ZOONOSES

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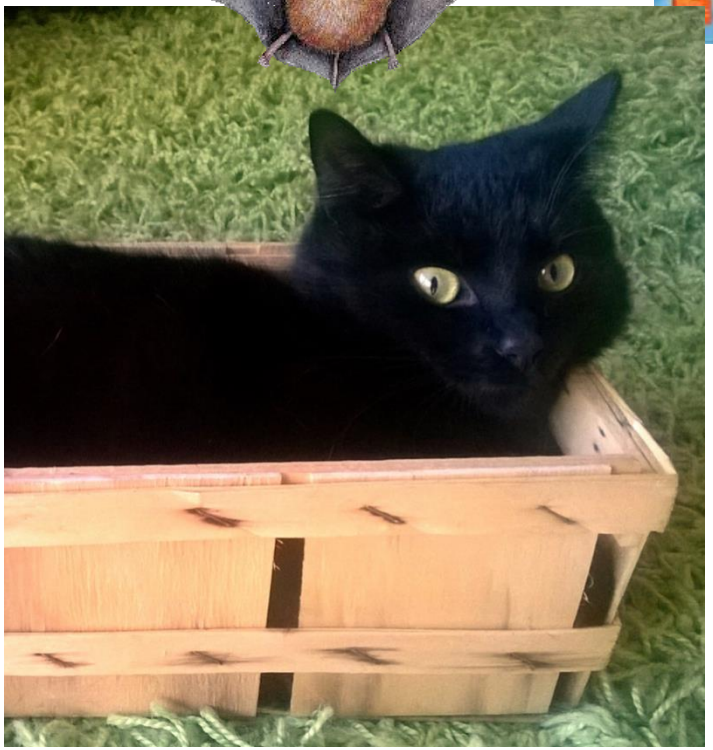
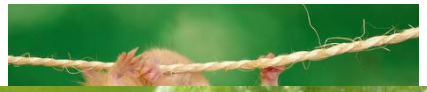
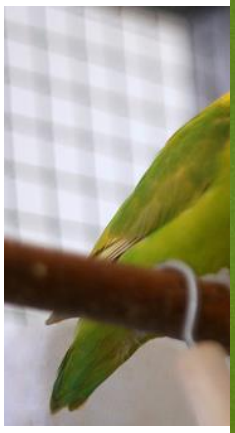
Discovery



Discovery

Zoonoses

- animal diseases, which can be transmited to humans
- source of infection is an animal, ethiological agent is bacterium, virus or parasite

Zoonoses

Transmission is possible

directly – by biting ,with saliva

indirectly

- ingestion- contamination of hands or food
- inhalation of contaminated dust or spray
- inoculation by vector (insect – mosquito, tick, flea)

transplacental- the only possible way of transmission from people to people

At present we know about 250 zoonoses, 50 of them are common

The most important zoonoses

Bacterial ethiology

- **tularemia**
- salmonelosis
- campylobacteriosis
- **listeriosis**
- **leptospirosis**
- **bartonelosis (CSD)**
- borreliosis
- brucellosis
- anthrax
- plague
- yersiniosis
- ehrlichiosis
- Q fever

Parasitic ethiology

- **toxoplasmosis**
- **toxocarosis**
- leishmaniosis
- trichinelosis
- echinococcosis

Viral ethiology

- rabies
- tick-born encephalitis

Prion ethiology

- Creutzfeld- Jakob disease

- toxoplasmosis
- cat scratch disease
- campylobacteriosis
- toxocariasis
- lyssa

- toxocariasis
- yersiniosis
- lyssa
- campylobacteriosis

- leptospirosis
- tularemia
- lymfocytic choriomeningititis
- Hantavirus infection

- ornitosis
- campylobacteriosis
- salmonelosis
- avian flu

Zoonoses – dif.dg

Zoonoses – dif.dg

Fever + lymphadenopathy	
regional	generalized
– tularemia – bartonellosis – yersiniosis	– toxoplasmosis – listeriosis – brucelosis – leishmaniosis

TULAREMIA

Tularemia

- **Etiology agent**: ***Francisella tularensis***- small gram-negative coccobacilli, cultivation on common culture medium is difficult

- **Source of infection**: small rodents, hares, rabits

Tularemia

Epidemiology:

widely distributed , esspecially in Northen Hemisphere

Transmission is possible via :

- cuts on hands during skinning and cutting up the ill animals
- aspiration of contaminated dust or aerosol,
- ingestion of contaminated fruit or vegetable or undercooked meat
- a tick bite

IP averages 5-7 days, ranges from 1 to 21 days.

Small infectious dose, 10-50 organisms

Tularemia

Pathogenesis

- bacteria spread from the site of entry to the regional lymph nodes
- lymph nodes are enlarged and tender, granulomatous inflammation of LN with suppuration, **necrosis** and **colliquation** are typical of tularemia

Clinical manifestations

- ***ulceroglandular*** tularemia- skin lesion + lymphadenopathy
- ***glandular*** tularemia – skin lesion (ulcer) is not present
- ***oculoglandular*** tularemia- the side of entry is conjunctiva, preauricular lymphadenopathy
- ***oralglandular*** tularemia - exudative unilateral pharyngitis or tonsillitis, cervical lymphadenopathy
- ***typhoidal (abdominal)*** tularemia – no prominent lymphadenopathy, nonspecific symptoms - fever with chills, a headache, muscle pain, nausea, vomiting, diarrhea, abdominal pain
- ***pneumonic*** tularemia – fever, cough with minimal sputum production, pleuritic chest pain

Skin rash may be found in up to 35 % of cases.

