Bacterial, protozoal infections

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FLORA of the ORAL CAVITY

- Mixed flora incl. fungal
- *Streptococcus, Neisseria, Staphyloccoccus, Lactobaccillus, Actinomyces, Bacteroides.*
- In the epidermal layer of the cheeks, gingiva, and on the surface of teeth.
- Found in saliva in large numbers

Host defense mechanisms

- **Competitive suppression** of potential pathogens by low-virulent microorganisms
- **Nonspecific defenses** incl. antibacterial saliva, humoral (secretory IgA), and cellular (submucosal lymphocytes + plasma cells) systems.
- Phagocytosis - important process
- Specific immune responses based on antibodies and specific reactions of T lymphocytes

Morphologic patterns of inflammation

- alterative
- exsudative
  - serous
  - fibrinous, pseudomembranous
  - suppurative
  - necrotizing, gangrenous
- proliferative
  - primary (rare) x secondary

Granulomatous inflammation

Morphologic patterns of inflammation

- serous - excessive accumulation of fluid, few proteins - blister, on serous membranes commonly initial phases of inflammation
- modification - catarrhal - accumulation of mucus (salivary glands)

Morphologic patterns of inflammation

- fibrinous - higher vascular permeability - exsudation of fibrinogen → fibrin
- fibrinolysis → resolution; organization by granulation tissue → fibrosis → scar
- pseudomembranous - fibrinous pseudomembrane (diphtheria - Corynebacterium) - fibrin, necrotic mucosa, etiologic agent, neutrophiles

Morphologic patterns of inflammation

- suppurative (purulent) - accumulation of neutrophillic leucocytes - formation of pus (pyogenic bacteria)
- interstitial
  - phlegmone – diffuse in soft tissue
  - abscess - localized collection
    - acute – border – surrounding tissue
    - chronic – border - pyogenic membrane
    - pseudoabscess – pus in lumen of hollow organ (dilated salivary gland duct)
- local complications, i.e. local spread, formation of suppurative fistule

Morphologic patterns of inflammation

- systemic complications of suppurative inflammation:
- bacteremia (no clinical symptoms!; danger of formation of secondary foci of inflamm. (endocarditis, meningitis)
- sepsis (= massive bacteremia + toxins) - septic fever, activation of the spleen, septic shock
- thrombophlebitis - secondary inflammation of the wall of a vein with subsequent thrombosis - embolization - pyemia - hematogenous abscesses (infected infarctions)
- lymphangiitis, lymphadenitis

Morphologic patterns of inflammation

- necrotizing - inflammatory necrosis of the surface - ulcer (skin, oral mucosa) – necrotizing ulcerative gingivitis, noma
  - gangrenous - secondary modification of a necrotic focus by bacteria - humid gangrene (debilitated patients)

Infections in stomatology

- Skin
- Oral mucosa
- Pharynx incl. tonsils
- Sinuses
- Salivary glands
- Teeth + surrounding structures
- Deep infections (muscle, bone, …)

Infections in stomatology

Bacterial - nonspecific

- Skin infections – impetigo, erysipelas, etc.
- Pharyngitis, tonsillitis
- Scarlet fever
- Diphtheria
- Gonorrhea
- Necrotizing ulcerative gingivitis
- Noma

Infections in stomatology

Bacterial – specific patterns

- Syphilis
- Tuberculosis
- Leprosy
- Actinomycosis
- Cat-scratch disease

Infections in stomatology

- **Fungal** – i.e. superficial pseudomembranous oral candidiasis
- **Viral** – i. e. herpetic stomatitis (HSV-1, less common HSV-2), vesicles → ulcers; herpes zoster; EBV, CMV, measles
- **Parasitic** – i. e. protozoa (toxoplasmosis)

- Sialoadenitis – non-purulent viral (mumps); purulent bacterial (Stph. aureus, Str. viridans)

Infections in stomatology

Pyogenic bacteria

- Streptococcus pyogenes
- Staphylococcus aureus
- Streptococcus pneumoniae
- Klebsiella pneumoniae
- others

Streptococci

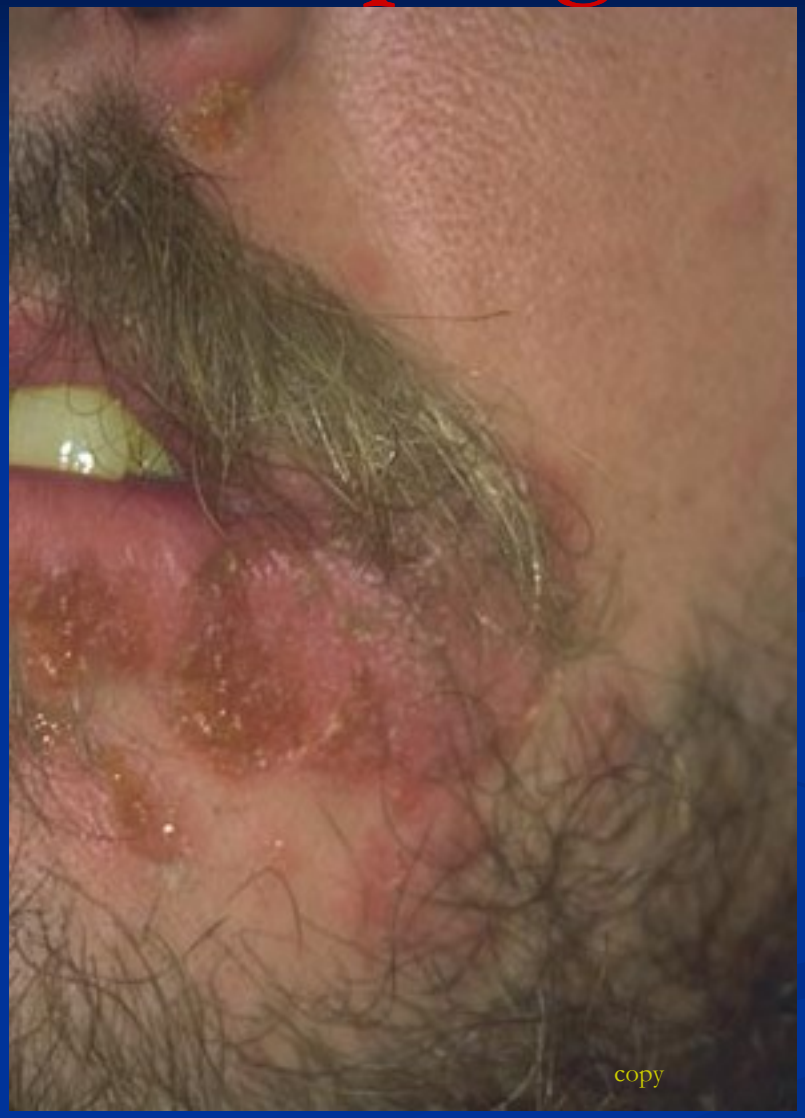
Str. pyogenes

- local inf. – phlegmona, impetigo, wound inf.
- erysipelas
  - skin erythema (lower limbs, face) + toxemia
  - lymphatic + blood vein thrombosis → lymphostasis → edema → elephantiasis
- angina (tonsillitis) → otitis, sinusitis
- scarlet fever (erythrogenic toxin)
  - angina + oral enanthema (raspberry tongue) + skin exanthema (face, trunk)

