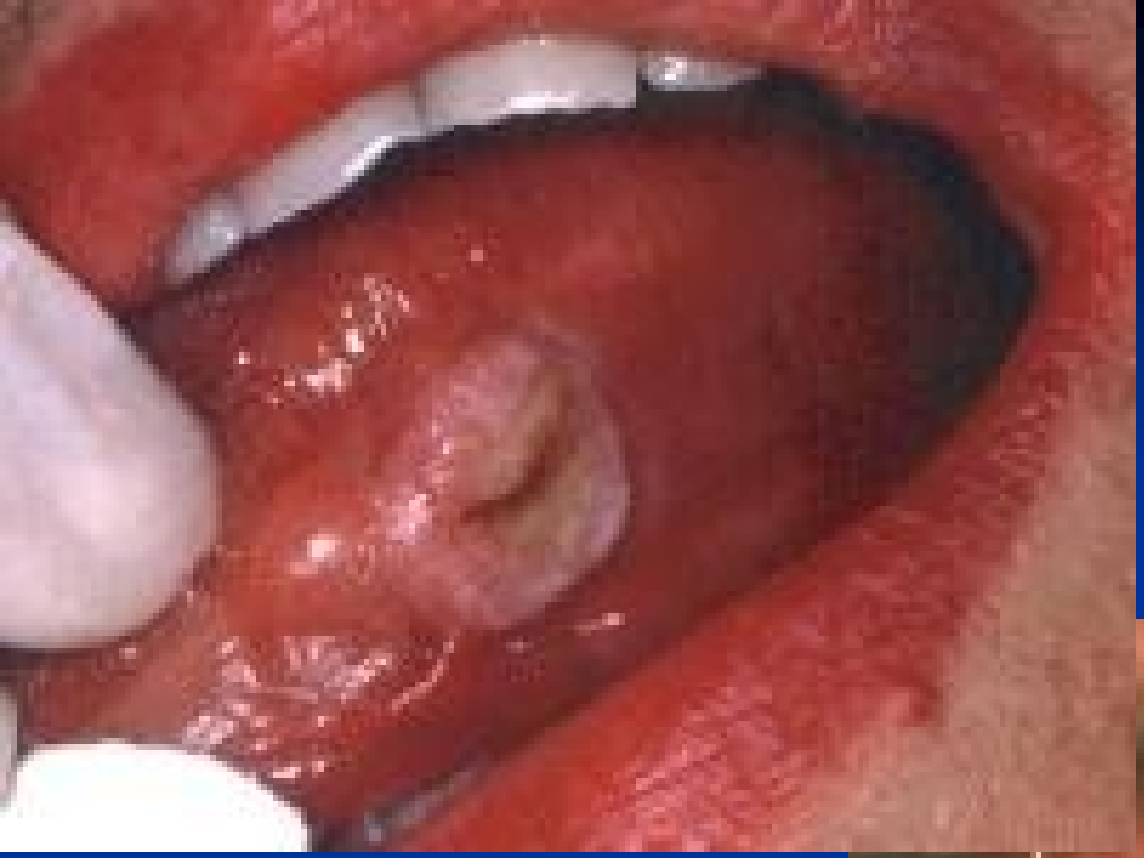


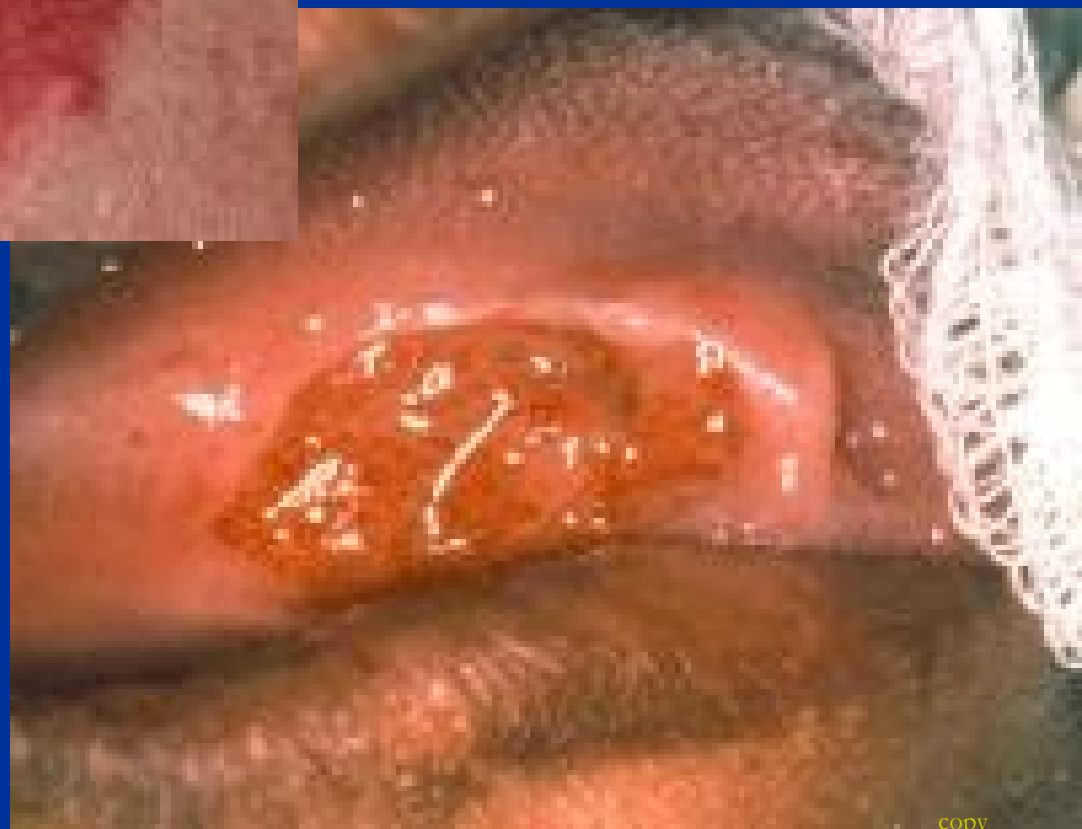
Cocaine induced necrosis



Eosinophilic ulcer

- Ulcer with elevated borders usually covered by a pseudomembrane.
- Commonly posterior aspect of tongue
- Rapid onset, spontaneously resolves in a few weeks. Benign, self-limited.
- Micro: predominantly eosinophilic infiltrate with histiocytes and neutrophils
- If multifocal and recurrent, CD30 positive lymphoproliferative disease may be present.