Tularemia

Diagnosis

- serologic diagnosis- IgM and IgG antibodies are positive from the end of the 2 week.
- direct cultivation is difficult
- ultrasonography of enlarged lymph nodes - typical picture of colliquation

Therapy

-the most widely used **tetracycline + gentamicin**, in children under 8years of age macrolid along with gentamicin, alternative: *fluorochinolons*

-exstirpation of whole suppurative lymph node

Zoonoses

Fever + meningeal symptoms

Zoonoses

Fever + meningeal symptoms

- listeriosis
- TBE
- Lyme disease (2.stage)
- leptospirosis
- ehrlichiosis
- lymfocytic choriomeningitis
- West Nile fever
- *toxoplasmosis*

LISTERIOSIS

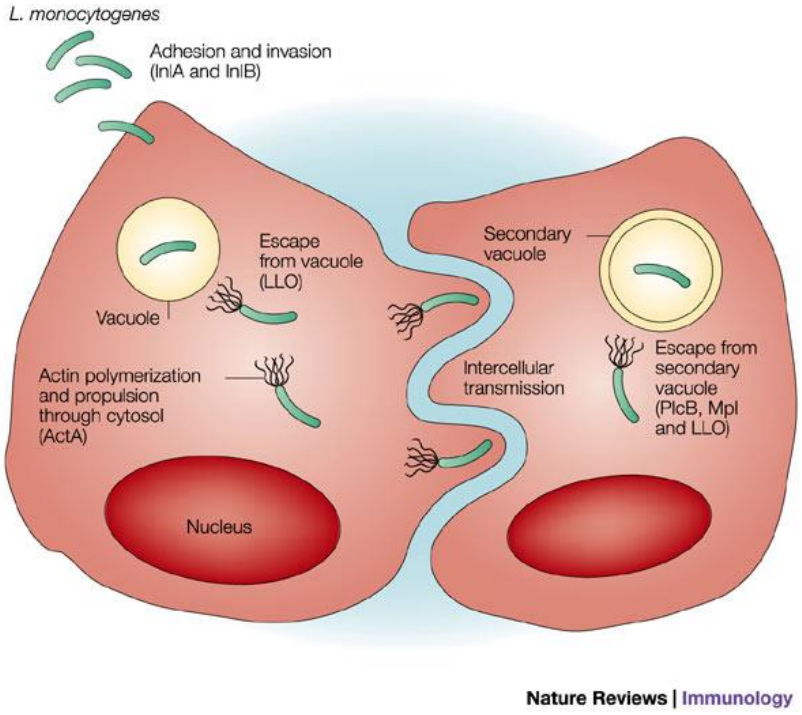
LISTERIOSIS

Ethiologic agent: bacterium ***Listeria monocytogenes***

Source : widespread in animals, soil, water.

Epidemiology: A human may get infected :

- ingestion of contaminated food -dairy products (cheeses) or sausages
- bathing in contaminated water
- contact with ill animal
- transplacental transmission



- Intracelular pathogen

- GIT→ bacteraemia →

CNS, placenta

High risk of infection:

patients with suppression of cell-mediated immunity

CLINICAL FEATURES OF LISTERIOSIS

1. **Acquired listeriosis** :

- inapparent or abortive form
- submandibular lymphadenopathy
- **sepsis**
- **purulent meningitis**

Immunocompromised persons are in a high risk of invasive listeriosis.

- newborn may acquire infection intrapartum- severe meningitis or sepsis

Risk factors of listerial infection

lymfoma
AIDS
pregnancy
biologic treatment
newborns
seniors
chronic metabolic diseases

CLINICAL FEATURES OF LISTERIOSIS

2.Congenital listeriosis :

- result of transplacental infection during maternal bacteriaemia, maternal illness is often mild and unrecognized
- in early pregnancy it mostly results in fetal death
- affected infants may be born prematurely, with **sepsis** or severe disease ***granulomatosis infantiseptica*** **-** granulomatous infiltration of parenchymal organs, papular rash