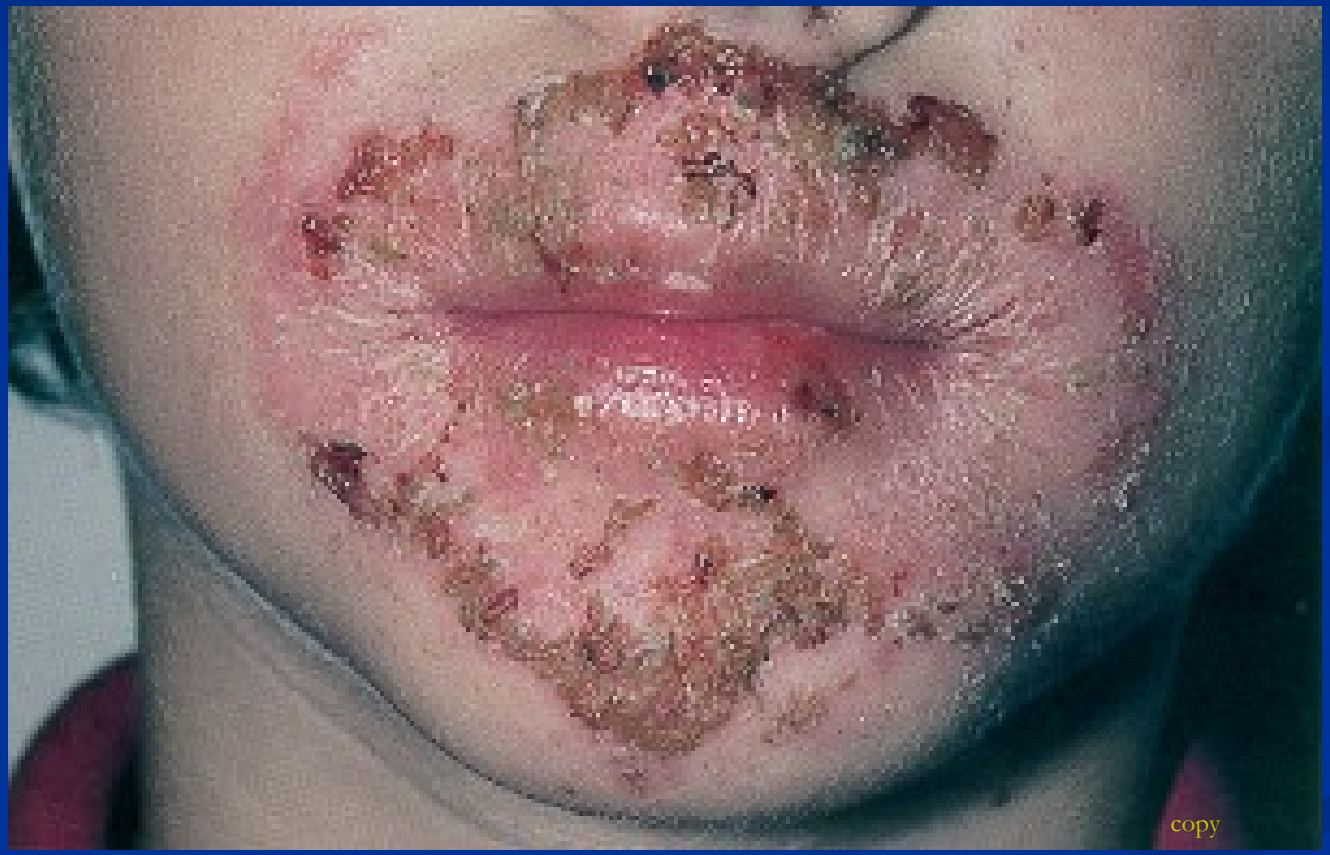
Impetigo

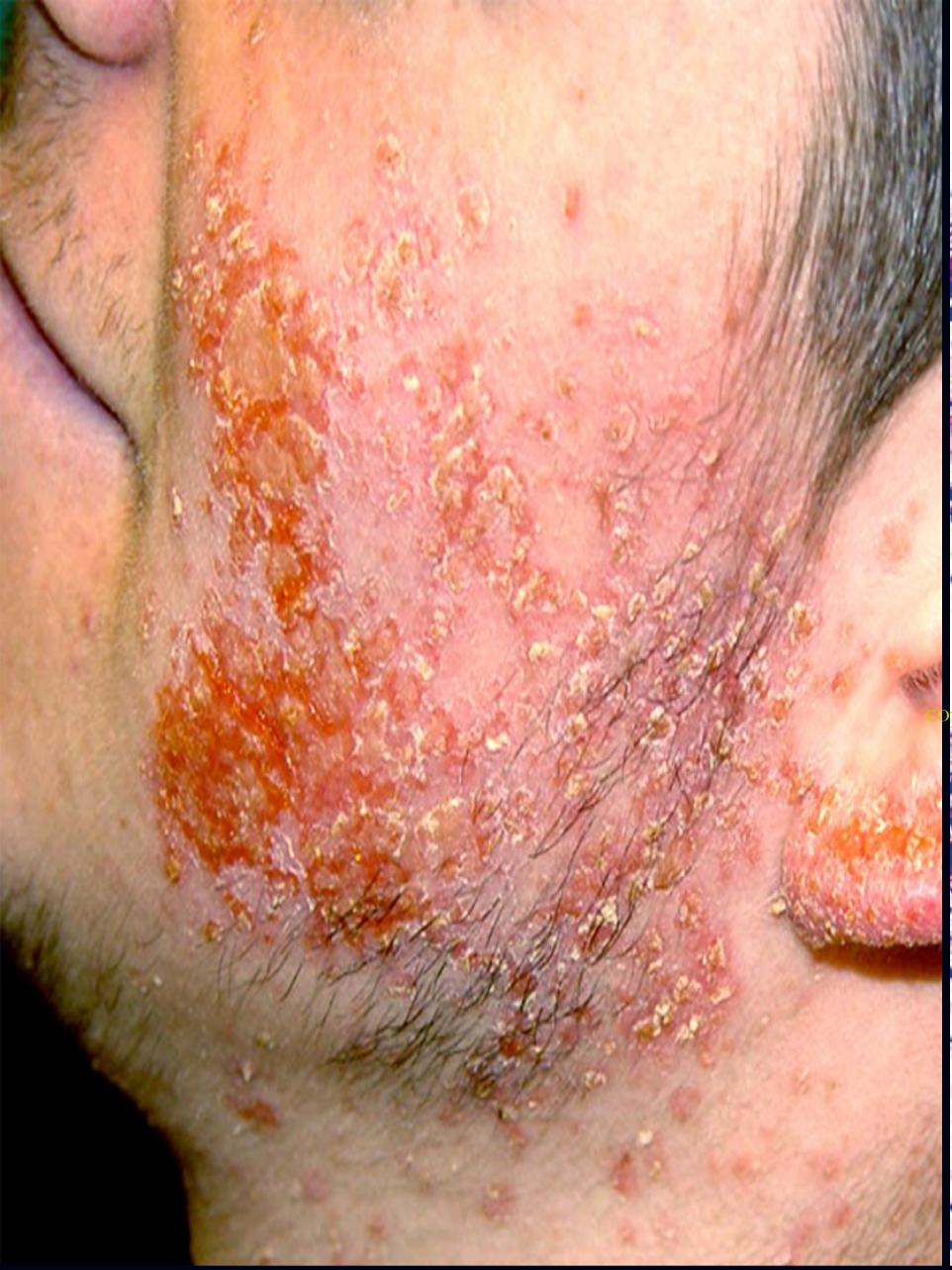
- superficial skin infection (commonly face)
- Str. /+ Staph.
- in damaged skin
- contact transmission, possible epidemics in children
- vesicles/bullae → pale brown crusts
- usually no systemic manifestations

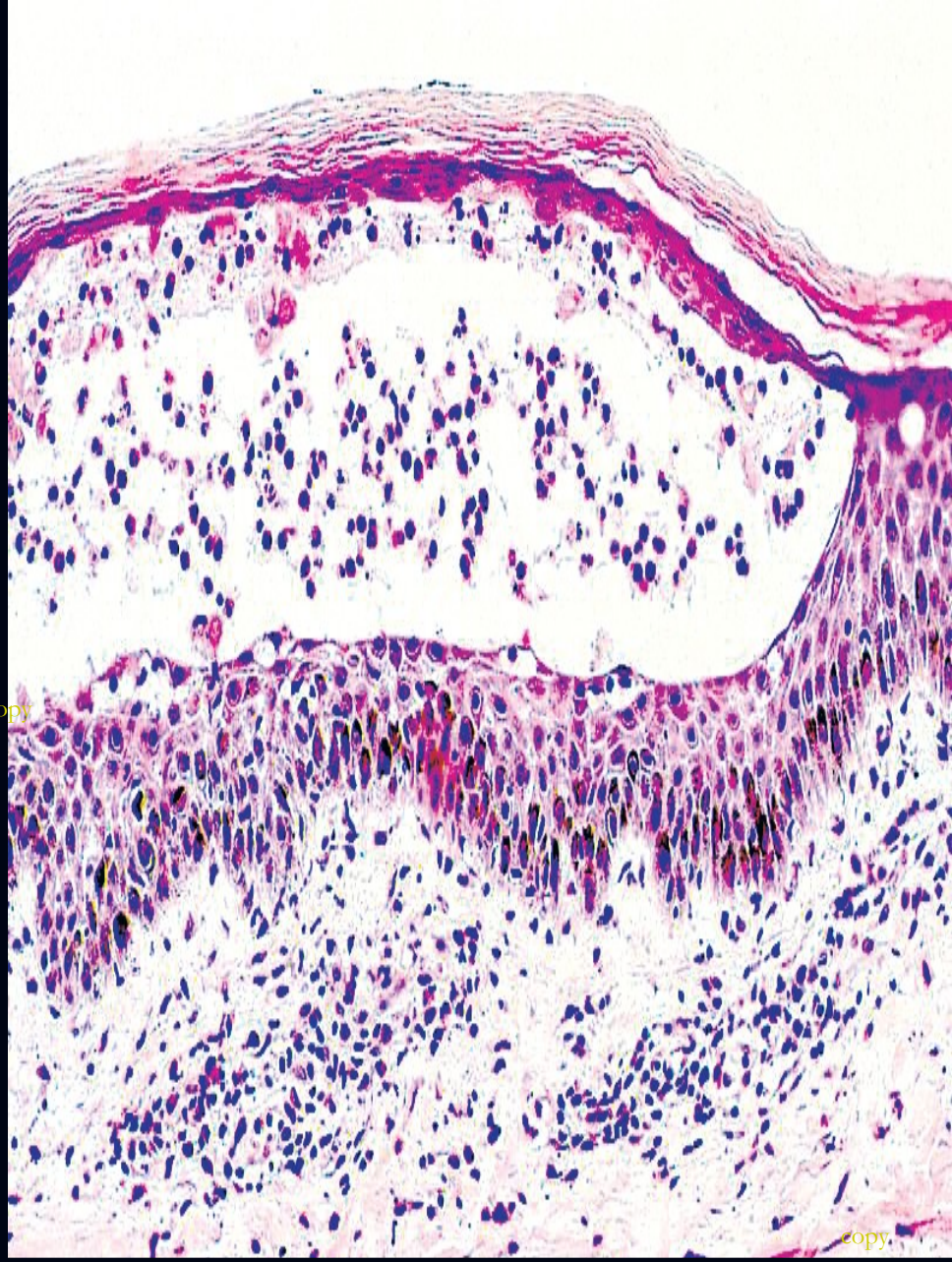
Impetigo



Impetigo







Bullous impetigo

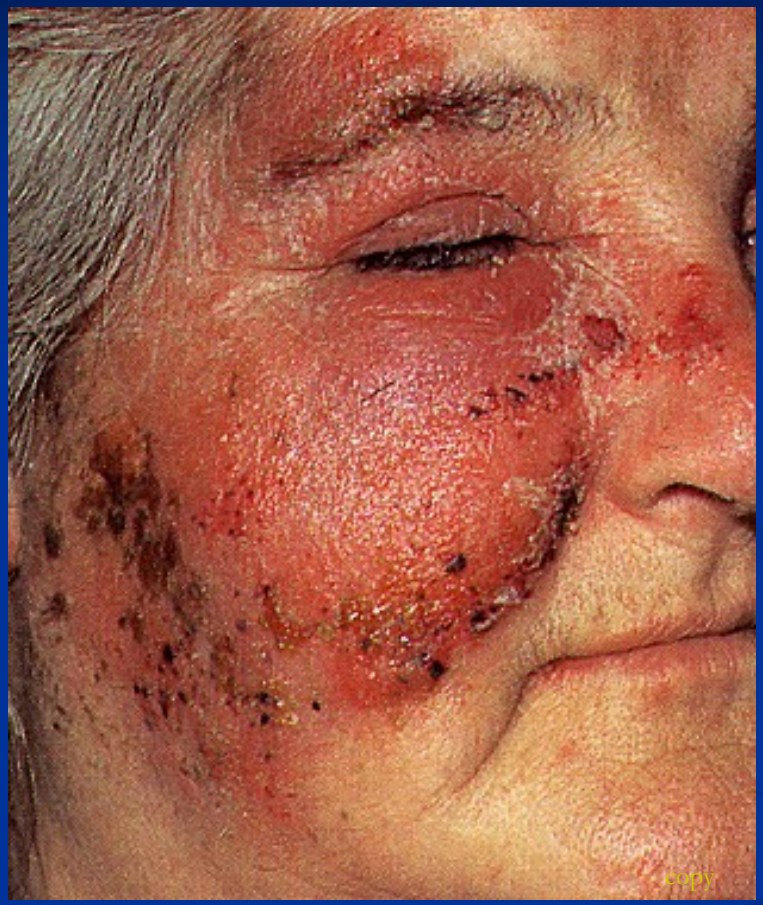
Erysipelas

- Skin + soft tissue purulent infection (cellulitis), phlegmone + local lymphatic spread, commonly bacteremia + systemic signs (fever, vomiting…)
- Usually β-hemolytic streptococci
- Children, elderly, debilitated, diabetics
- Painful, swollen, red, warm foci
- Complications – abscess, gangrene, thrombophlebitis, shock, distant streptococcal sequelae (endocarditis, glomerulonephritis)
- Possible recurrence

Erysipelas

- Well-demarcated cellulitis with fever and malaise
  - upper dermal edema lifts epidermis except fixed foci of hair follicles or sweat glands
  - leads to the typical „peau d'orange" appearance

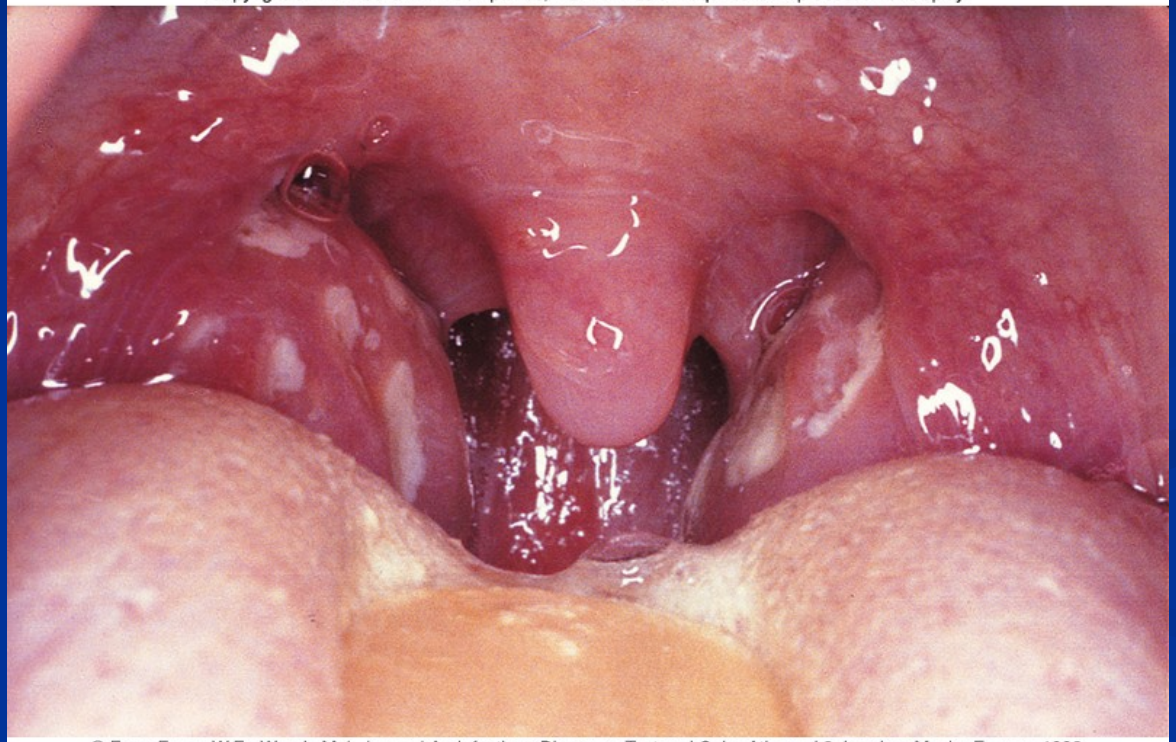
Erysipelas



Tonsillitis and pharyngitis

- bacterial (Str. – 25%, Staph., Fusobacterium, dipthteria, …)
- viral (EBV, influenza, adenoviruses, …)
- Clinical – sore throat, dysphagia, red + swollen tonsils + focal/confluent yellowish exudate, cervical lymhadenopathy, fever, malaise, …
- In viral + rhinitis, laryngitis