Healing of ulcers

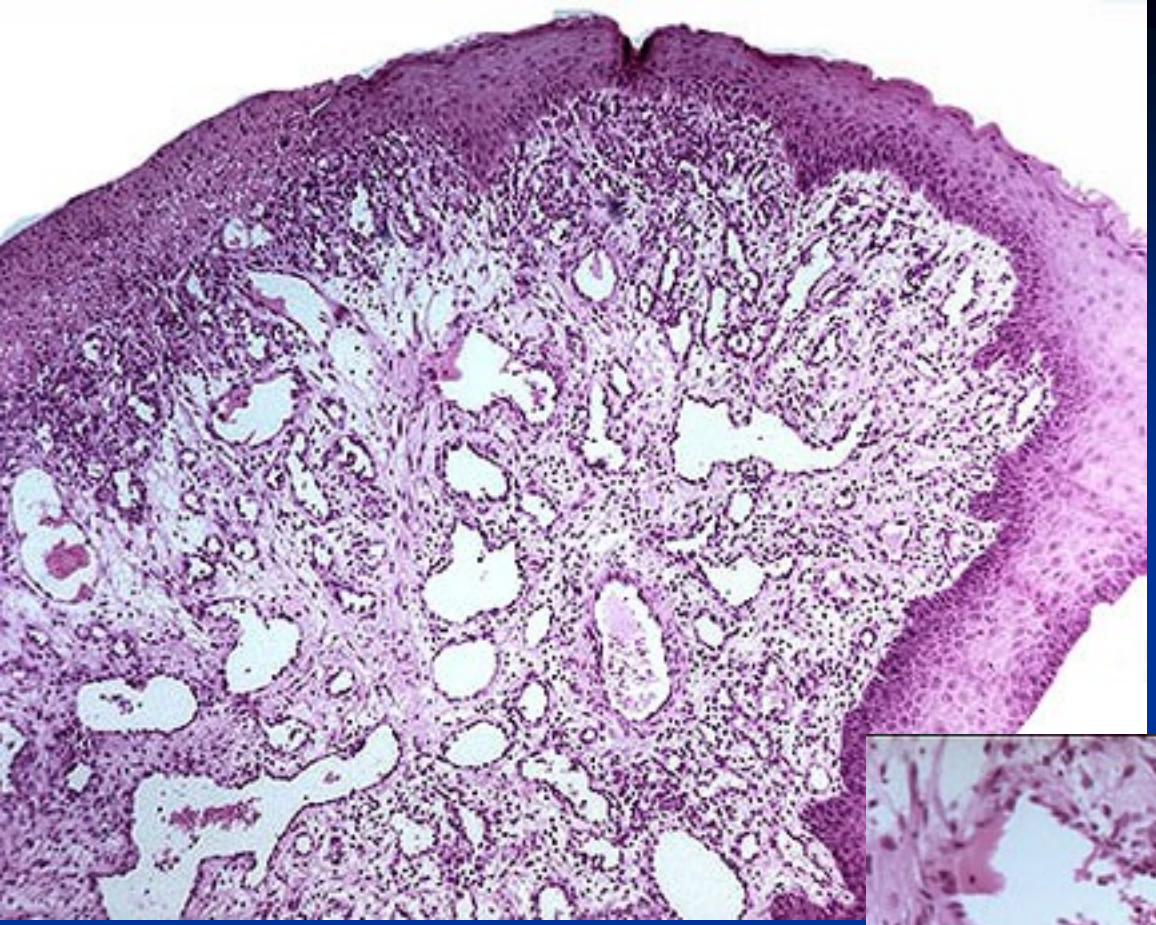
- Mixed inflammatory infiltrate (neutrophils, lymphocytes, macrophages; eosinophils in eosinophilic ulcer)
- Granulation tissue proliferation → possible elevated lesion (nodule) → maturation into connective tissue → fibroepitelial polyp („fibroma“, „epulis“)
- Deep vascular connective tissue

Epulis

- Benign exophytic/polypous lesion situated on the gingiva.
- Reactive, inflammatory
- Peripheral giant cell granuloma: solitary bluish- red, 10-20 mm tumor between or near bicuspids, incisors.

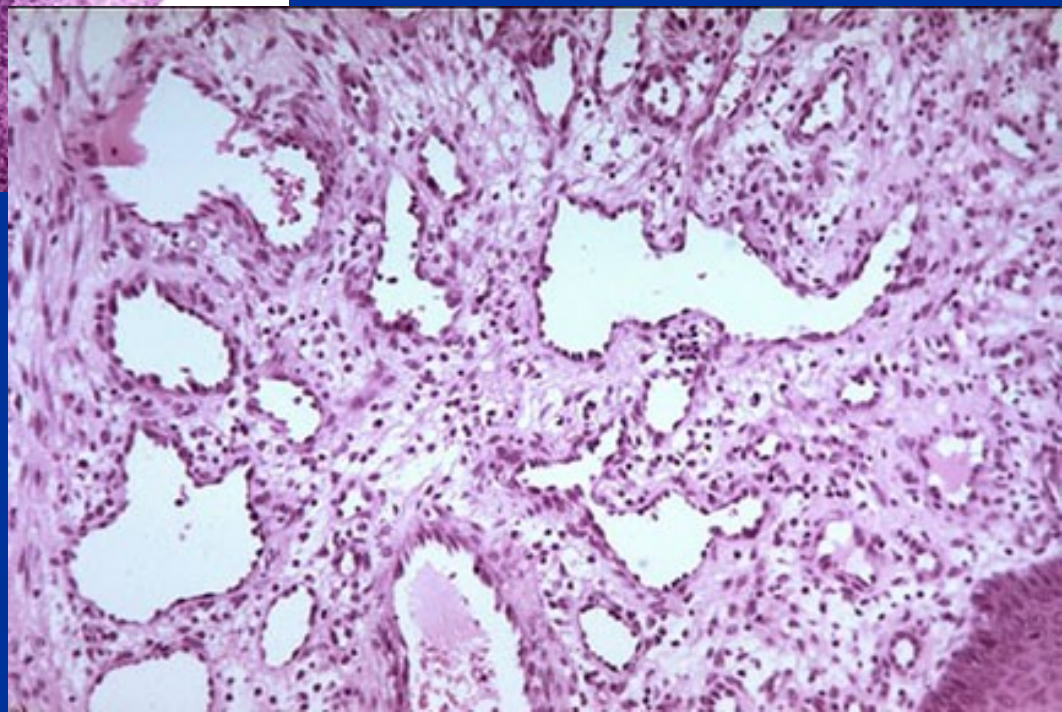
Pyogenic granuloma
(lobular capillary hemangioma)

- Exuberant overgrowth of granulation tissue
- Bleeds easily
- Rapidly growing
- Asymptomatic



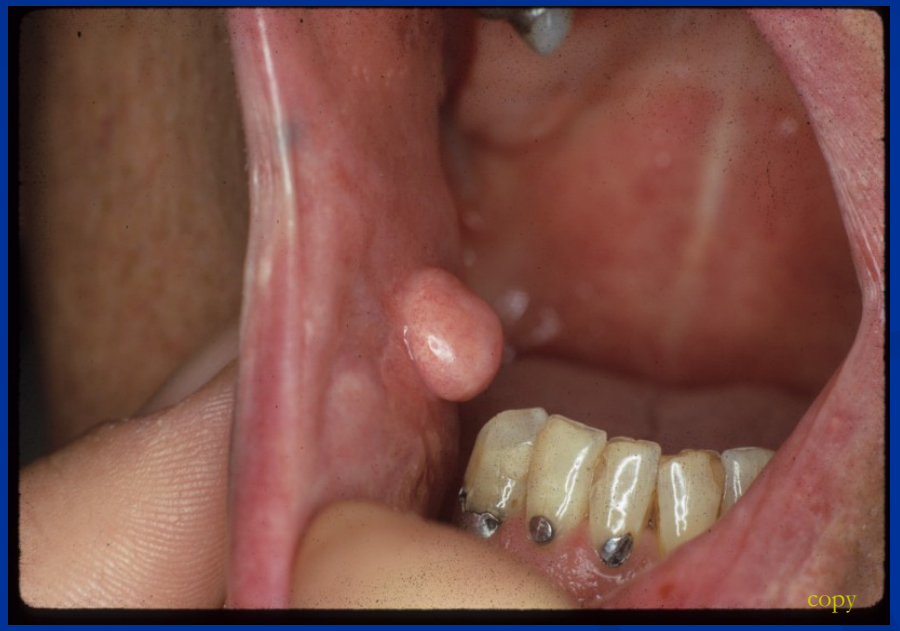
well circumscribed nodule with lobules of dilated and congested capillaries