LISTERIOSIS

Diagnosis

- culture of blood, CSF, sputum, amniotic fluid
- serology

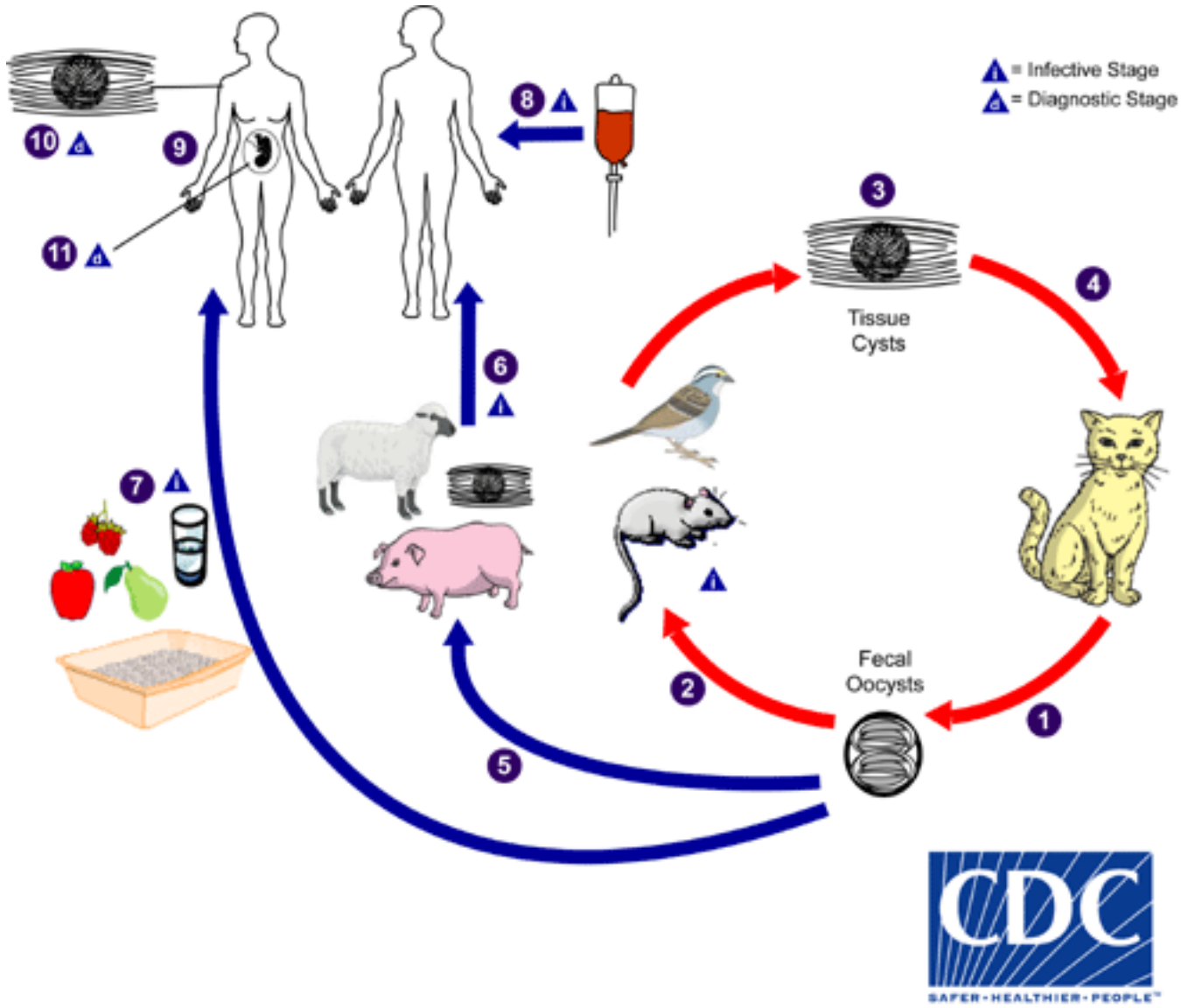
Therapy: ampicilin, in serious cases in combination with gentamicin

Cotrimoxazol

Vancomycin, meropenem

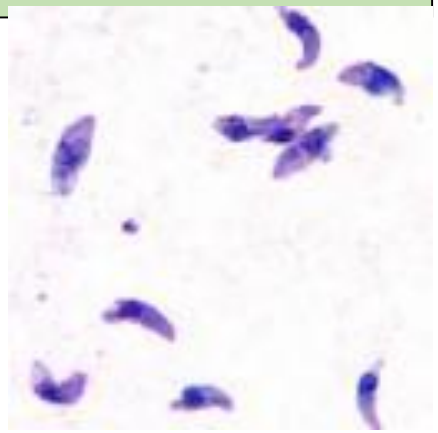
Resistance to cephalosporines!!

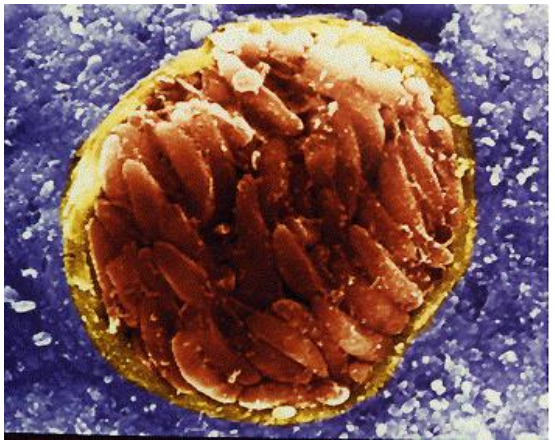
TOXOPLASMOSIS


= Infective Stage
= Diagnostic Stage
Tissue Cysts
Fecal Oocysts
1
2
3
4
5
6
7
8
9
10
11
CDC
SAFER·HEALTHIER·PEOPLE™

Toxoplasmosis

- **Etiologic agent**: protozoan parasite ***Toxoplasma gondii***
  - ***oocyst***
  - ***bradyzoit***
  - ***tachyzoit***





Toxoplasmosis

- **Source of infection**: members of cat family are the main reservoir
- oocyst→ GIT → tachyzoites → blood → neural and muscle tissue → tissue cyst bradyzoites

- If a pregnant woman becomes infected , tachyzoites can infect the fetus via the bloodstream

Toxoplasmosis

Epidemiology

Cats get infected by carnivorism and shed oocyst in their faeces.