Tonsillitis



Scarlet fever

- Hemolytic streptococcus B group A
- Systemic bacterial infection, result of an erythrogenic toxin → capillary damage
- Most common in children
- Complication: local spread (otitis media, abscess)

systemic spread (pneumonia, septicemia, toxic shock syndrome);

poststreptococcal heart, kidney and joints diseases

Scarlet fever

- Incubation period: 2-3days (1-7days)
- Usual type:

Fever: 39°C, 1 week

Vascular dilation and damage with an erythematous macular rash on the skin (chest area), after 1 week desquamation.

Face →flushed except for zone of circumoral pallor

Pharyngitis, tonsillitis: red enanthema, edema, yellow exudate

Cervical lymphadenitis

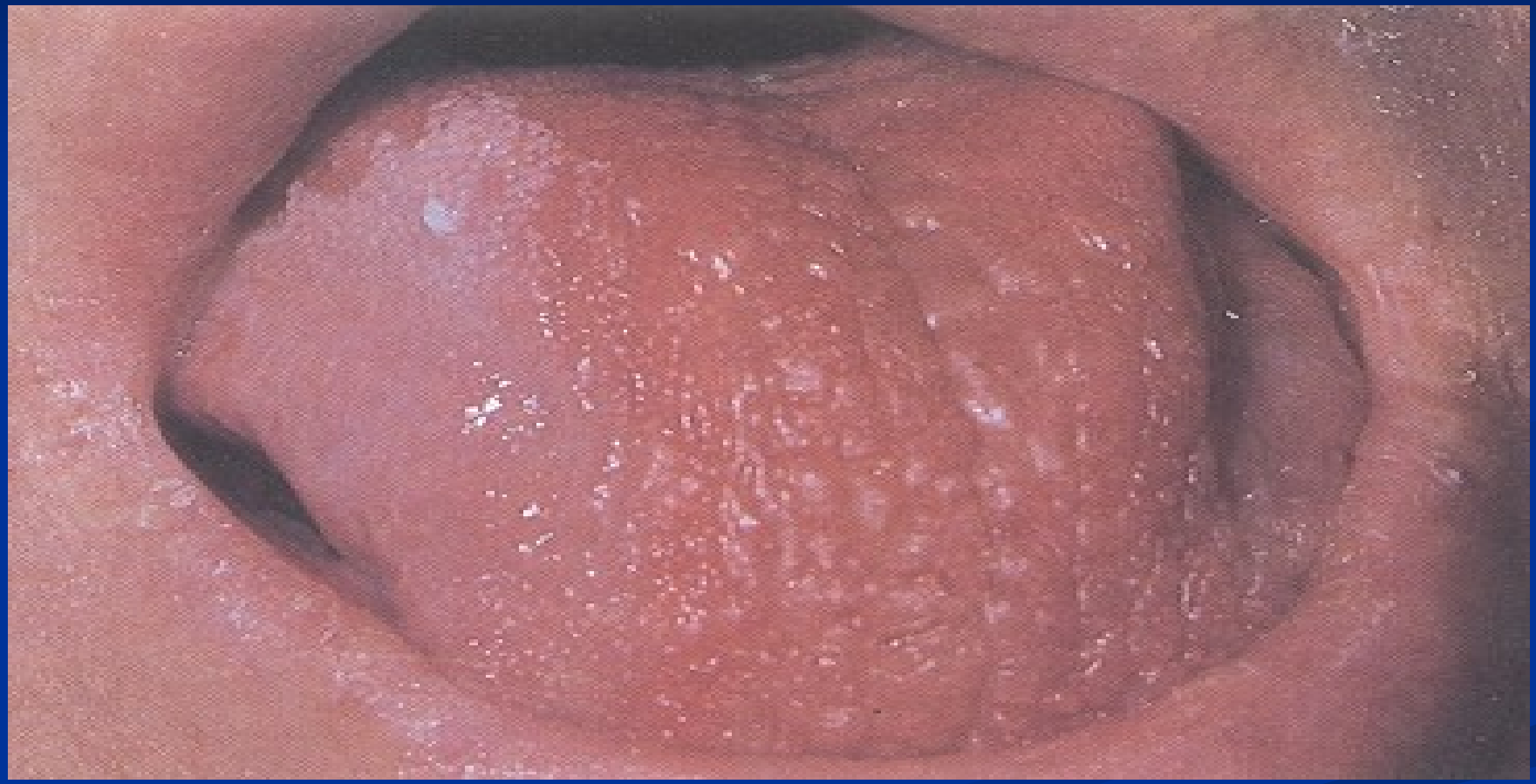
Scarlet fever

Tongue: start with white coating + visible fungiform papillae – white strawberry tongue

4.-5. day – desquamation, red strawberry tongue

Soft palate: possible petechiae

Scarlet fever



Diphtheria

- *Corynebacterium diphtheriae*
- mostly children
- outbreaks in urban poor populations, developing countries + native populations, immigrants
- in immunosuppressed
- without booster vaccination
- epidemics still possible

Diphtheria

- **Pathology**
  Pseudomembranes cover the mucosal membranes (nose, tonsils, oropharynx, larynx, genital), adherent to the tissue, bleeding by removal attempt. Progression to necrosis possible.
- Damage by exotoxins to heart muscle, liver, kidneys, and adrenals. Also nerve damage resulting in paralysis of the soft palate, eye muscles or extermities.

Diphtheria

- **Clinical findings**

  Fever, sore throat, dyspnea (obstruction by the membrane). Later on difficulties with vision, speech, swallowing, or movement of the arms or legs. Var. gravis more severe.

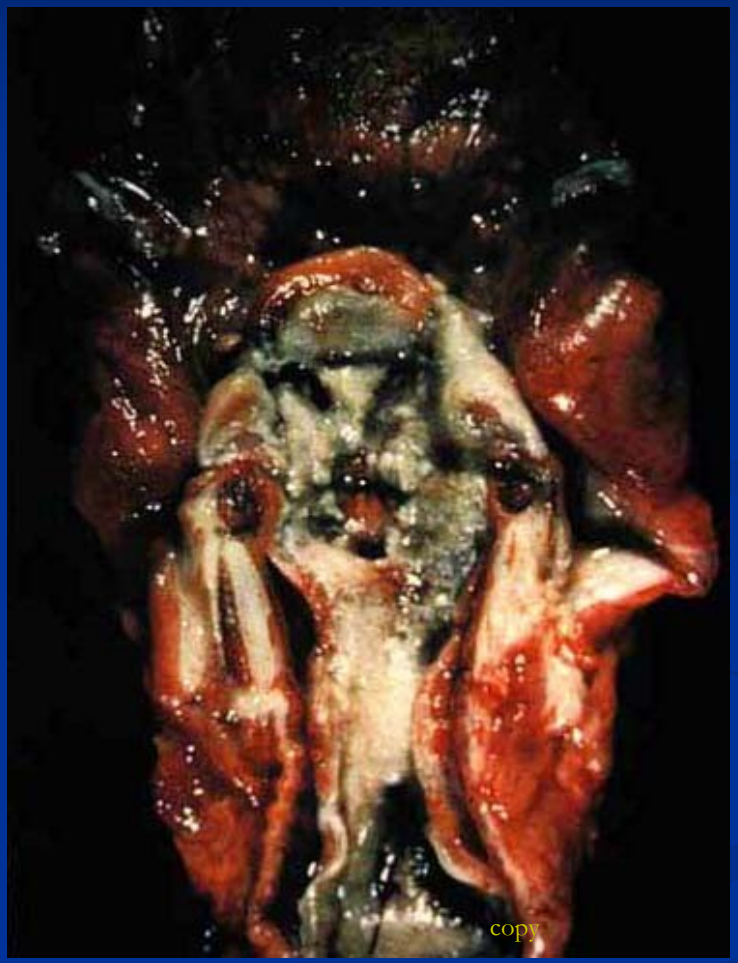
Pharyngeal diphtheria

- The most common type, >80%.
- Sites of infection: tonsils, pharynx.
- Symptoms: malaise, sore throat, anorexia, vomiting and middle-grade fever.
- Usually +/- systemic absorption of toxin.
- With enlarged lymph nodes in the submandibular areas of neck.

Pharyngeal diphtheria

- **Ordinary type**
  - Within 2-3 days, small patches of white pseudomembrane on the tonsils
  - Typical adherent, bluish- or greyish-white pseudomembrane forms on the congested tonsils.