myxoid stroma and bland endothelial cells



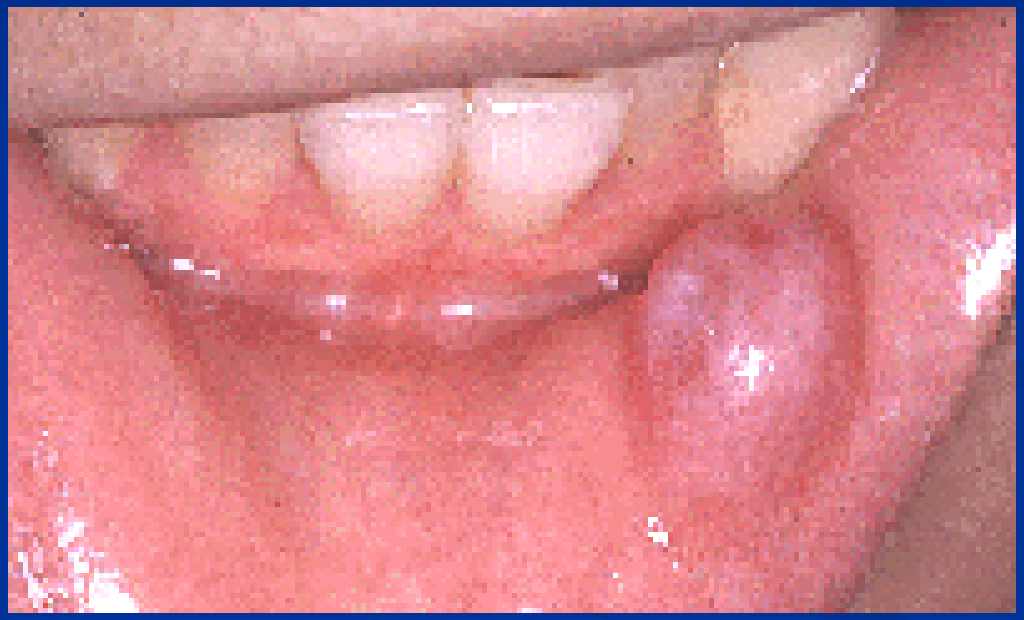
Traumatic fibroma

- Firm, smooth, pink nodule similar in color to surrounding mucosa
- Usually present as late response to trauma
- Usually present for long periods unchanged
- Pseudotumor, histologically fibroepithelial polyp (fibrotic stroma + superficial slightly hyperplastic epithelium)



Mucocele

- Diff. dg.
- Dilatation of salivary gland duct – mucinous cyst +/- reaction
- Result of trauma or obstruction of salivary ducts, usually on the lower lip
- Soft rounded translucent cystic lesion often with a bluish tint.

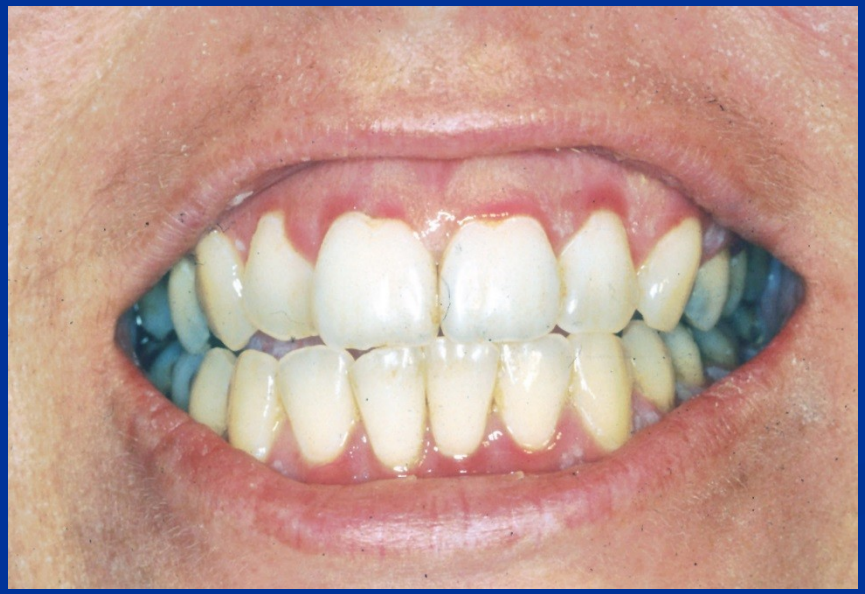


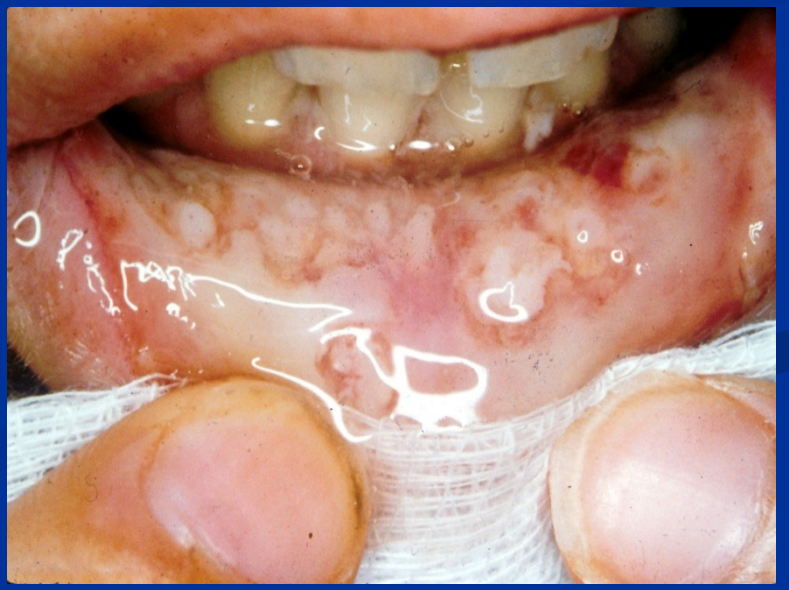
Noninfectious complications of antineoplastic therapy

- Radiation therapy and/or chemotherapy
- **acute changes** – oral mucositis, dermatitis
  haemorrhage (thrombocytopenia)
- **chronic sequelae** – xerostomia, loss of taste, osteoradionecrosis, chronic dermatitis, in children developmental abnormalities

Oral mucositis

- collective consequence of a number of concurrent and sequential biological processes
- Can be the most debilitating of side effects
  - oral and GI
- Ranges from mild inflammation to ulceration