Human infection may be acquired :

- ingestion of undercooked infected meat with bradyzoites
- ingestion of the oocyst from fecally contaminated hands or food
- transplacental transmission (tachyzoits)
- organ transplantation or blood transfusion (tachyzoits, bradyzoits)

Toxoplasmosis

Clinical manifestations:

1.Congenital toxoplasmosis

2. Acquired postnatal toxoplasmosis

Congenital toxoplasmosis

- only **primary infection** in a pregnant woman may result in transplacental transmission

The clinical signs of congenital toxoplasmosis :

- intrauterine death or abortion
- cerebral calcification , hydrocephalus, chorioretinitis (Sabin trias)
- myocarditis, microphtalmus, hepatosplenomegaly, strabism, deafness, blindness
- psychomotoric retardation

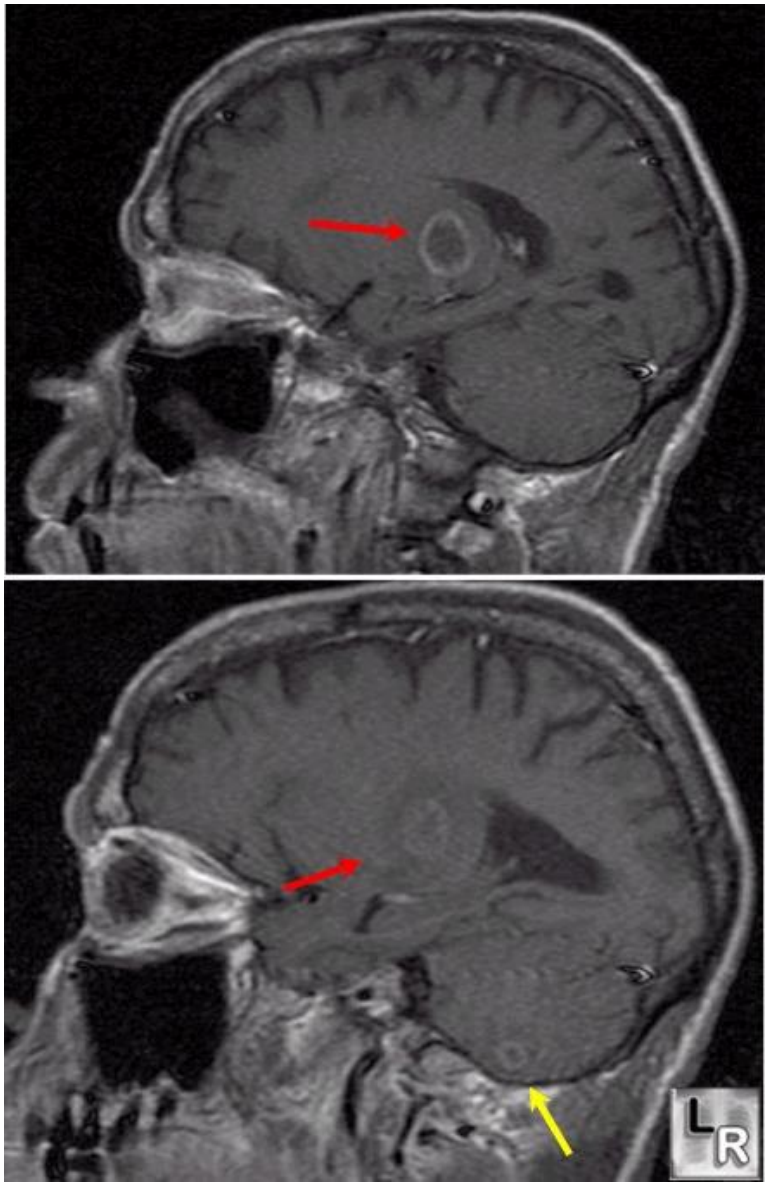
Acquired postnatal toxoplasmosis

- ***inapparent or abortive*** – only nonspecific mild symptoms: fever, tiredness, musclepain
- ***lymphadenopathy***

- ***chorioretinitis***

- ***brain toxoplasmosis*** – in immunocompromised hosts , mostly AIDS patients , result from reactivation of latent infection

(bradyzoite cyst). Multiple cerebral lesions on CT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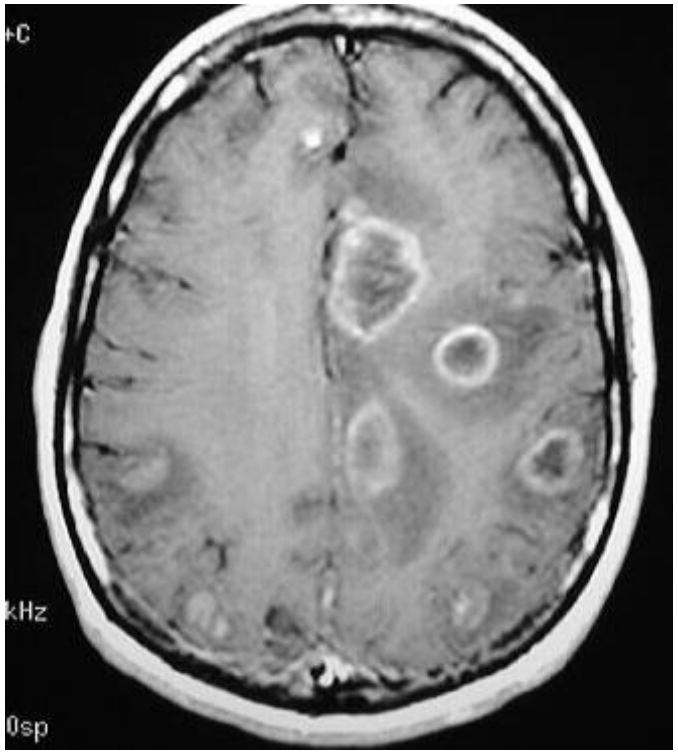
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Toxoplasmosis