Diphtheria



Diphtheric pseudomembrane

Pharyngeal diphtheria

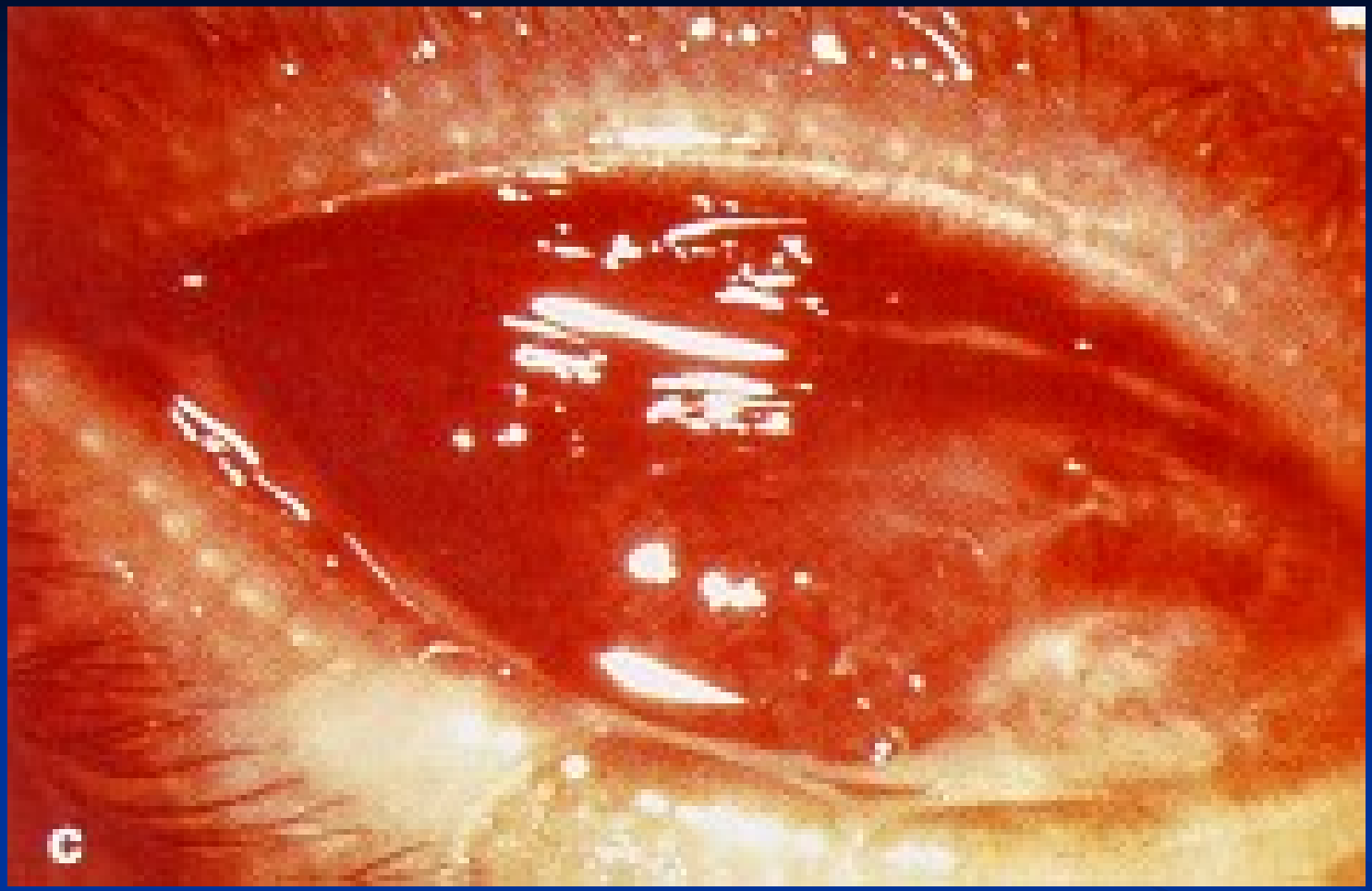
- **Grave type**
  - Serious early symptoms, high-grade fever.
  - Large, thick pseudomembrane, greyish-green or black (if bleeding), covering the tonsils, uvula, and some soft palate, odoriferous in mouth
  - Skin becomes pale, tachycardia, blood pressure may be normal or slightly depressed (shock).

Differential diagnosis

- **Streptococcal pharyngitis**
  - The pus covering the tonsils (yellow color, easy to remove) x the pseudomembrane of diphtheria.
- **Oral candidiasis**
  - often in infants, in good general conditions. The membrane white, and easily removable
- **Infectious mononucleosis and Vincent's angina**
  - Possible pseudomembrane-like covering on the surface of tonsils or pharynx, removable without bleeding.

Gonorrhea

- sexually transmitted acute mucosal purulent inflammation (anogenital region, internal genital in females)
- in 20% + oropharyngeal region (direct mucosal contact, rarely due to septicemia)
- pharynx, tonsils, uvula – erythema, edema, possible pustules
- anterior oral cavity – erythema, possible → ulceration
- gonococcal ophthalmia neonatorum



Ophtalmia neonatorum caused by *Neisseria gonorrheae*
Source: Microbiology Perspectives, 1999

Tissue space infections

- source from apical abscess, pericoronitis
- extension along the planes of muscles/fascia
- accumulation of exudate/pus
- disruption of blood supply, anaerobic space
- variable localization of facial cellulitis

Tissue space infections

Facial cellulitis - flegmona

- commonly from molars
- diffuse edema (hard consistency)
- pain
- systemic signs (fever, leucocytosis, toxemia)
- tender enlargement of cervical LN
- possible fatal complications
  - laryngeal edema – glottis
  - mediastinitis
  - extension to carotid artery

Tissue space infections

Cavernous sinus thrombosis

- possibly fatal complication
- source – upper teeth, sinusitis, skin abscess
- retrograde venous blood flow
- cyanosis + edema of the eyelid
- limited eye movements, pain
- headache, vomiting, fever
- fatal without prolonged antibiotic therapy
- rapid progession

Oral ulcerative lesions

- Acute (traumatic, infectious, drug reactions, immunologically mediated)
- Chronic (vesiculobullous lesions, malignancy)
- Recurrent (rec. aphthous stomatitis, etc.)

Acute ulcerative lesions

- Drug reactions

  Barbiturates, salicylates, phenolphthalein, quinine, digitalis, griseofulvin, dilantin, …

Acute ulcerative lesions

- **Bacterial**

  Necrotizing ulcerative gingivostomatitis

  Streptococcal gingivostomatitis

  Oral tuberculosis

  Primary syphilis

  Gonococcal stomatitis

Infective gangrene

Necrotizing soft tissue infection.

- acute onset
- rapidly progressive
- deep tissue affected

1) **infective conditions leading to tissue destruction:**

Bacterial infect**:** localized (carbuncle),
extensive (necrotizing fasciitis, etc.)
Fungal (Zygomycosis etc.)
Mixed: Fusospirochetal - Cancrum oris (noma)

2) **preexisting tissue destruction complicated by infection**

Necrotizing ulcerative gingivostomatitis

- term „acute“ not necessary – no chronic form
- psychologic stress (↑ adrenal hormones →↓ immune response + local ischemia)
- important factors: immunosuppression (incl. HIV), smoking, local trauma, poor nutrition, poor oral hygiene, inadequate sleep, recent illness (EBV)
- young – middle-aged adults

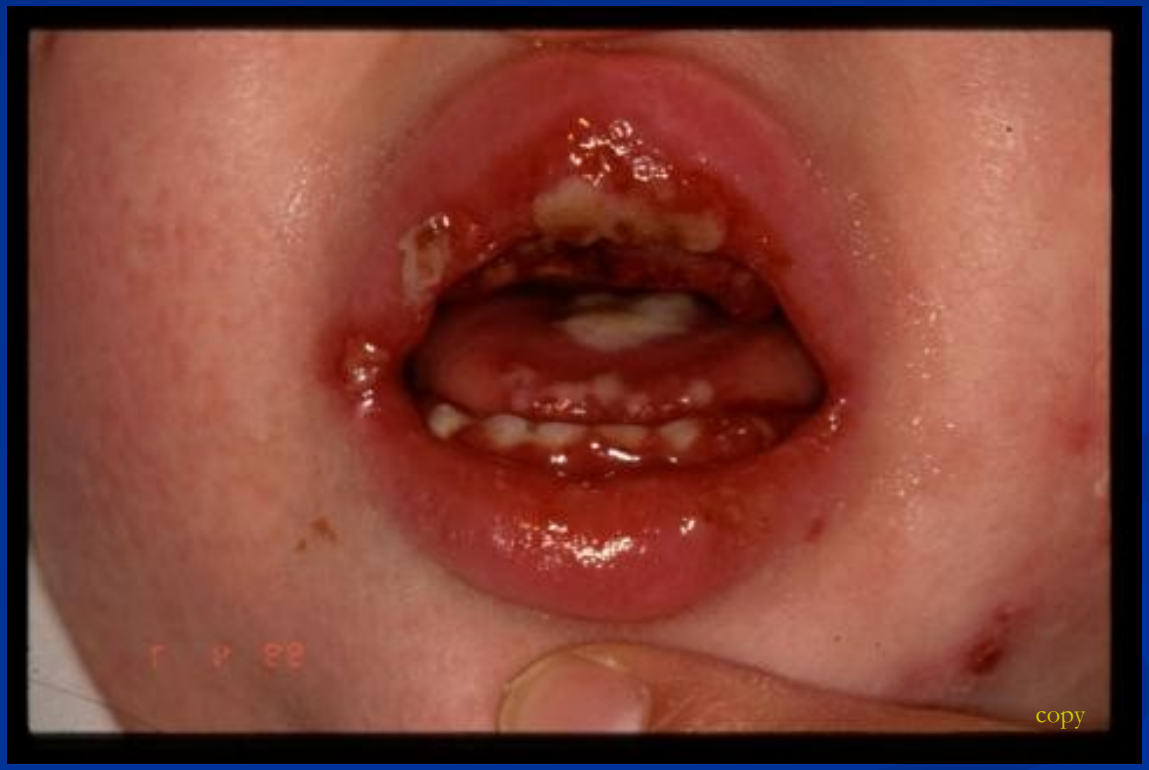
Necrotizing ulcerative gingivostomatitis (Vincent's disease)

- „Punched out" ulcerations + necrosis, rapid onset, painful, foul, fetid odor, + event. fever, lymphadenopathy
- start in interdental papillae, → stomatitis, mucositis,
- progression to the facial skin + bone – noma (children with poor nutrition, often fatal)
- *Fusobacterium* + *Borrelia vincentii* (fusospirochetal complex), polymicrobial, endogenous

Necrotizing ulcerative gingivostomatitis

- nonspecific histopathology: fibrinopurulent pseudomembrane + cellular debris + bacteria, mixed inflammatory infiltrate.
- usually quick resolution with therapy
- in HIV+ persistent

Necrotizing ulcerative gingivostomatitis



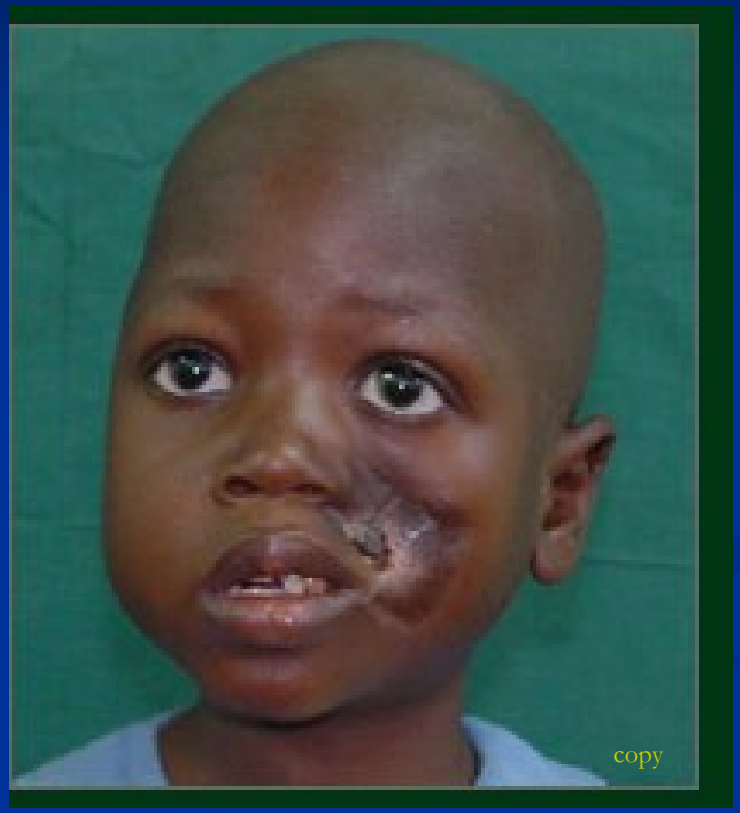
Noma (cancrum oris)

- rapidly progressive orofacial gangrene
- in predisposed patients (immunodeficiency – HIV; malignancy - leukemia; recent illness – measles, herpes simplex, scarlet fever)
- in risk populations (poverty; malnutrition + dehydratation; poor oral hygiene, poor sanitation, proximity to livestock) „Face of poverty”
- commonly starts as NUG

Noma (cancrum oris)

- Children 1-10 yrs
- Noma neonatorum – low-weight infants, *Pseudomonas*
- Fatal in 70% - 90% of cases, with aggressive therapy 10%, survivors disfigured for life (healing → scar → bony fusion and tight mouth closure → microstomia)
- Fusobacterium necrophorum or Prevotella intermedia + Borrelia vincenti (or other bacteria – Staph., Str.)
- synergistic infection → endotoxin → gangrenous necrosis of the gingiva → extending to oral mucosa, perioral tissue and face