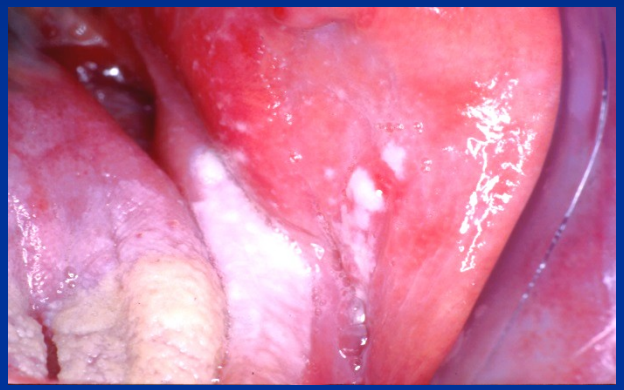




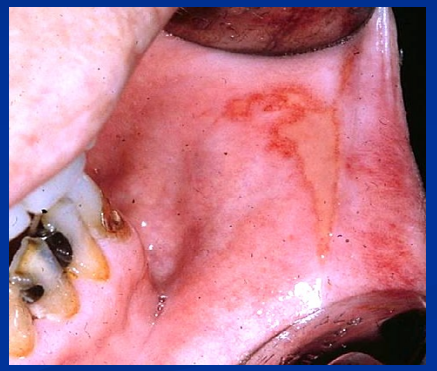
WHO Oral mucositis scale

- Grade 0: No changes

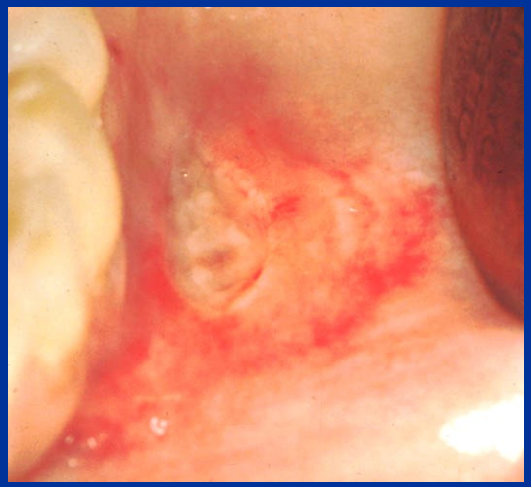
- Grade 1: Soreness/erythema



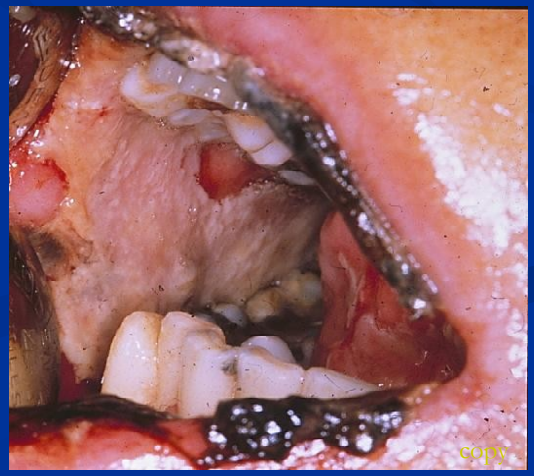
Grade 2: Ulceration/solid foods



- Grade 3: Liquid diet



Grade 4: No alimentation

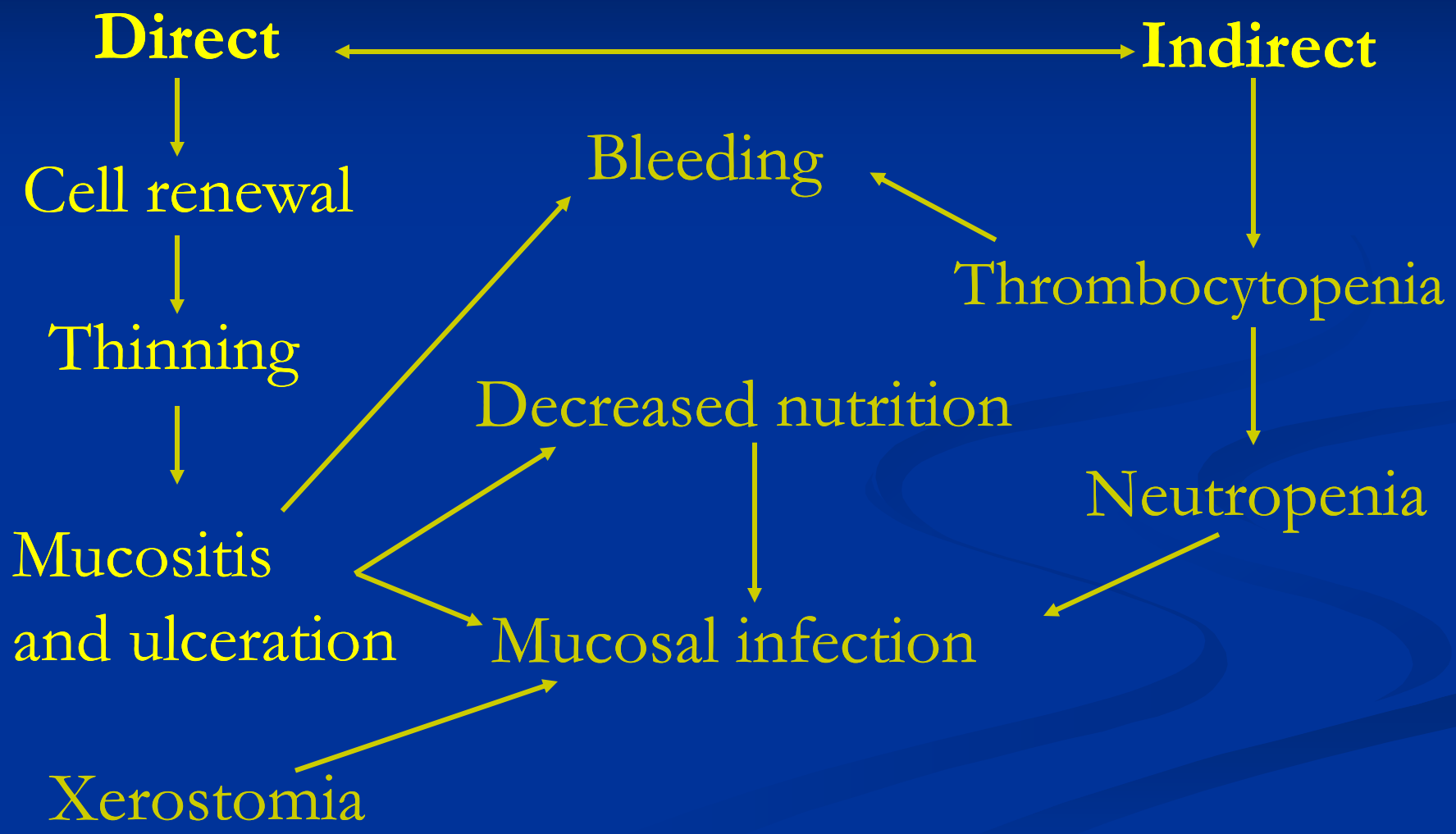


Oral mucositis – incidence

- High dose chemoth. + Stem cell therapy
  - Near 100% for any grade
  - 30-50% Grade 3/4

?

Chemotherapy stomatotoxicity



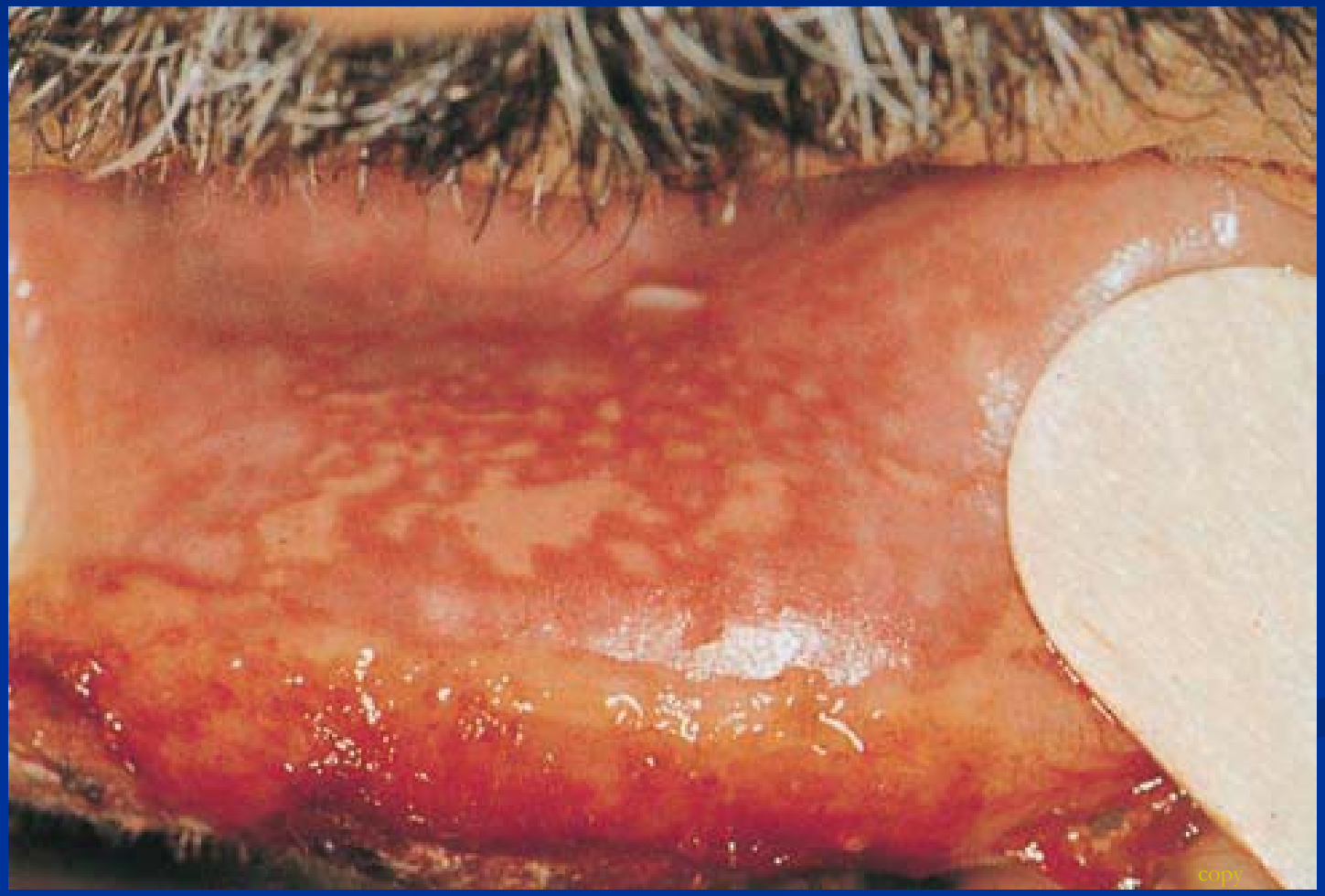
Radiation mucositis

- **Clinical features**:
  - Radiation therapy in excess of 3500 to 4000 rad.
  - Painful diffuse erythema with telangiectasia of the mucosa.
  - Initially red zones → white pseudomembrane at areas of maximal radiation. (+candidiasis?)
  - Ulcers - extremely painful
  - Possible xerostomia