Diagnosis

- serologic diagnosis

- IgG - culminate 6 months after infection, high titers persist months , low titers life- long
- IgM – disappear within 9 months
- IgA - dissapear within 6 months

- PCR diagnosis is possible

TOXOPLASMOSIS

Therapy

Toxoplasmosis is a self limiting disease in most cases

Treatment is necessary only in :

- pregnant women
- infected neonates
- immunosupressed persons

Drugs: **sulphonamide + pyrimetamine** (folate inhibitor, its side effect is decreasing of leucocytes in blood- supplementation of folinic acid and monitoring of blood count is necessary)

- **Spiramycin** in pregnancy.

LEPTOSPIROSIS

LEPTOSPIROSIS

Ethiologic agent:

Leptospira grippotyphosa, Leptospira icterohaemorrhagiae, Leptospira sejroe

Source:

rats and cattle

.

LEPTOSPIROSIS

Epidemiology:

- trasmission via animal urine or contaminated water and food
- the site of entrance is damaged skin, conjunctiva or mucous
- risk- group persons are farmers, vets, sewer workers, or people bathing in contaminated water or fishing
- incidence is increasing after floods.

IP 1-3 weeks

Clinical manifestations of leptospirosis

asymptomatic x mild course x severe complications

Mostly 2 periods:

1.leptospiremia - flu like syndrome (fever, musclepain, a headache)

LEPTOSPIROSIS

2. damage to the organs – liver, kidneys, CNS

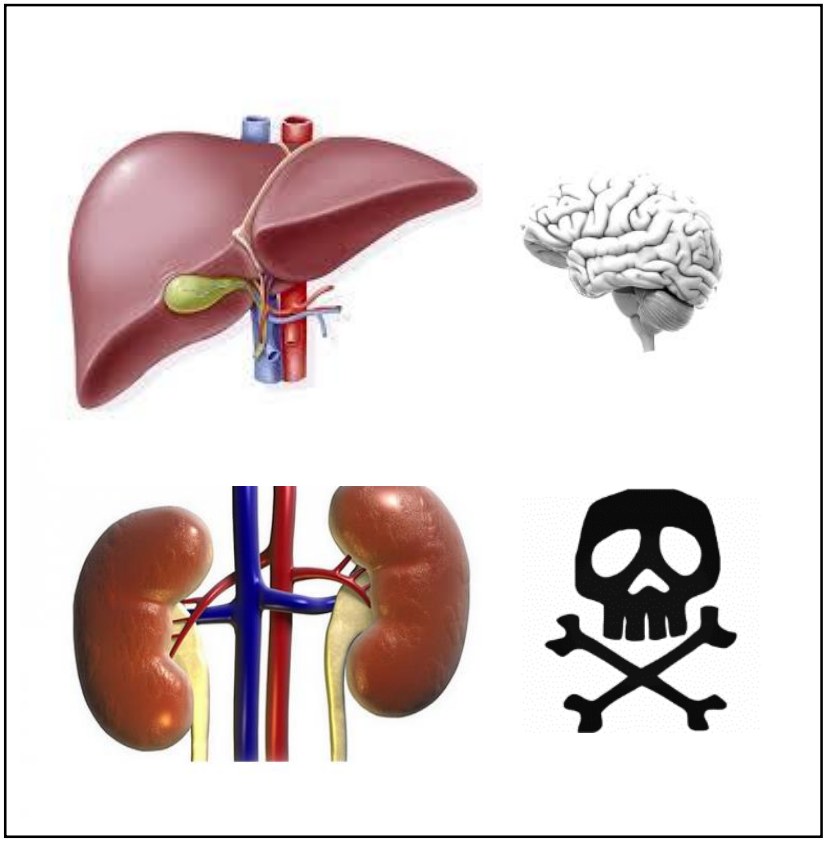
immunopathologic reaction

- **icteric form** – the most serious, Weil´s disease. Often complicated by renal failure, hepatitis, aseptic meningitis, DIC, severe trombocytopenia , SPHS (Severe Pulmonary Haemorrhagic Syndrom)
- **anicteric form** – aseptic meningitis, damage to the liver and kidneys is mild