Noma



Noma

Granulomatous inflammation

- Bacteria
  - **TBC**
  - leprosy
  - syphilis (3rd stage)
  - anthropozoonoses - cat-scratch disease,
- Parasites or fungi (i.e. toxoplasmosis)
- Inorganic metals or dust
  - silicosis, berylliosis
- Foreign body
  - suture (Schloffer „tumor“), prosthesis
- Unknown – **sarcoidosis**
  - **vasculitis (Wegener)**

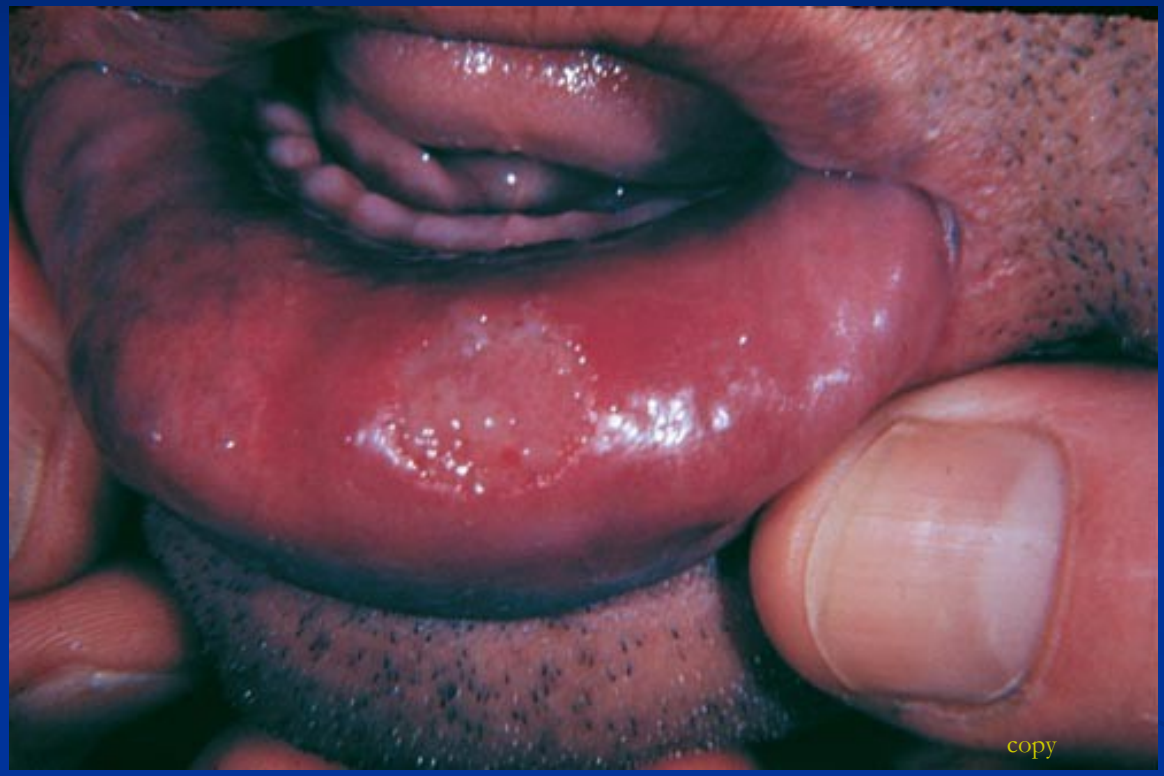
Granulomatous inflammation

- distinctive chronic inflammation type
- cell mediated immune reaction (delayed)
- aggregates of activated macrophages → epithelioid cell → multinucleated giant cells (of Langhans type x of foreign body type)
- NO agent elimination but walling off
- intracellulary agents (TBC)

Syphilis - primary

- **Chancre**: primary lesion, hard + raised ulceration, painless. Primary complex: chancre + regional lymphadenopathy
- granulation tissue + mononuclear, predominantly plasma cell infiltrate
- Lips, tongue, palate, … ! highly infectious!
- Average incubation 20-30 days
- Spontaneous healing in 3-6 wks

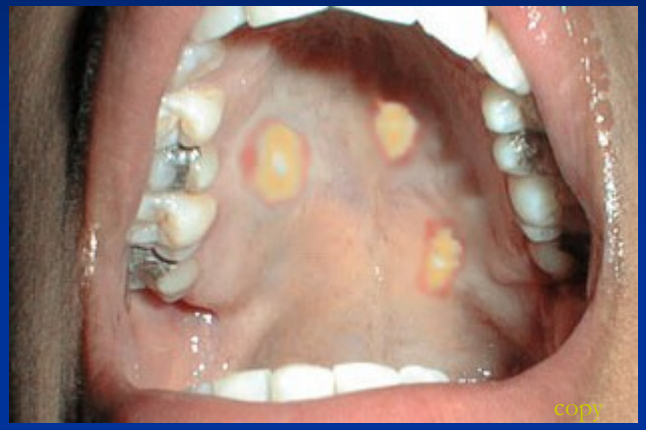
Syphilis – primary



Syphilis - secondary

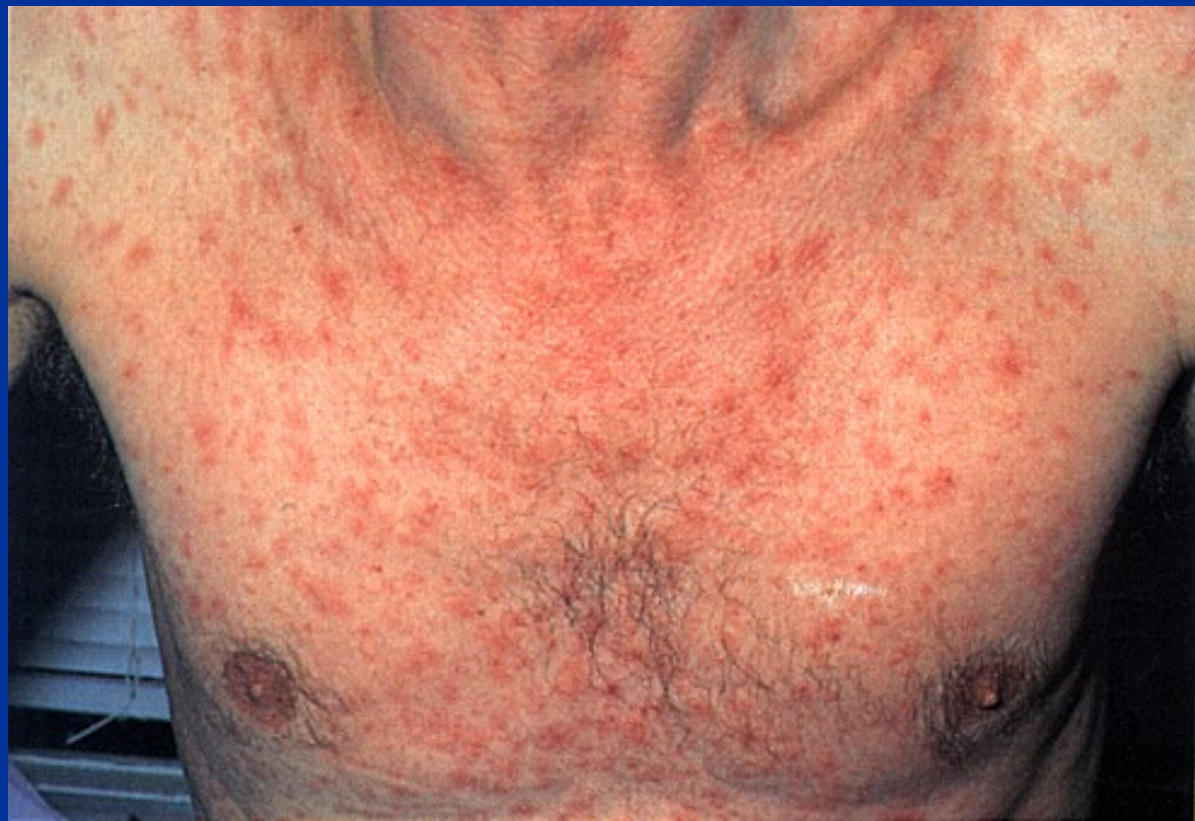
- early generalisation (disseminated s.)
- flu-like symptoms, sore throat, generalized lymphadenopathy
- any time from 2 weeks to 6 months after initial chancre disappears, in 75% of untreated people
- various cutaneous lesions – rash, typ. palms, soles; maculopapular, pustular;
- mucous patches+ erosions in oral cavity; flat, broad-based wart-like papules in mouth corner - condylomata lata; multiple, infectious
- nonspecific histopathology, similar to I. st., ↑ plasma cells
- disappears within 2-6 weeks

Syphilis - secondary

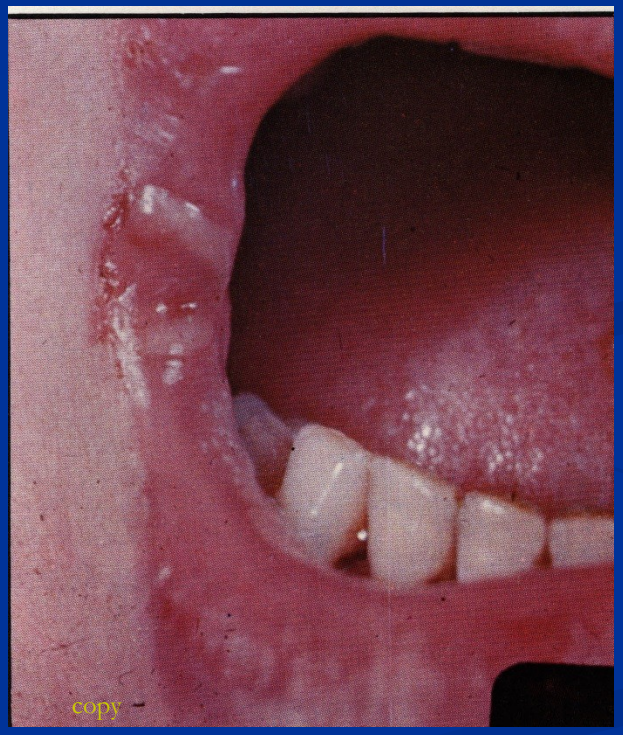


Condylomata lata

Syphilitic rash



Syphilis - secondary



Syphilis: latency period

- Usually not counted as a „stage“
- During this period no symptoms; 5-50 years
- Not transmissible by sexual contact; it can be spread by blood during this time
- Much shorter in HIV infected

Syphilis - tertiary

- in 1/3 of untreated patients
- cardiovascular syphilis (mesaortitis), neurosyphilis (progressive paresis, dementia),
- more benign tertiary syphilis: gummas in skin, mucous membranes, bones, liver; specific granulomas – delayed hypersensitivity reaction;
- histopathology: proliferative endarteritis (endothelial hypertrophy → intimal fibrosis → local ischemia) + inflammation (plasma cells)

  gumma – central coagulative necrosis + specific granulation tissue + fibrous tissue

