Bacterial, protozoal infections

V. Žampachová I. ÚP

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

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

- 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

- 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

- 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

- 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)

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

Bacterial - nonspecific

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

Bacterial - specific patterns

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

- 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)

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



- 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

- 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

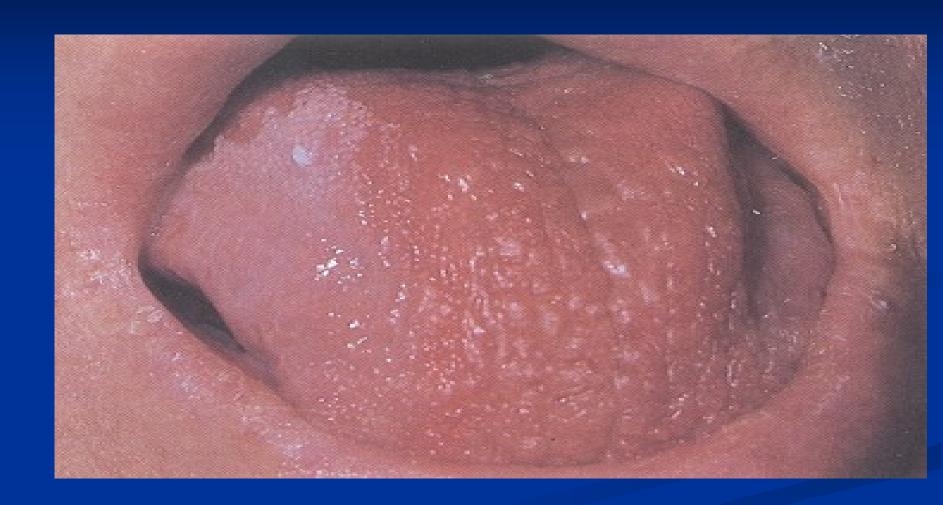
Cervical lymphadenitis

Tongue: start with white coating + visible fungiform papillae

white strawberry tongue

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

Soft palate: possible petechiae



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

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.

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.

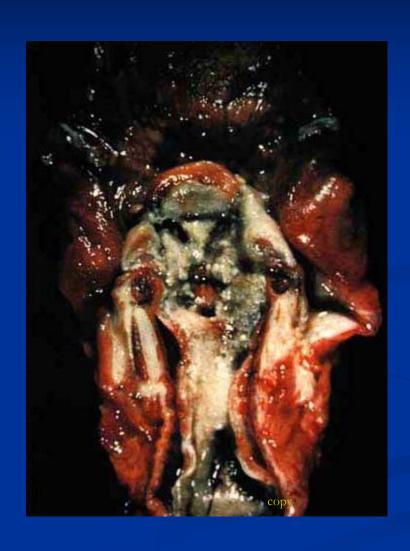
Pharyngeal diphtheria

- 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.

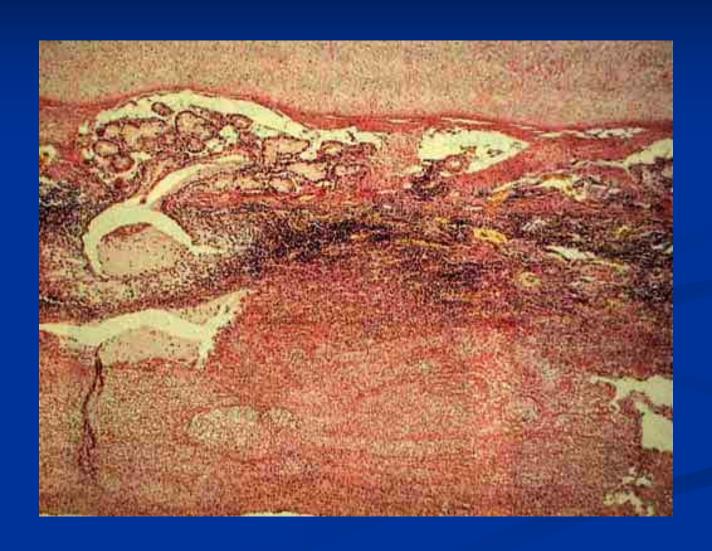
Pharyngeal diphtheria

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.