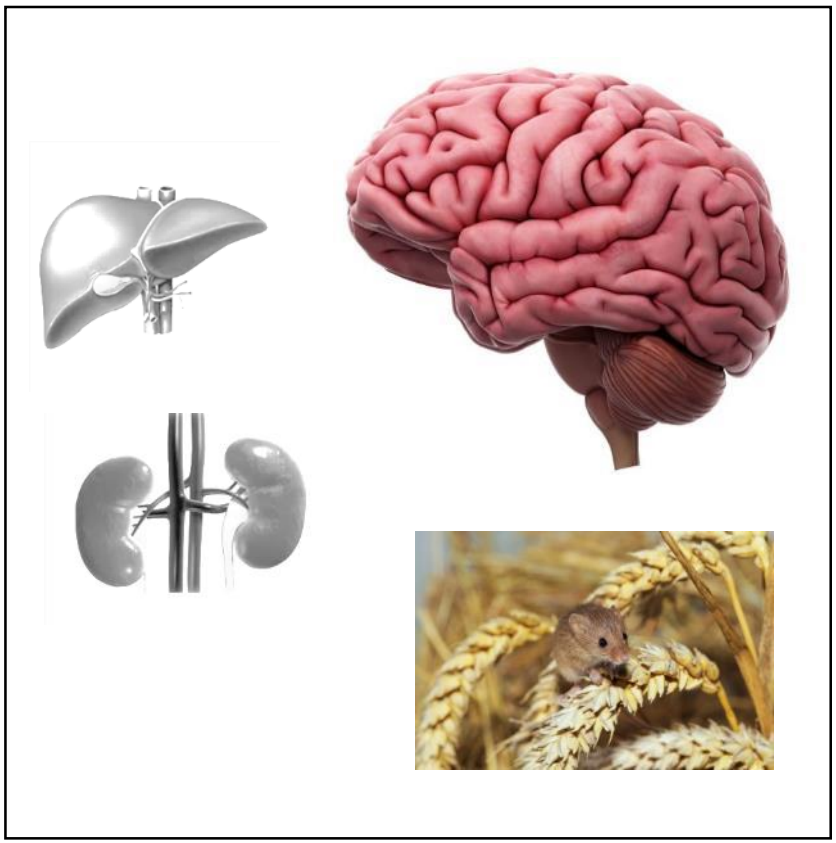
- Complications: myocarditis, iridocyclitis

Leptospirosis

Icteric form (Weil disease)



Anicteric form



Lyme Borreliosis
(Lyme disease LD)

Early LD	**Early localised stage** (days to weeks after infection)	**Erythema migrans** -sometimes with general flu symptoms
	Early disseminated stage (weeks to months after infection)	**Multiple erythema migrans** lymphocytoma **Neuroborreliosis** -cranial neuritis: paresis of n. VII and other cranial nerves - aseptic meningitis - Bannwarth syndrome (radiculopathy) **Lyme arthritis** - polyartralgia, migrating arthritis **Lyme carditis** - dysrhythmia, myocarditis, pericarditis, AV block
Late LD	**Late disseminated stage** (months to years after infection)	**Acrodermatitis chronica atrophicans** **Late neuroborreliosis** - chronic progressive encephalitis, encephalomyelitis , chronic polyneuritis **Late Lyme arthritis – most oftens affect the knee, monoarthritis**

Lyme disease – EM

Erythema migrans

- develops not earlier than 36 hours after tick bite, lasts at least 3 days
- no itching or pain
- regional lymphadenopathy (rare)
- overall manifestations mild or none
- fatigue, muscle and joint pain, loss of appetite, subfebrile illness

Dg: clinical picture, serological examination is not indicated,

detection of antibodies unreliable

Lyme disease – neuroborreliosis

clinical manifestations

aseptic meningitis

aseptic meningitis + paresis of the cranial nerves

meningopolyradiculoneuritis (Bannwarth's syndrome)

+

lymphocytic pleocytosis

+

intrathecal synthesis of antibodies

Bannwarth‘s syndrome (meningopolyradiculoneuritis)

- **Aseptic meningitis**
- **Radicular pain caused by inflammation of the spinal roots**
  - usually with sensory disturbances and muscle weakness up to paresis of the affected limbs
  - lumbosacral nerves are most commonly affected
- in almost half of the cases **cranial neuritis-** paresis n.VII, less often paresis of oculomotor nerves

Woman, age 62

- 10-day persistent pain of Th, L spine with irradiation on the anterior abdomen, sensory deficit of Th 10-12 l. sin.
- no limb paresis, no fever.
- MRI of the spine - general degenerative changes, no spinal cord compression, no myelopathy.
- during hospitalization progression of severe pain with minimal response to analgesics
- LP - lymphocytic pleocytosis
- after 12 days transfer to our department

- Lab tests :normal basic biochemical and hematological parameters

Woman, age 62

- cephalosporins III.g. was used for suspicion of LNB
- intrathecal synthesis of Ab was positive
- treatment with partial effect (CTX 21 days)
- significant pain, which was present at the beginning of the disease, subsided during therapy
- only low doses of analgesics were needed for discomfort in the abdomen and hips

DG:

Bannwarth's syndrome, radiculitis Th12-L2, sensitive deficit in the lower abdomen and on the inner thighs, paresis of the muscles of the abdominal wall on the left

Woman, age 62

OUTPATIENT EXAMINATION
after 8 weeks

- the pain has practically disappeared
- the skin hypoesthesia of the abdomen and perigenital area disappeared
- slightly reduced skin sensitivity of the front of the right thigh persists
- the paresis of the muscles of the abdominal wall on the left lasts

Woman, age 62

OUTPATIENT EXAMINATION after 8 weeks

- patient feels good, she would like to return to work
- no back pain, but still abdominal muscles palsy on the left
- skin hypoesthesia almost completely disappeared
- rehabilitation of the abdominal muscles still continues