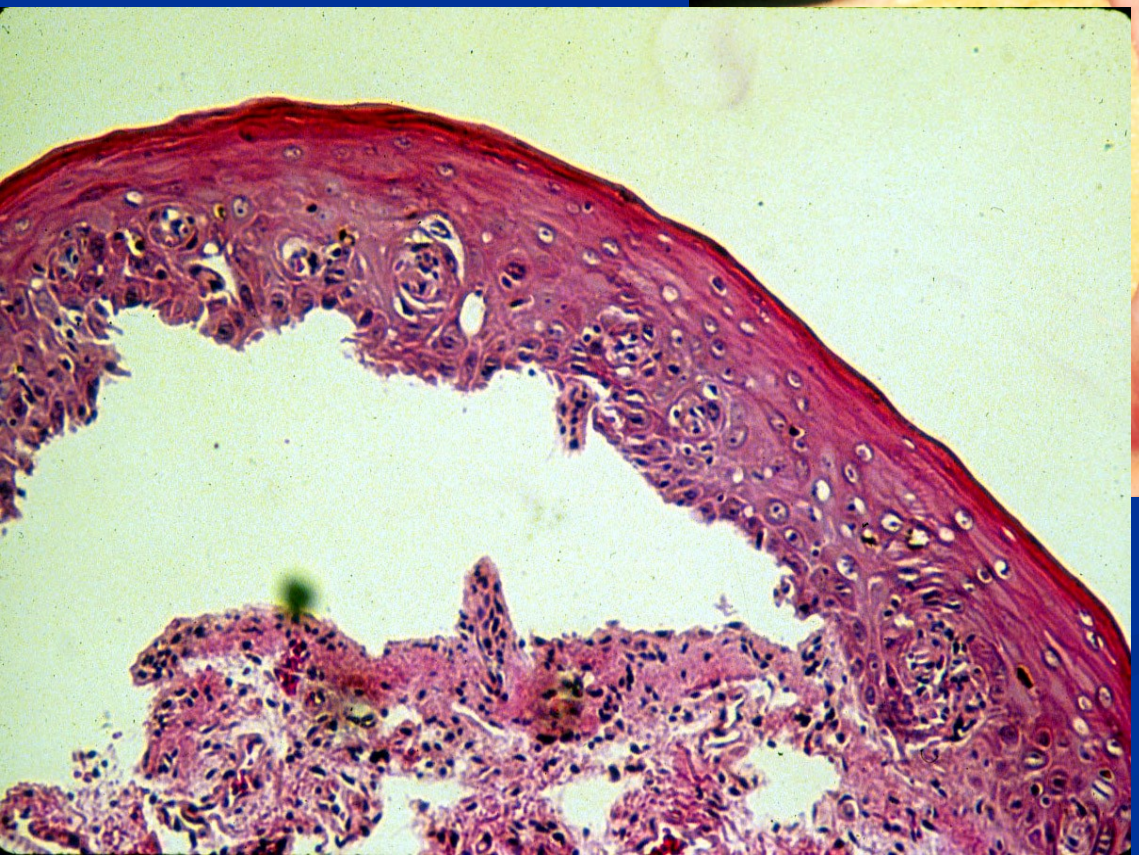
Radiation mucositis

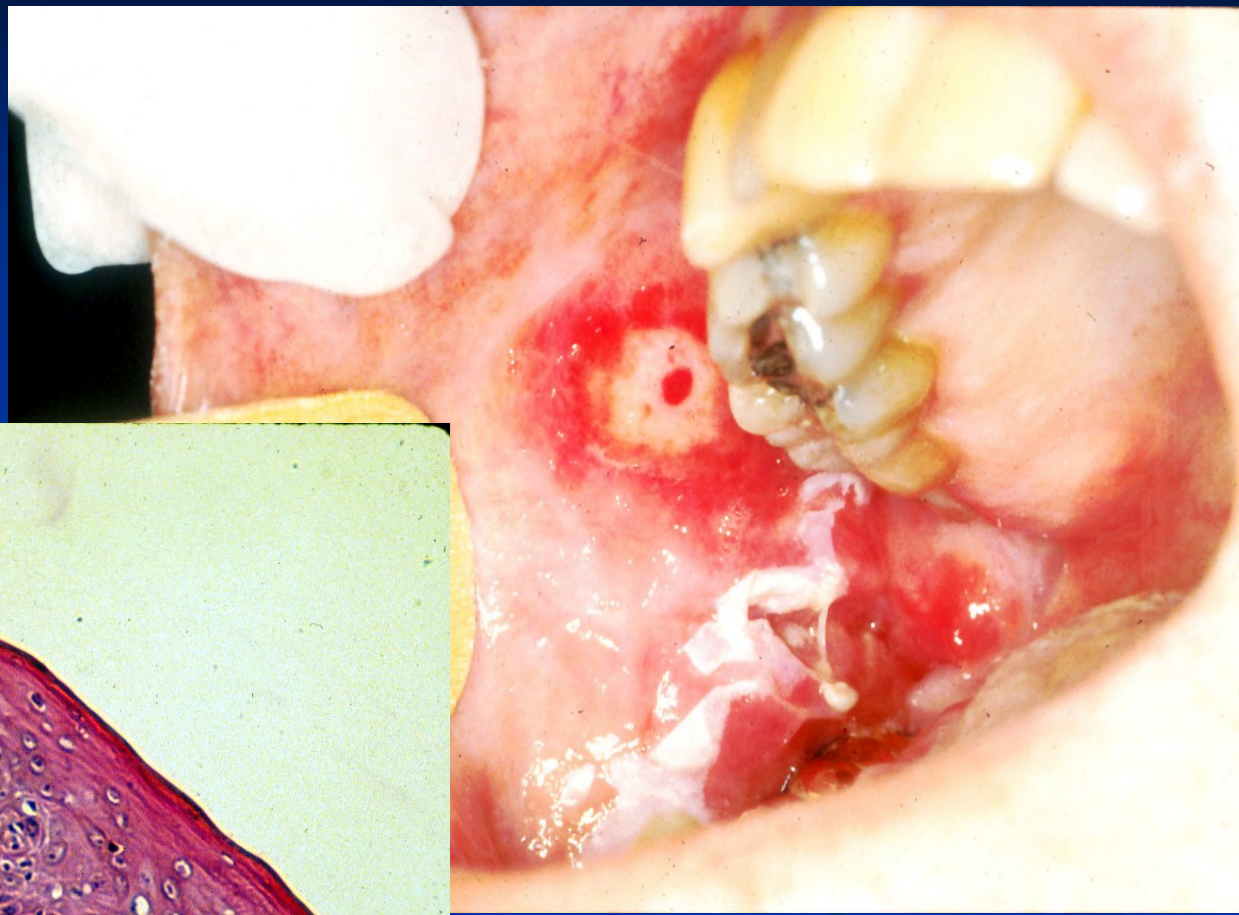
- Dysphagia and oral soreness (maximal 2-4 weeks after radiotherapy but usually subside in further 2-4 weeks)
- Slow/defective healing of the ulcers (inhibition of proliferation by radiation).