Syphilis - tertiary

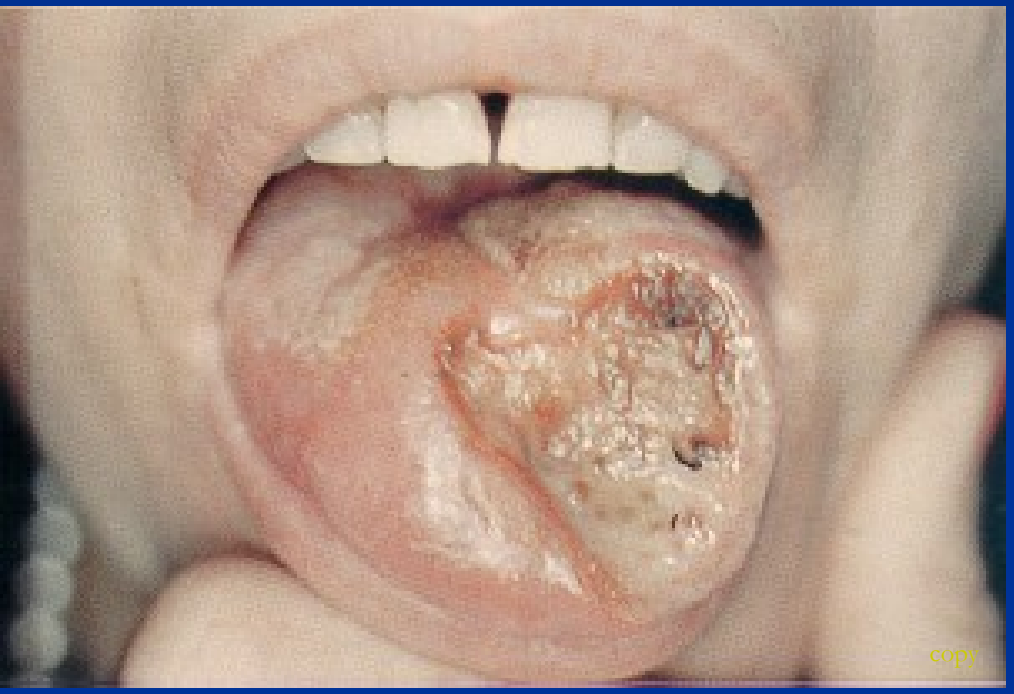
Oral cavity:

- palatal ulcerations – may perforate to the nasal cavity
- tongue - *atrophic luetic glossitis* – diffuse atrophy, loss of papils

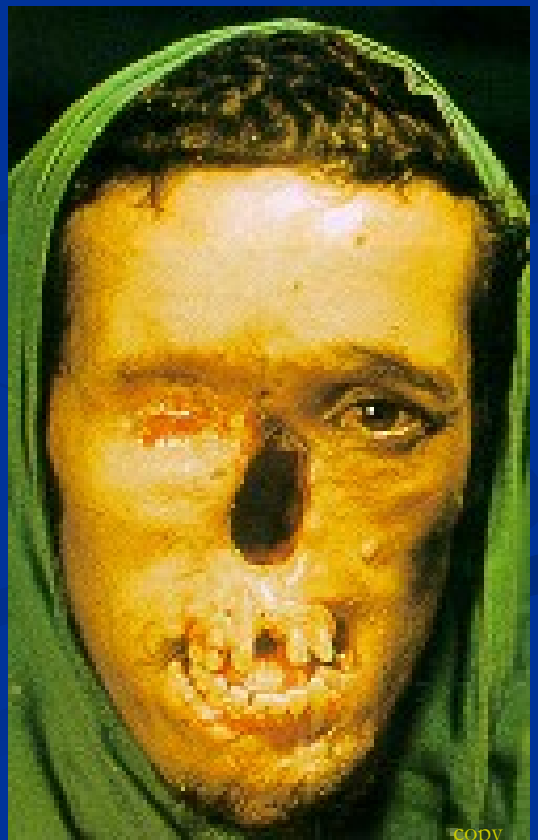
  interstitial glossitis – enlarged, irregular shape (gummata)

Syphilis - tertiary

Gumma + ulceration



Bone destruction

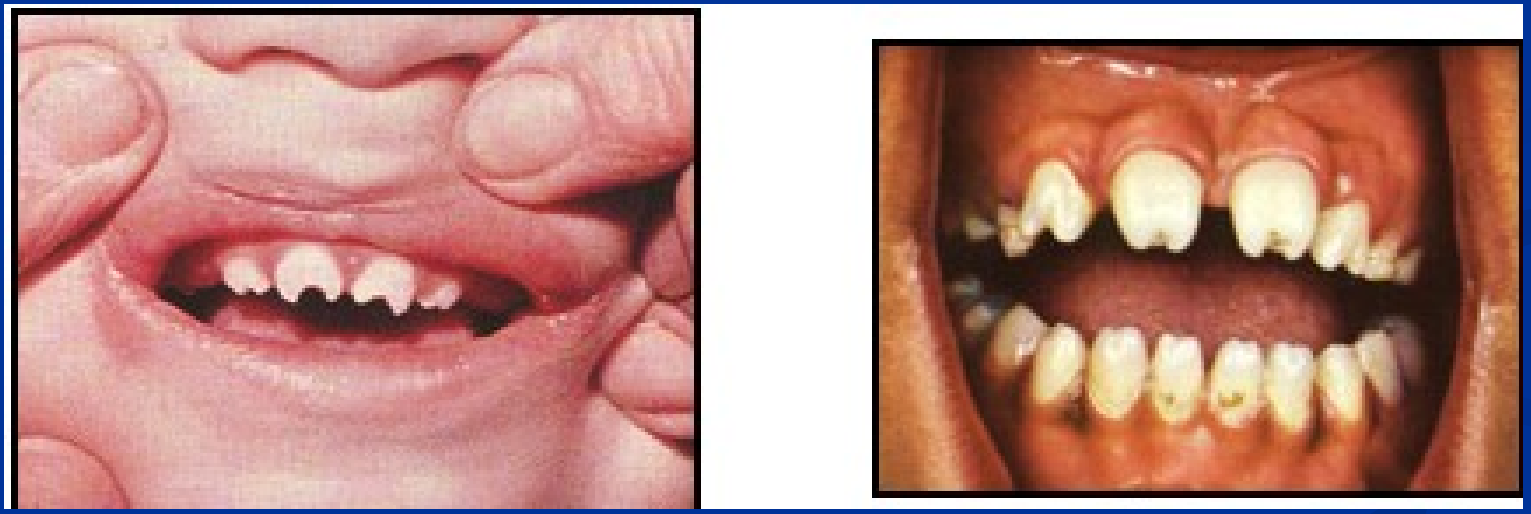


Congenital syphilis

- 1) abortus
  - hepatomegaly + pancreatitis + pneumonia alba
- 2) infantile syphilis
  - chronic rhinitis (snuffles) + mucocutaneous lesions
- 3) late (tardive, congenital) syphilis
  - > 2 years duration
  - Hutchinson triad – notched central incisors + keratitis (blindness) + deafness (injury of n. VIII)
  - mulberry molars + saddle nose

Congenital syphilis

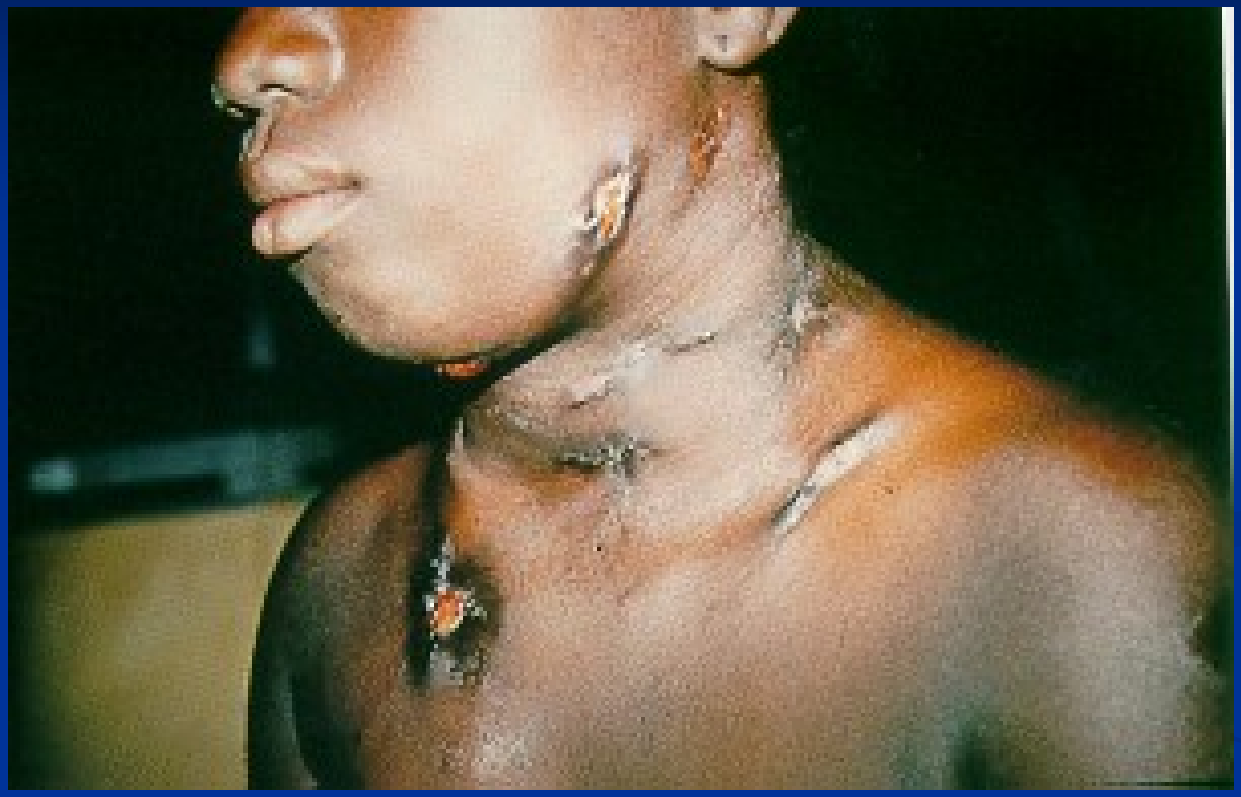
- Hutchinson's incisors and mulberry molars



Tuberculosis

- Mycobacterium tuberculosis, M. bovis
- Primary – usually in lungs; possible gingiva+ cervical lymph nodes
- M. bovis: contaminated milk→ scrofula (↑ oropharyngeal lymphatic tissue + cervical lymph nodes → caseous necrosis → skin fistulae
- Secondary – tongue, palate, lip – painless ulcer; skin – lupus vulgaris
- typical granulomas

Tuberculosis



Tuberculosis with multiple fistulous tracts secondary to lymph node necrosis in patient with scrofula.
Photo by Dr. I. Small

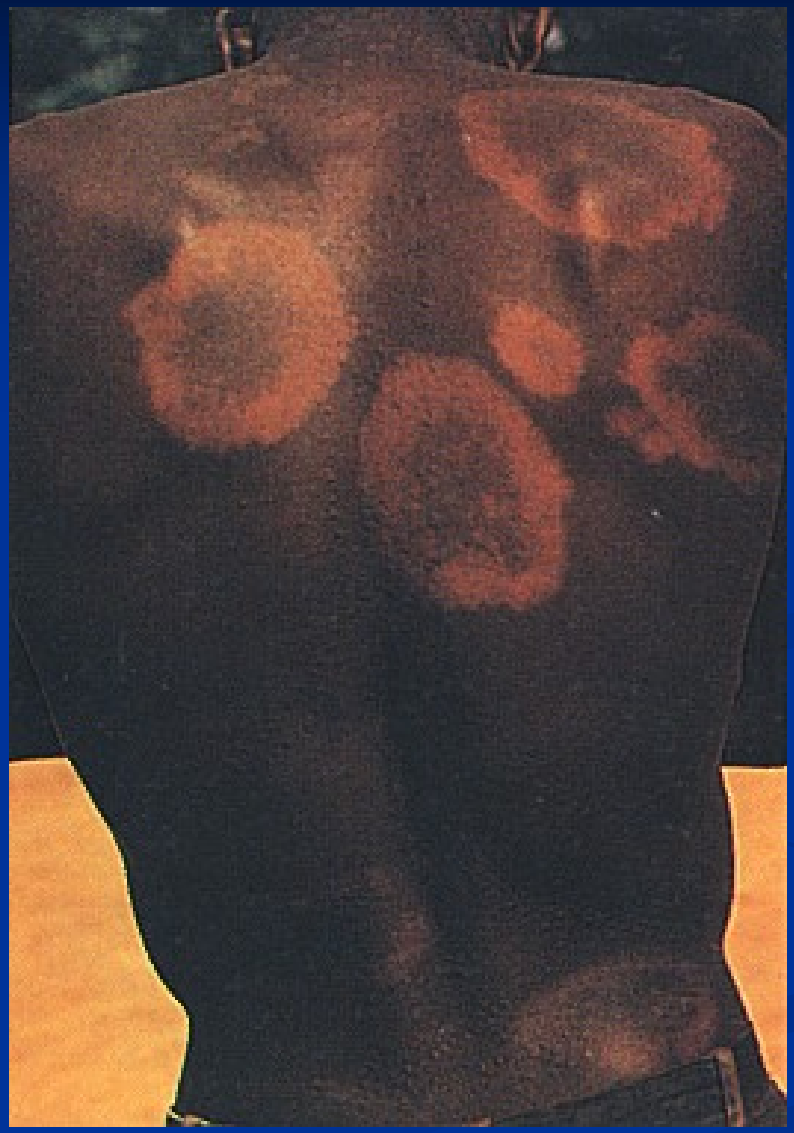
Leprosy (Hansen's disease)

- *M. leprae,* Asia, Africa
- in dermal macrophages and Schwann cells
- air droplets + long contact
- Incubation period: 2 to 12 years or longer
- Neural, tuberculoid (anesthetic) form: Lesions on skin and peripheral nerves. Loss of pigment and sensation. High immunity → sterile lesion.
- Cutaneous, lepromatous form: Progressive disfiguring nodules (*lepromas*) in skin, invades body. Destroys skin, mucous membranes, and bone. Infectious, in ↓ cellular immunity.