Woman, age 62

OUTPATIENT EXAMINATION

after 17 weeks

- goes to work, has no pain,
- normal neurological status

LEPTOSPIROSIS

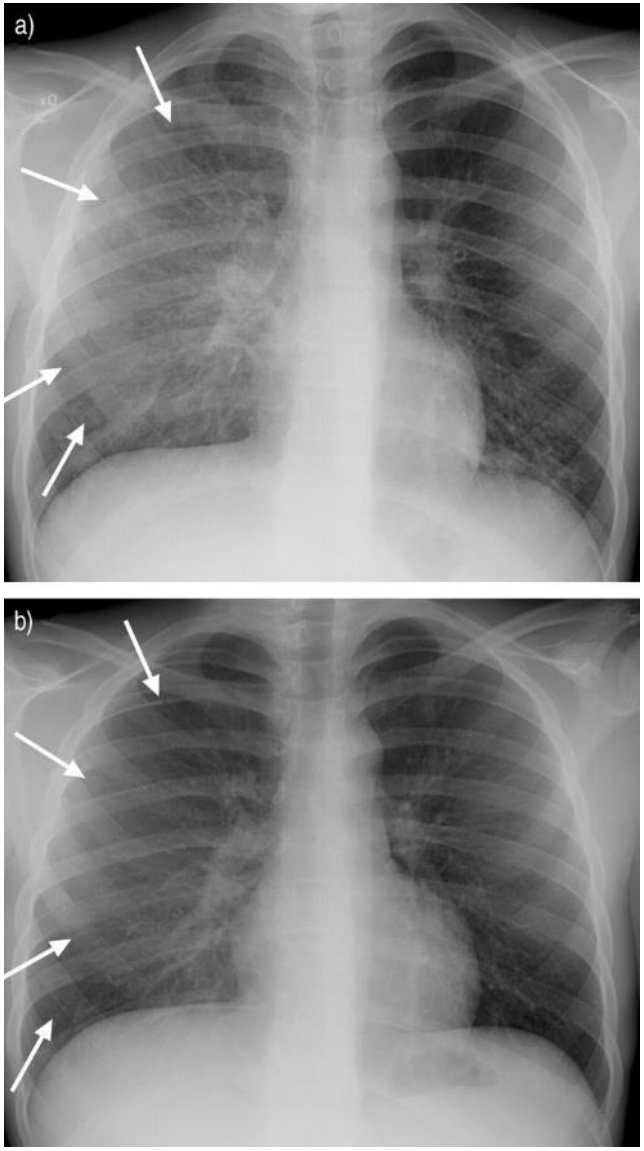
Diagnosis:

serology, specific antibodies appear in the second week of disease

PCR

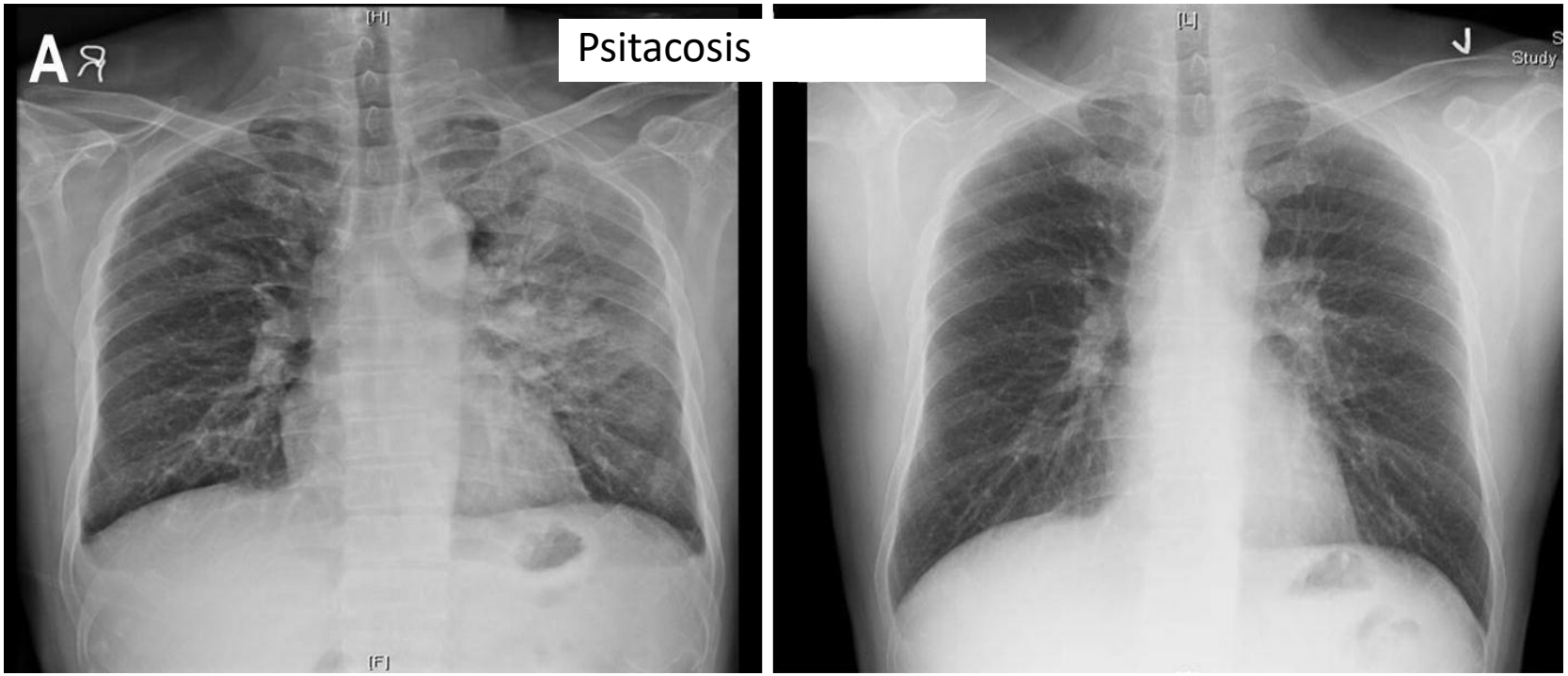
Therapy: antibiotics (PNC, AMPI)