Erythema and erosions on the lower lip, caused by ionizing radiation



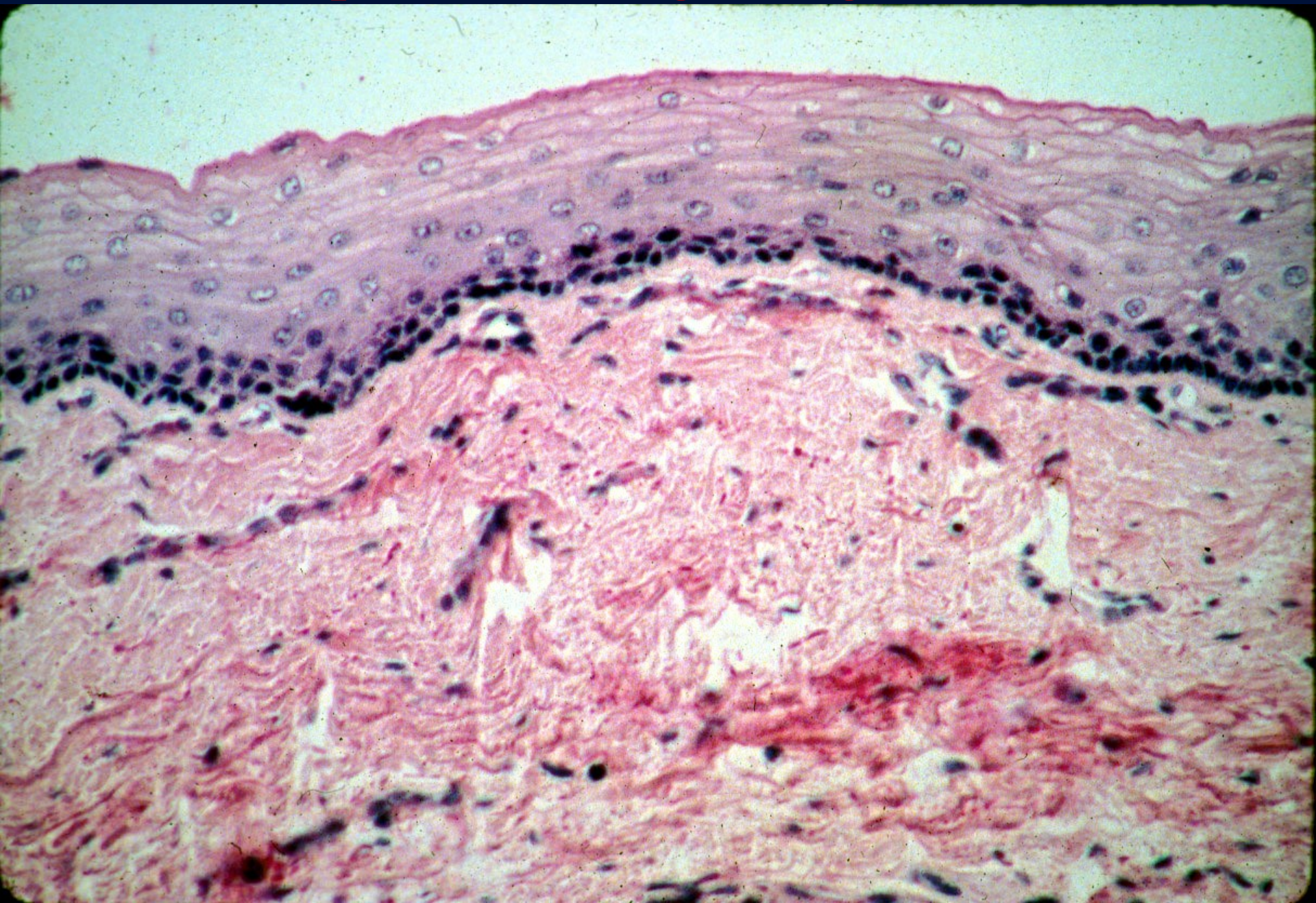
Mucosal sloughing





Diff. dg. x bullous oral lesions (pemphigus etc.)

Atrophy and collagen degeneration

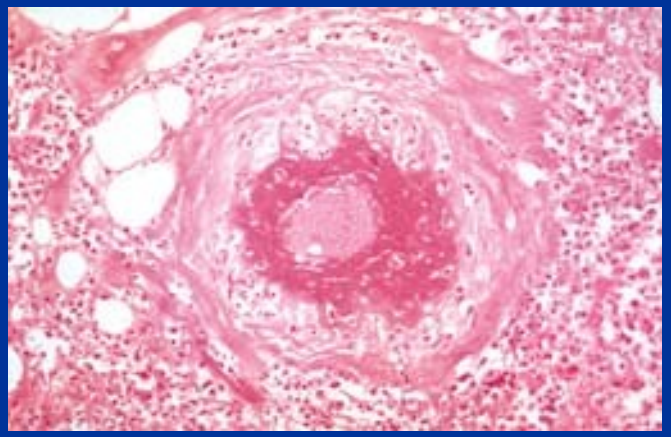


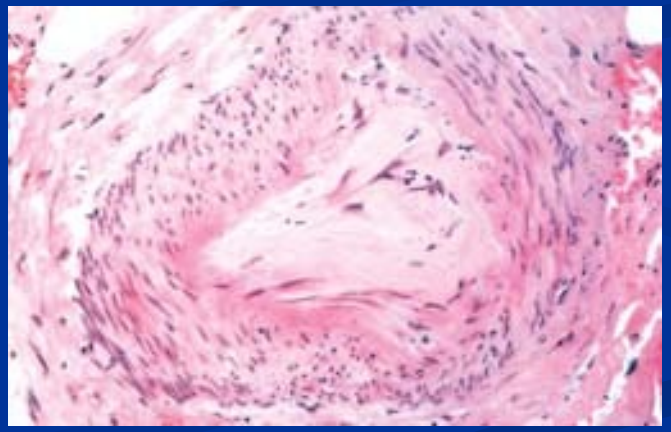
Mucositis complications and sequelae

- Pain
- Oral infection
- Systemic infection?
- Bacteremia/Sepsis
- Oral bleeding
- Xerostomia
- Taste
- Hydration/Nutrition
- Fatigue
- Interrupted cancer treatment

Radiation effects on tissue

- Acute (vasculitis, possibly „fibrinoid" necrosis)
- Chronic (obliterative vasculopathy, fibrosis)