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/fascial 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 (dentoalveolar abscess, ↑ proportion of anaerobes), esp. in immunodeficiency
- diffuse edema (hard consistency), taut shiny skin
- limited mouth opening, dysphagia
- pain
- severe systemic signs (fever, leucocytosis, toxemia)
- tender enlargement of cervical LN
- possible fatal complications
 - laryngeal edema glottis
 - mediastinitis
 - extension to carotid artery

Tissue space infections

Ludwig's angina

- severe form of cellulitis
- source from lower 2nd /3rd molars
- bilateral spread → sublingual + submandibular space → parafaryngeal space, neck, mediastinum
- possible oedema of glottis →risk of suffocation
- respiratory distress, headache
- treatment
 - tooth extraction + drainage
 - agressive antibiotic treatment
 - sm. tracheostomy necessary

Tissue space infections

Cavernous sinus thrombosis

- possibly fatal complication
- source upper teeth, sinusitis, skin abscess
- retrograde venous blood flow up into the skull
- cyanosis + edema of the eyelid
- limited eye movements, pain
- headache, vomiting, high fever
- fatal without prolonged antibiotic therapy
- sequels common incl. blindness
- rapid progession (untreated fatal within 24 hrs)

Oral ulcerative lesions

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

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, broadbased 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

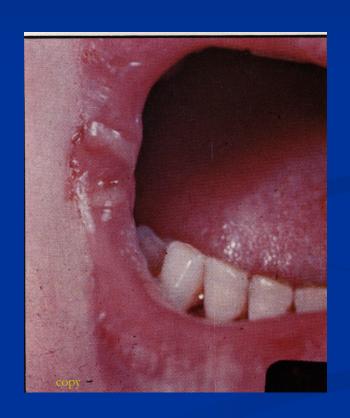


Condylomata lata

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

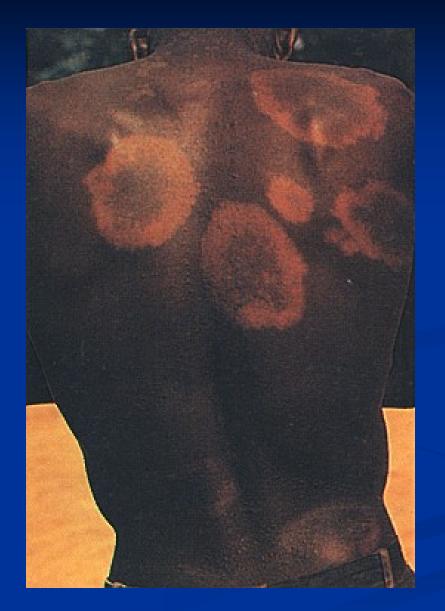
Leprosy (Hansen's disease)