Leprosy

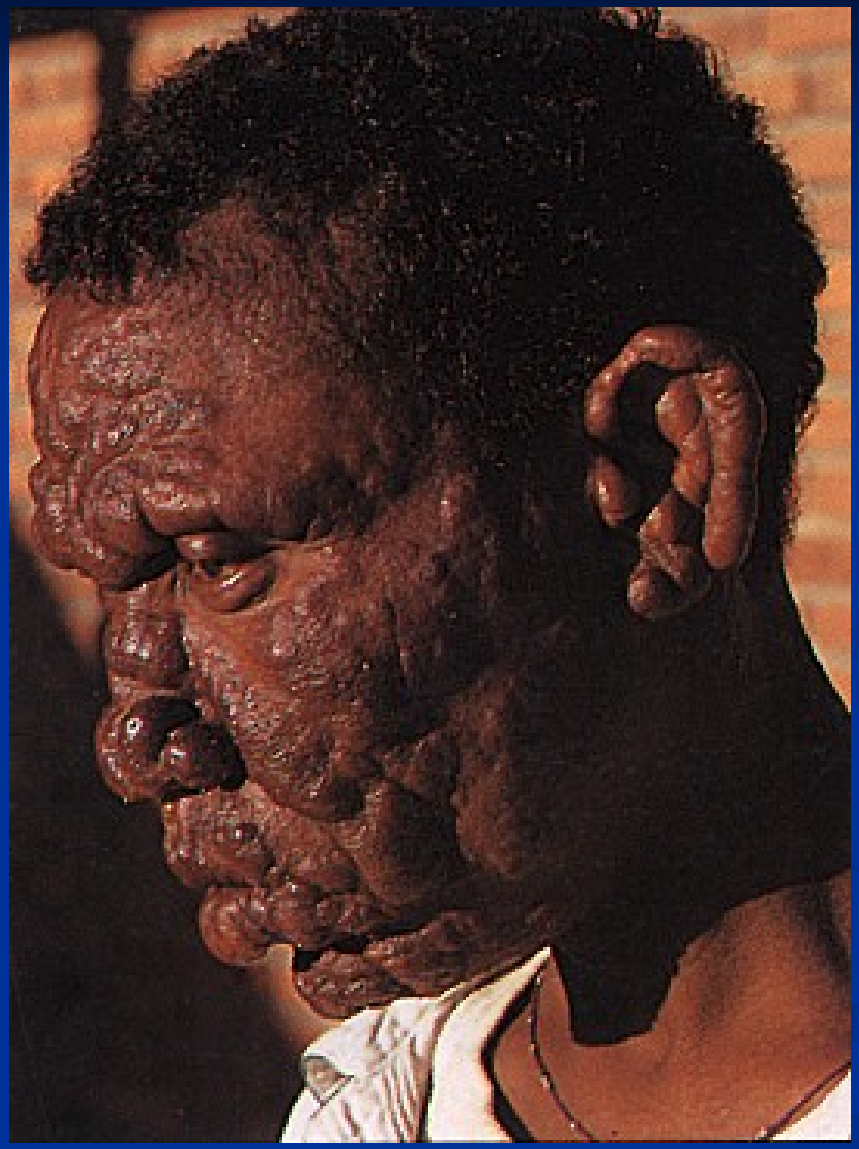
- Paucibacillary ~ tuberculoid, low number of circumscribed hypopigmented lesions. Oral rare.
- Multibacillary ~ lepromatous, start as numerous papules. Invasion → proliferation → ulceration → fibrosis. Facial skin involvment in 1/3, oral lesions in 10%. Sessile papules → necrosis → scarring

Tuberculoid leprosy lesions with depigmentation



Source: Tropical Medicine and Parasitology, 1995

Lepromatous leprosy lesions



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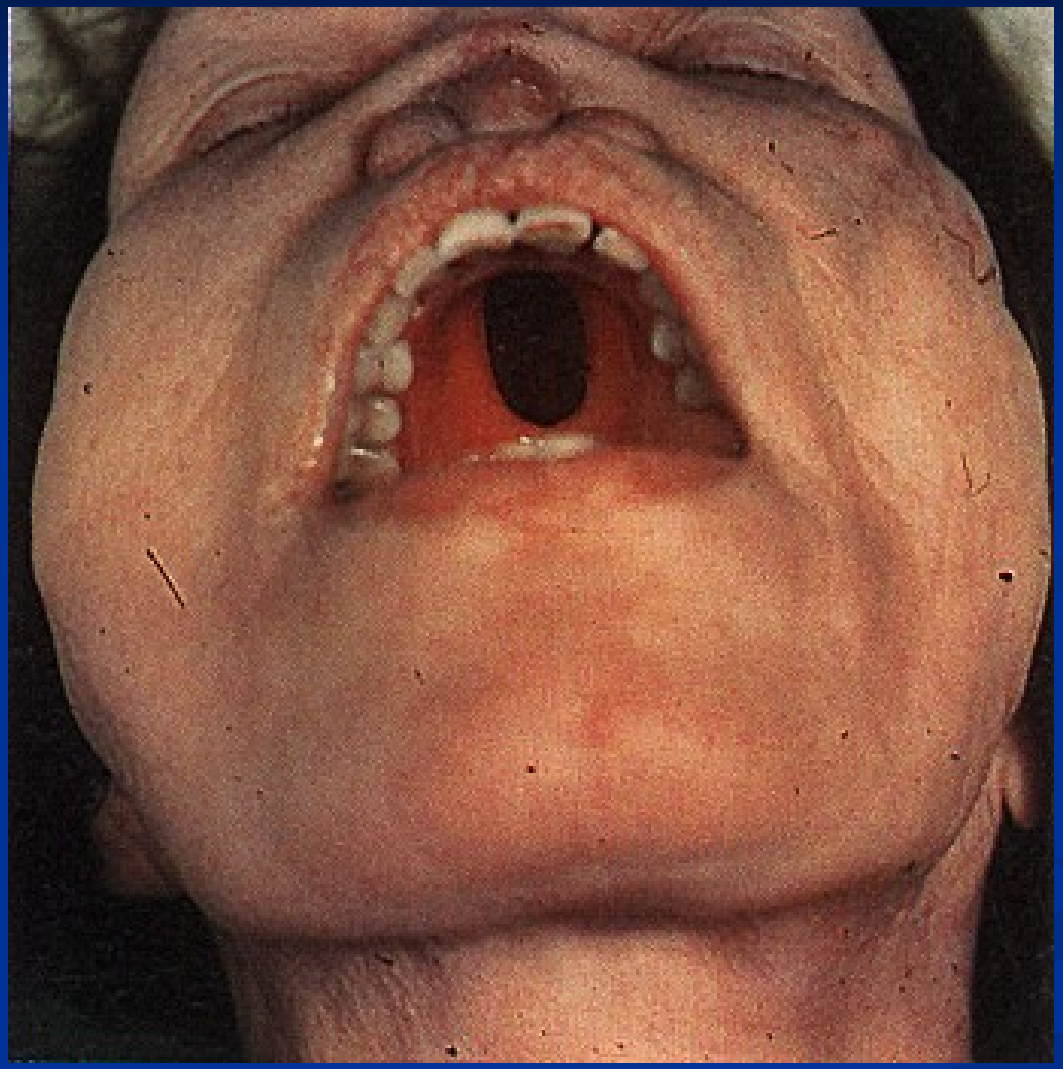
Severe bone destruction in advanced leprosy