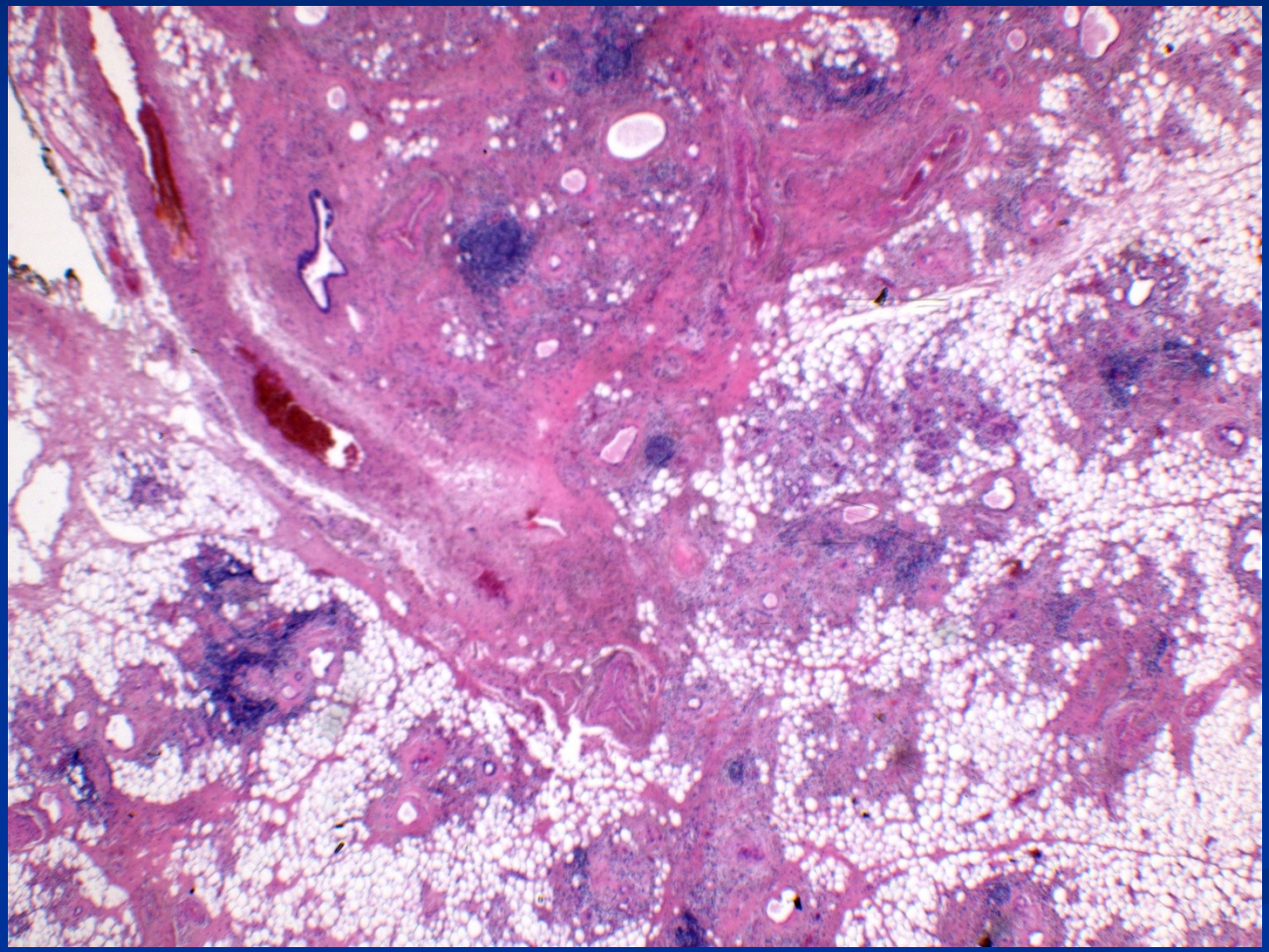




Delayed radiation injury

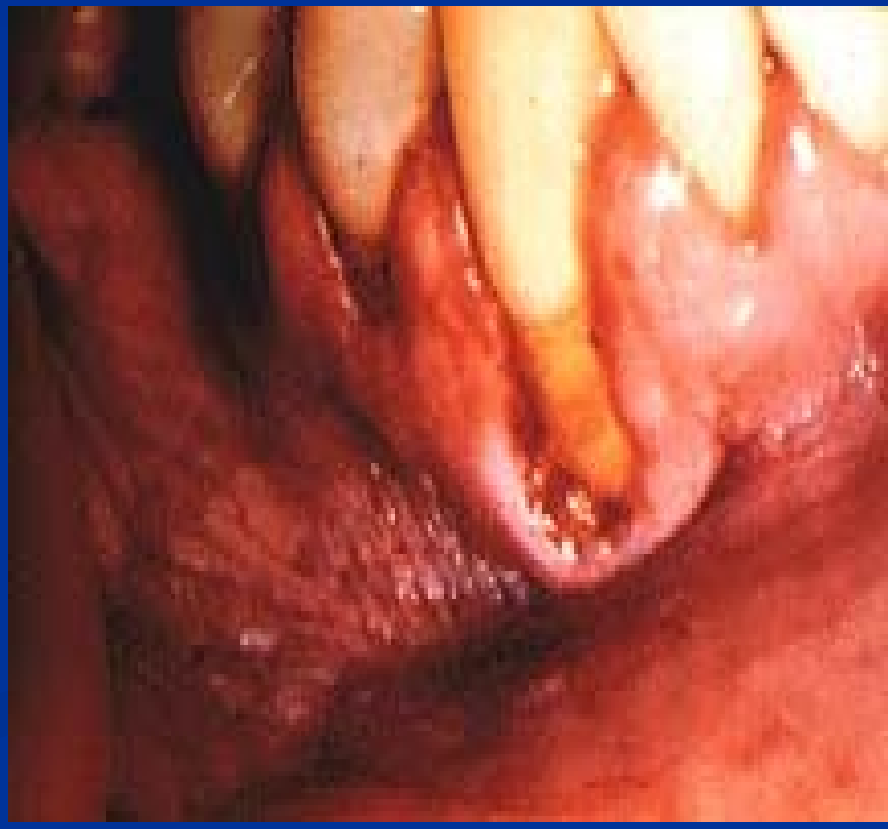
- Carcinogenesis (atom bomb survivors)
  - myeloid leukemias peak 5 to 7 years after exposure
  - breast and thyroid cancers may show greater latency
  - no germline mutations noted in progeny of survivors
- Vascular effects
  - endothelial necrosis followed by intimal and medial fibrosis
  - capillaries may become thrombosed and obliterated or ectatic
- Parenchymal atrophy and fibrosis

Parotid atrophy post RT



Attached gingiva

- Recession of gingival margin
- Loss of attachment
- Tooth abrasion
- Hyperkeratinized soft tissues



Radiation mucositis

- Differential diagnosis:
- History.
- Persistence/increase in size → erythroplakia.
- Treatment:
  - Regresses with time.
  - Persistence/ increase → cytological smears.
  - A soothing mouth rinse, soft diet.
  - Antifungal.

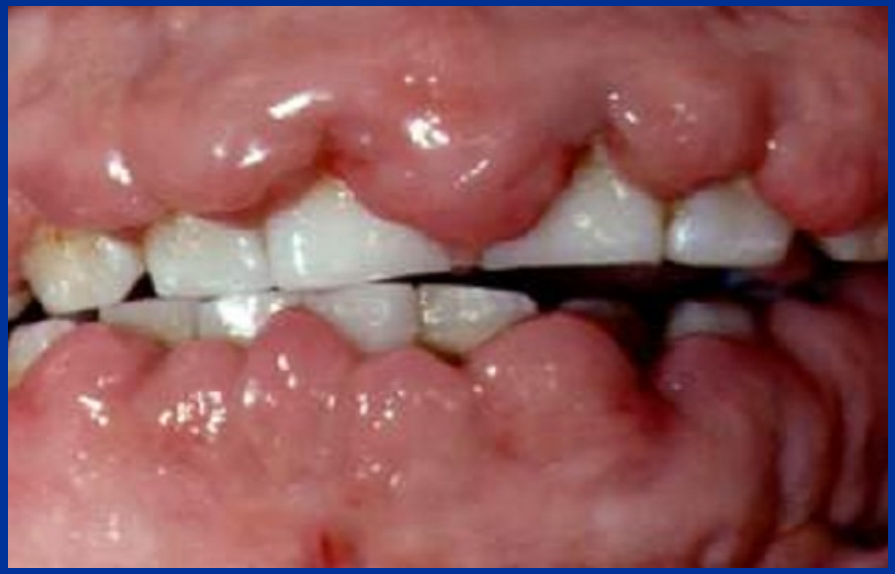
Gingivitis associated with pharmacology

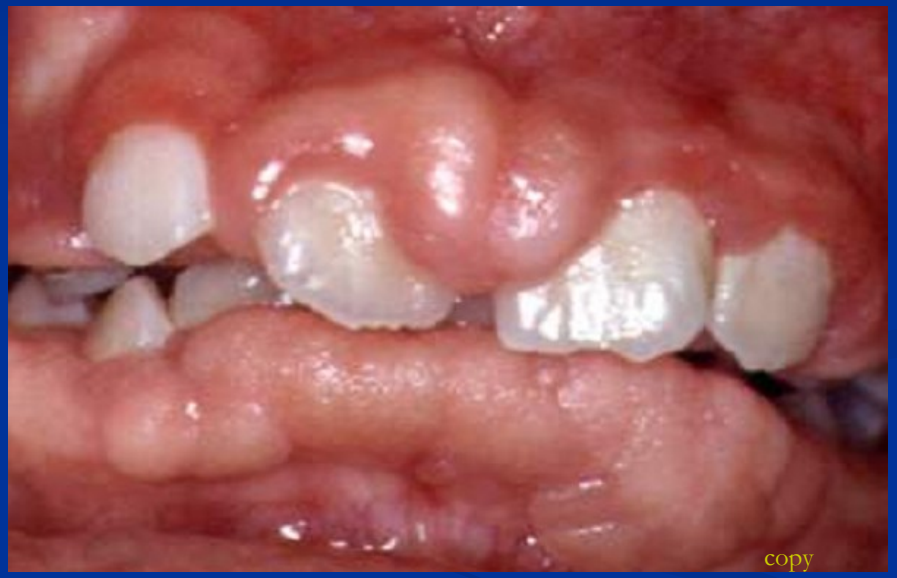
- Drugs known to cause gingival inflammation:
  - Dilantin (Phenytoin)
  - Calcium channel blockers
  - Cyclosporin