- 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



Source: Tropical Medicine and Parasitology, 1995

Severe bone destruction in advanced leprosy

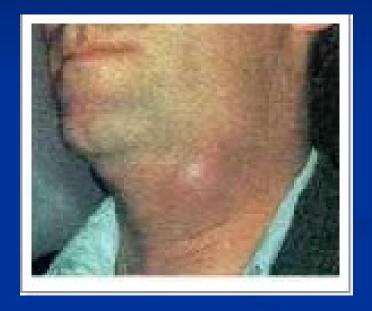


Source: Diagnostic Picture Tests in Infectious Diseases, 1994

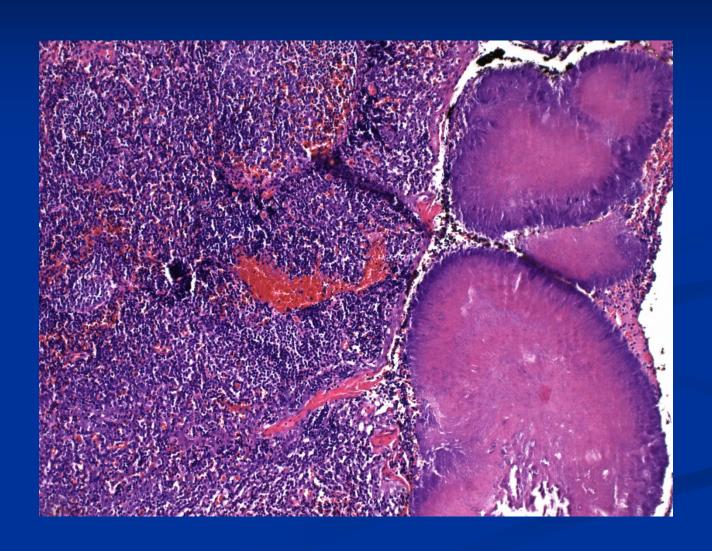
Actinomycosis

- 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

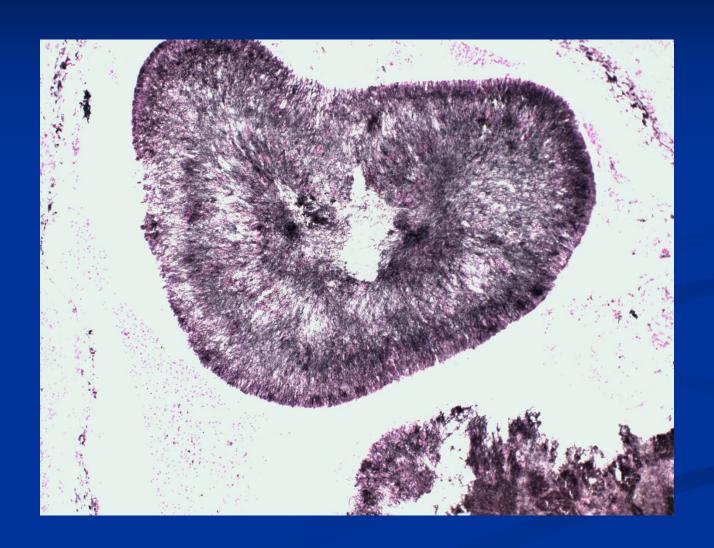
Actinomycosis



Actinomycosis



Actinomycosis - impregnation



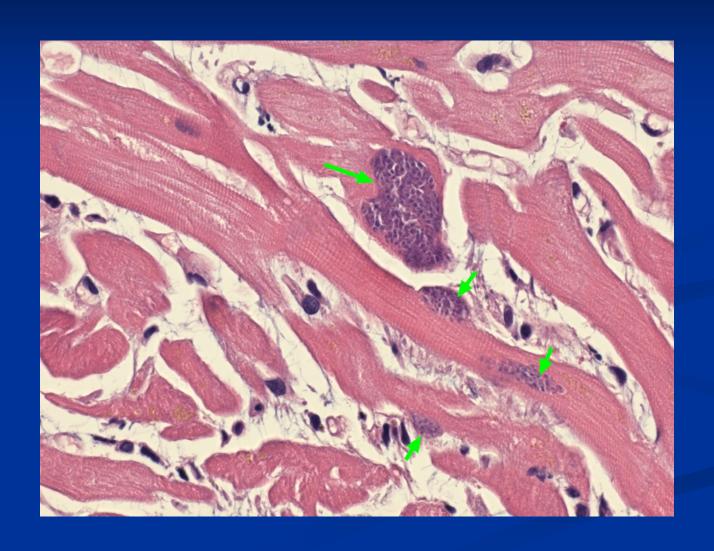
Cat-scratch disease

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

Parasitic infections

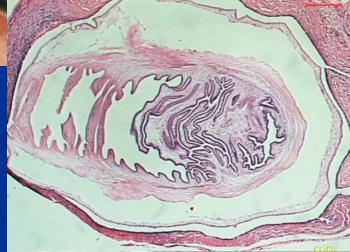
- **Toxoplasmosis** intracellular protozoan dangerous in immunocompromised patients (lymphadenopathy incl. paraoral, encephalitis, pneumonia, myositis); congenital t.
- **Cysticercosis** frequent in developing countries, hematogenous dissemination, possible encysted taenia larvae in the mouth

Toxoplasmosis in muscle



Cysticercosis





■ Debridement - essential component of wound care; the presence of devitalised tissue ↓ the healing process. Larval therapy used for the debridement of wounds during hundreds of years.