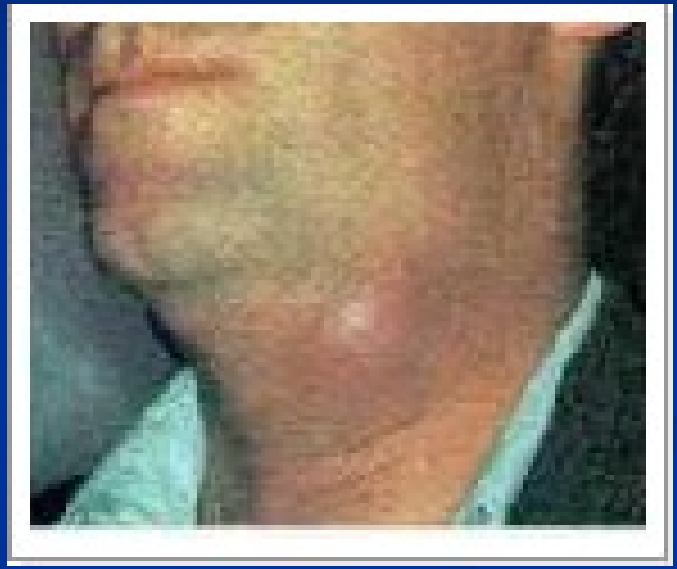


Source: Diagnostic Picture Tests in Infectious Diseases, 1994

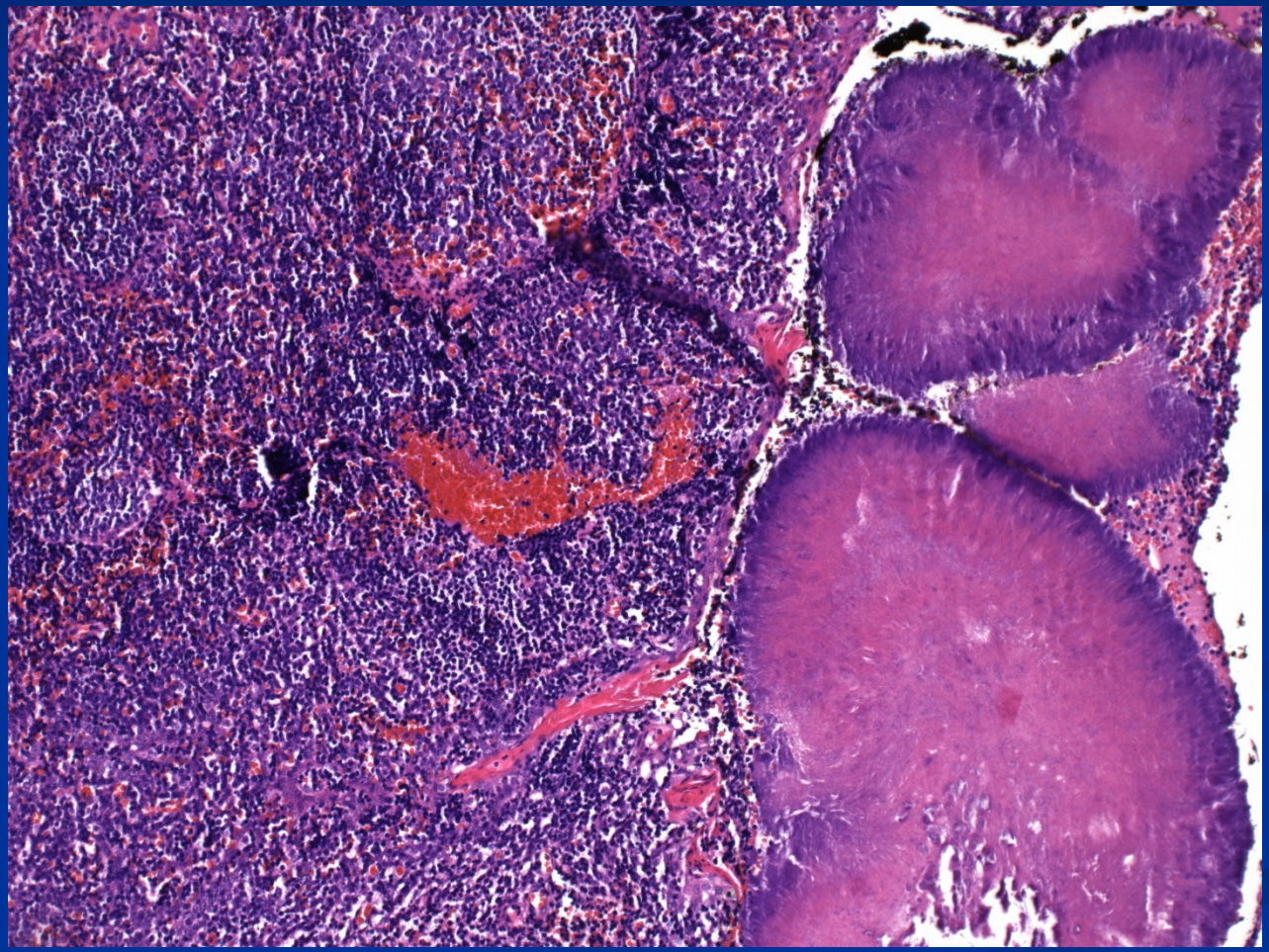
Actinomycosis

- ***A. izraelii* – actinomycosis**
- normal in oral cavity, access due to local lesion (extraction, root infection, …), direct extension
- firm edematous infl. infiltrate → fistulas, yellow „sulphur granules“, fibrosis (scar)
- cervicofacial – most common (submandibular, neck)
- thoracic – lung abscesses
- abdominal – IUD → salphingo-oophoritis
- Micro: G+ PAS+ filamentous colonies surrounded by neutrophilic, granulomatous reaction

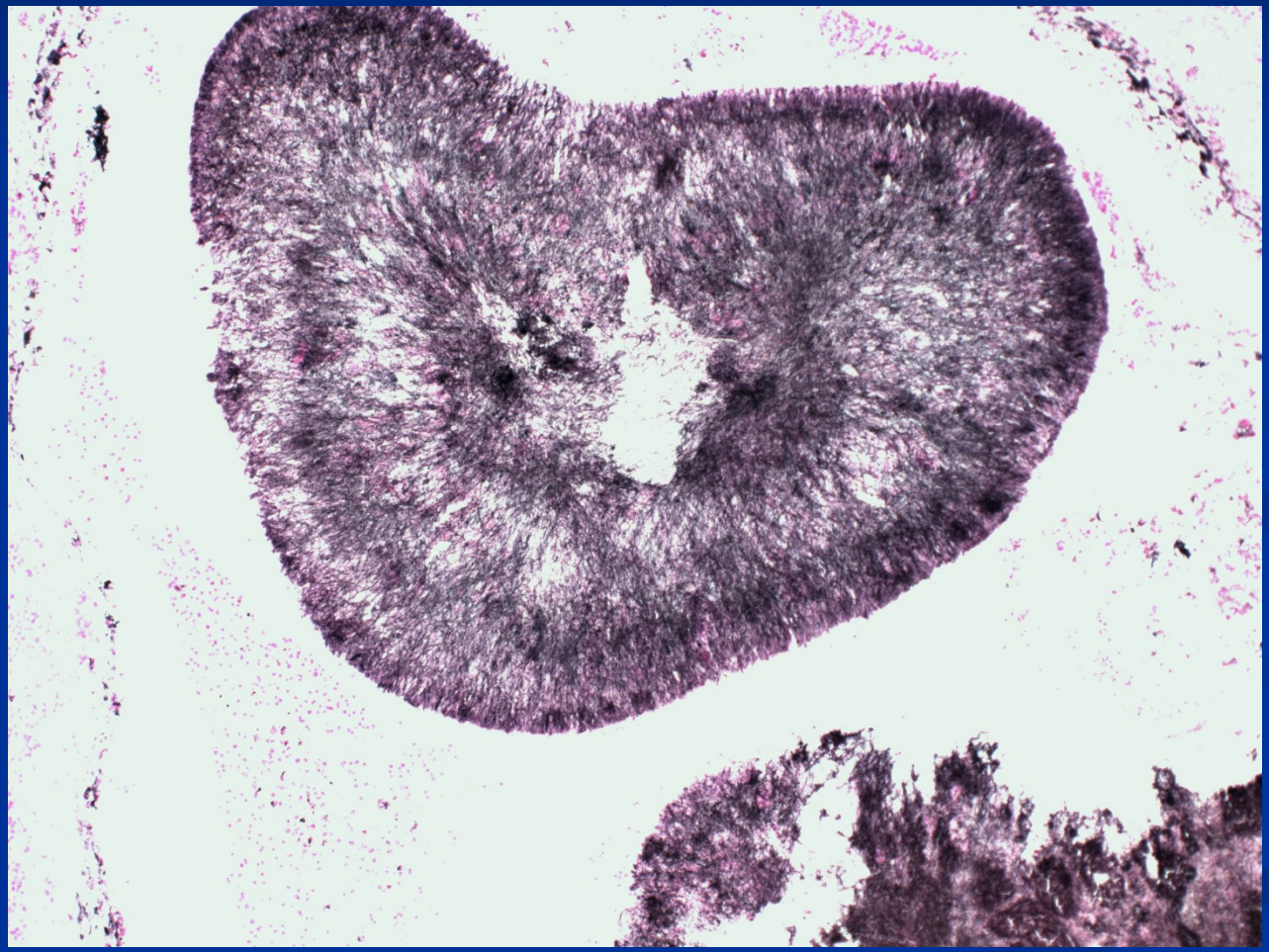
Actinomycosis



Actinomycosis



Actinomycosis - impregnation



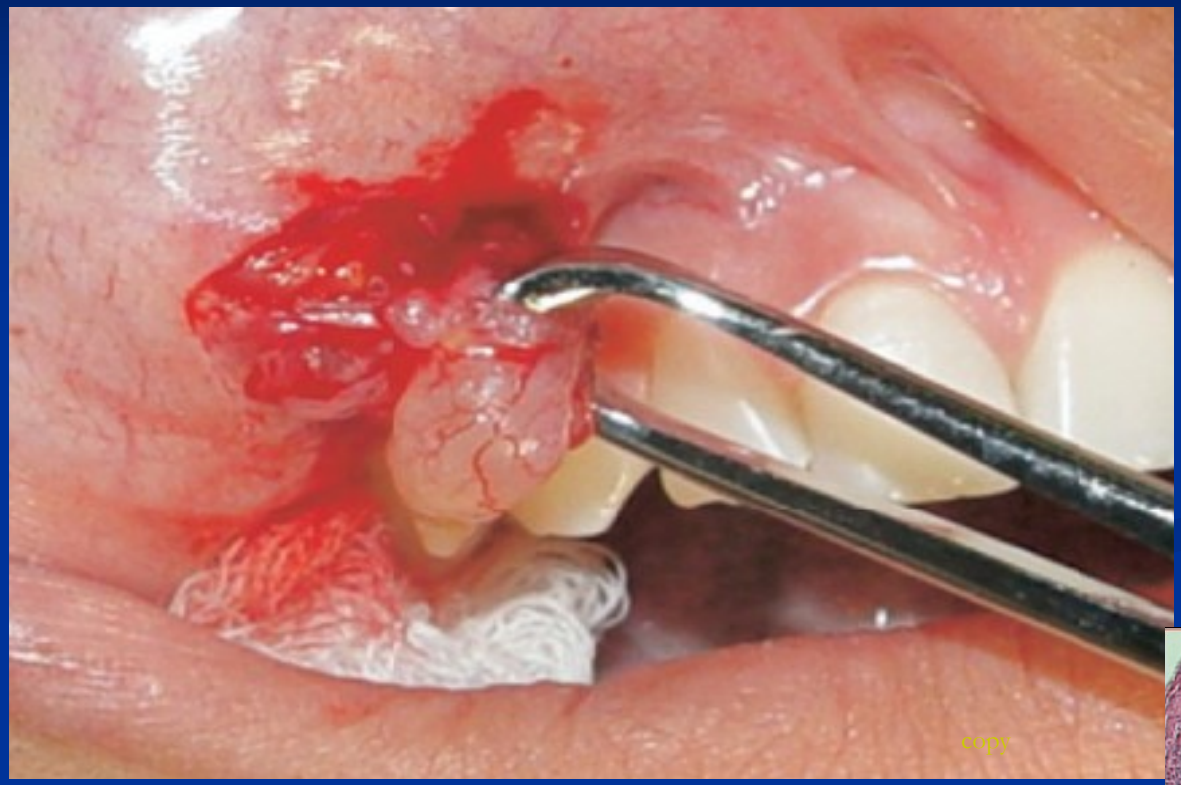
Cat-scratch disease

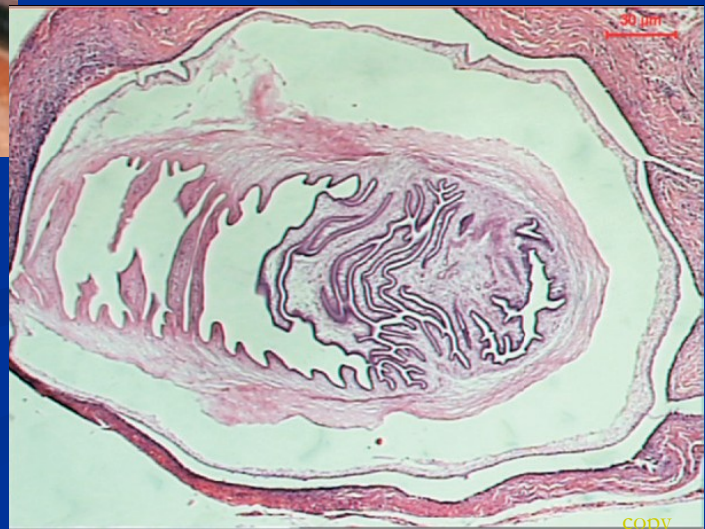
- Bartonella henselae
- skin inflammation - nodule, ulcer
- regional lymphadenopathy in 1-3 weeks
- suppurative necrosis + histiocytic rim
- self-limited

Parasitic infections

- **Toxoplasmosis** – intracellular protozoan dangerous in immunocompromised patients (lymphadenopathy incl. paraoral, encephalitis, pneumonia, myositis); congenital t.
- **Cysticercosis** – frequent in developing countries, hematogenous dissemination, possible encysted taenia larvae in the mouth

Cysticercosis





- **Debridement** is an essential component of wound care as the presence of devitalised tissue can impede the healing process. Larval therapy has been used for the debridement of wounds for several hundred years.