Gingivitis associated with pharmacology

- Phenytoin induced **gingival hyperplasia**
  - Leads to pseudopocketing → increased probing depth due to gingival hypertrophy, no due to bone loss
  - Occurs in 3 → 85% of those taking medication
  - Most likely due to increased platelet derived growth factor
- Calcium channel blockers:
  - Causes gingival hypertrophy in 25-50% of those on it
- Cyclosporin:
  - Causes gingival hypertrophy in 30%

Dilantin and cyclosporin induced gingival hypertrophy



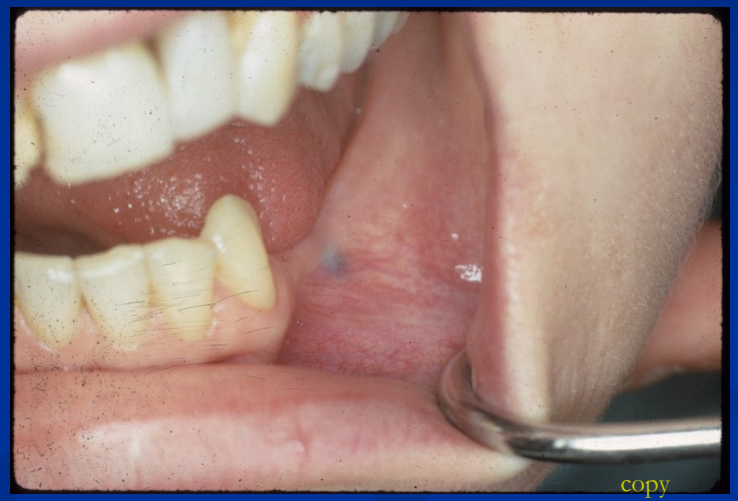


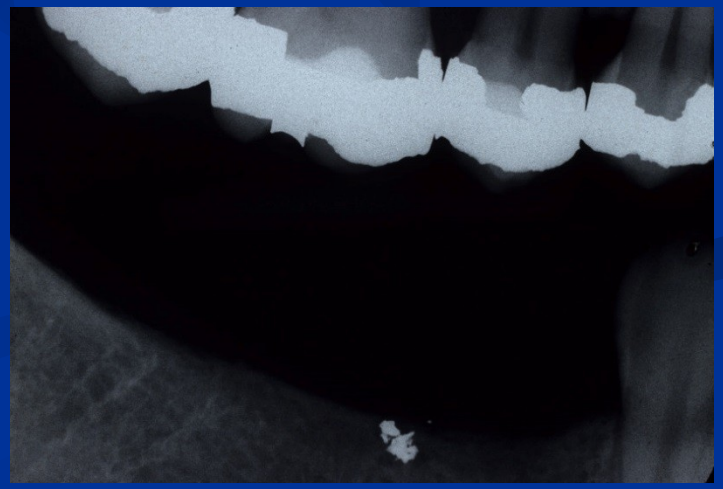
Localized exogenous pigmentations

- Amalgam tattoo
- Intentional intraoral tattoo
- Pigmentation due to systemic metallic intoxication – lead, silver (incl. colloidal), arsenic (drinking water), gold (medication)

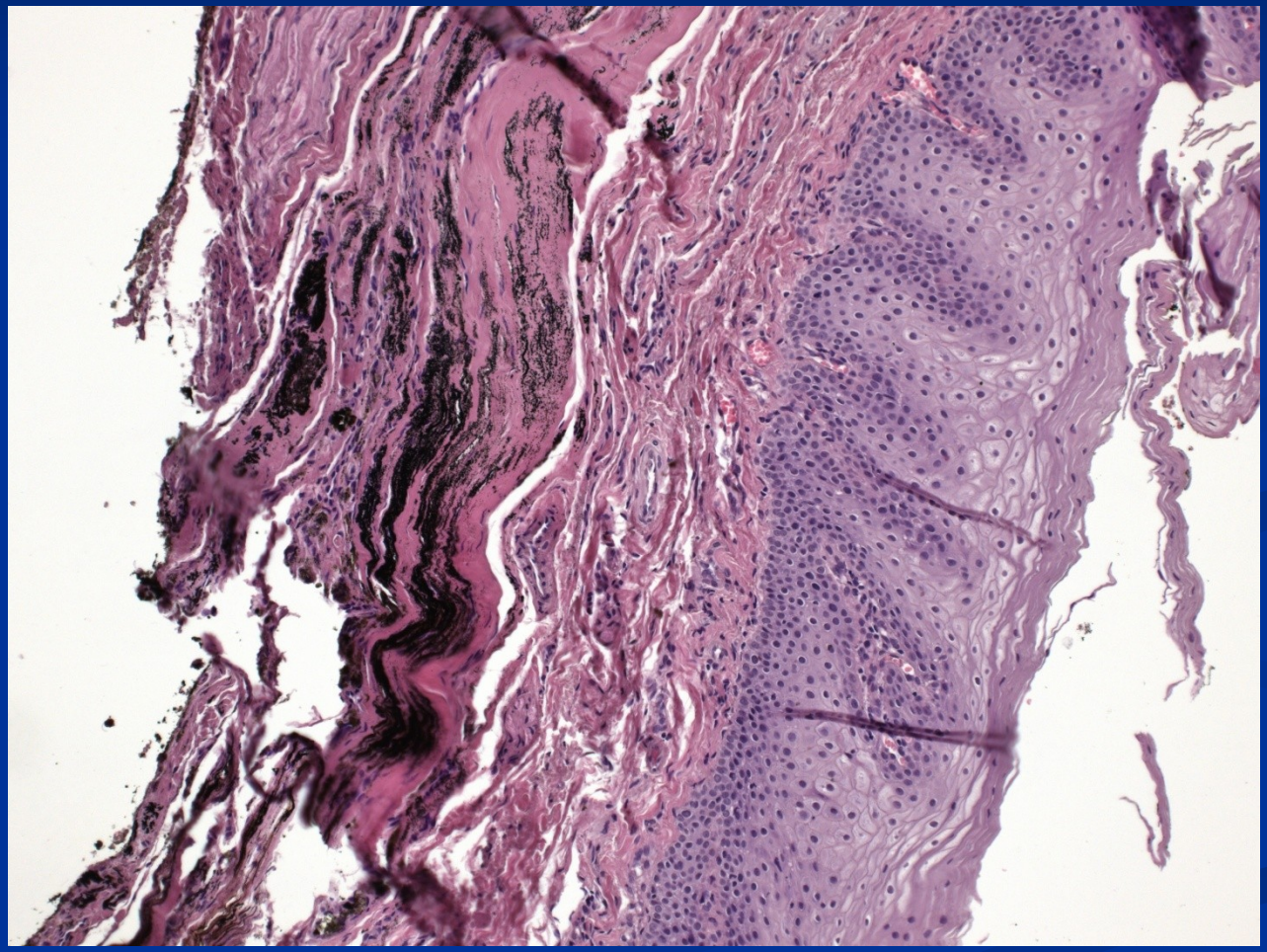
Amalgam tatoo

- A blue or black area usually on the gingival ridge adjacent to a large restoration
- Result of impregnation of amalgam fragment into the tissue





Metallic tatoo



Lead

- Lead: commonly used heavy metal (others: mercury, arsenic, cadmium, …)
- Source of exposure
  - lead paint
  - lead in plumbing (older houses)
  - lead-glazed ceramics
  - industrial exposure
- Route of exposure
  - inhalation with industrial exposure
  - ingestion with household exposure

Lead Lines