Zoonoses – respiratory tract infection

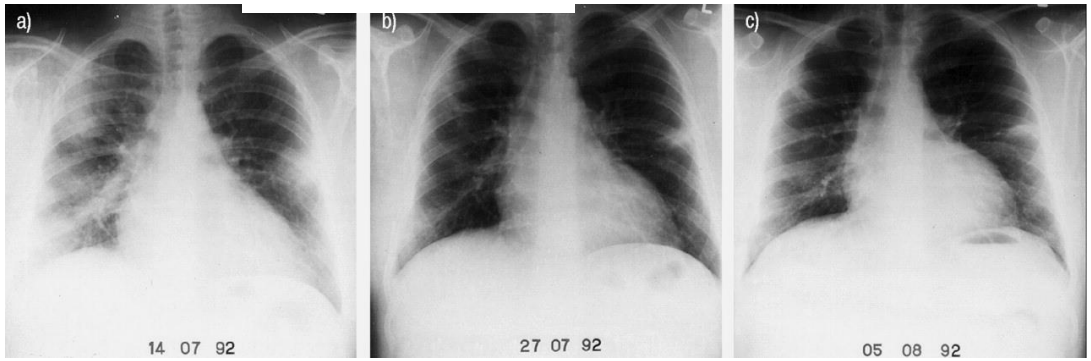


Pulmonary tularemia

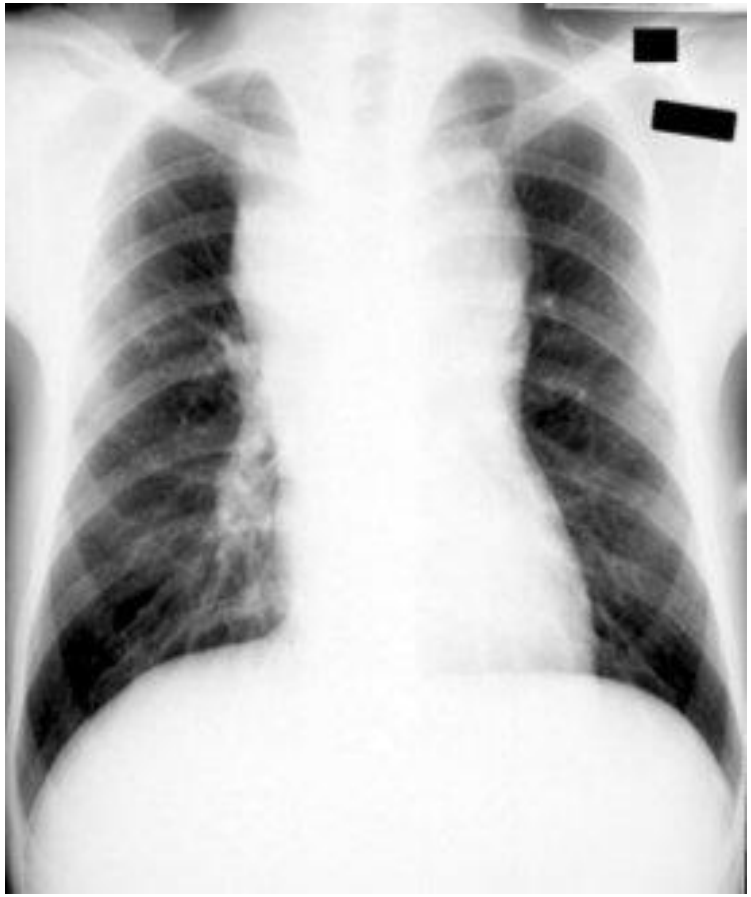
Psitacosis



Q fever



Legionella disease



anthrax

Fever + respiratory symptoms

- tularemia (pulmonary form)
- legionellosis
- ornitosis
- Q - fever
- bird flu

Zoonoses – dif.dg

Fever + lymphadenopathy	
regional	generalized
– tularemia – bartonellosis – yersiniosis	– toxoplasmosis – listeriosis – brucelosis – leishmaniosis

Fever + meningeal symptoms

listeriosis
TBE
Lyme disease (2.stage)
leptospirosis
ehrlichiosis
lymfocytic choriomeningitis

Zoonoses – dif.dg

Fever + gastrointestinal symptoms
- salmonellosis
- campylobacteriosis
- tularemia (abdominal form)
- yersiniosis
- bird flu

Fever + respiratory symptoms
- tularemia (pulmonary form)
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