Syphilis and Treponema pallidum

Syphilis	
Syphilis	
Syphilis, lues venerea	
Treponema pallidum	
Originator	*Treponema pallidum*
Transmission	sexual intercourse, through the placenta, contaminated objects
Incubation time	21 days (9-90 days).
Clinical picture	Primary stage - ulcus *durum* , regional lymphadenitis. Secondary stage - manifestations of generalization of infection: maculopapular rash on the skin and mucous membranes. Tertiary stage - organ changes, vascular damage and CNS damage.
Diagnostics	clinical picture, serological examination of antibodies, examination under shadow microscope, PCR
Infectiousness	the first and second stages are infectious, the third stage non-infectious
Therapy	penicillin, erythromycin
Vaccination	does not exist
Classifications and references	
ICD-10	A50
MeSH ID	D013587
MedlinePlus	000861
Medscape	229461

Syphilis (acute , *lues* or *lues venerea*) is a worldwide infectious chronic systemic disease with a characteristic course of alternating symptomatic and asymptomatic periods. It is transmitted mainly through sexual intercourse.

Treponema pallidum

The causative agent of syphilis is an anaerobic spiral fibrous bacteria belonging to spirochetes. It is primarily pathogenic to humans, who are its only host in nature.

Pathogenicity

Treponema pallidum is naturally pathogenic to humans only. There are three possible transmission paths:

1. sexual intercourse;
2. transmission by contaminated objects;
3. penetration through the placenta and infection of the fetus (adrenal syphilis develops).

By far the most common transmission is sexual intercourse, because the resistance *of T. pallidum* is very low and it dies quickly when away from the body. It is destroyed by oxygen, temperatures above 39 ° C, diluted solutions of common disinfectants and dies in blood cans within 4 hours. Transmission by contaminated objects is extremely rare.

Pathogenesis

Treponema pallidum penetrates the body through the mucous membranes or skin. It multiplies extracellularly and does not have a chemotactic effect on polymorphonuclear leukocytes. Virulent strains are coated with a mucous layer that prevents treponem from killing in phagocytes and from attaching complement or antibodies to its surface. Morphological changes (characteristic of all stages) are caused by capillary endothelial damage and dysfunction. It is not the bacteria themselves but the immunopathological processes that arise in response to the presence of *Treponem* in the body and which lead to extensive destruction at a time when *Treponemas* are almost eliminated from the body.

Causes of syphilis symptoms

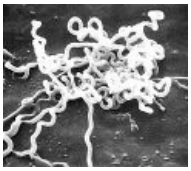
1. capillary endothelial damage;
2. immunopathological changes:
    - immunocomplexes of antigens and antibodies (antigens are largely released during the breakdown of bacteria);

- cell hypersensitivity.

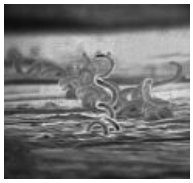
Immunity

Specific immunity (both antibody and cellular) against treponemas develops during the course of the disease. Cell hypersensitivity is due to activation of macrophages. Immune reactions can lead to complete recovery in the untreated. Protection against a new infection is only in the early stages, reinfection is possible.

- It can affect virtually any tissue or organ, and untreated disease can cause death or disability. The infection can be transmitted to the fetus during pregnancy (early and late congenital syphilis).



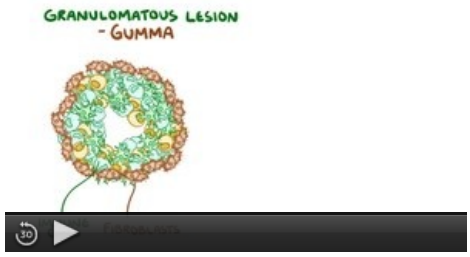
T. pallidum in an electron microscope.



Treponema pallidum .

Characteristics

- Incidence in the Czech Republic in 2009: 997 cases (ie 10 per 100 thousand inhabitants, of which 22% of foreigners), in 2006: 502 cases (5 per 100 thousand inhabitants).
- Originator: spirocheta *Treponema pallidum* .
- Transmission: sexual intercourse, transplacental, blood transfusion.
- Place of entry: any area of skin and mucous membranes, most often the genital, rectal and oral mucosa.
- Incubation period: 21 days (9-90 days).
- Risk groups: drug addicts , patients with AIDS .
- It is infectious from primary infection until the end of the 2nd year of the disease (ie stage 1 and 2). Isolation in the venereology department is therefore mandatory in patients with stage 1 and 2 syphilis.
- Subject to reporting.
- There is no vaccination.



Syphilis (video).

Clinical picture

Acquired syphilis typically develops in three clinical stages.

Diagnostics

- clinical picture, anamnesis
- indirect diagnostics - serological examination of antibodies:
  - non-treponemal tests (VDRL, RRR) - show non-specific anticardiolipin antibodies ("reagins")
  - treponema tests (TPHA, FTA, ELISA, ...) - show specific antibodies against *Treponema pallidum antigens*
  - Non-specific antibodies disappear within weeks to months after effective treatment, but specific IgG remains detectable over a long period of time.
- direct diagnostics:
  - examination under a shadow microscope
    - basic, standard examination in case of suspicion of a specific lesion on the skin or mucosa
    - allows to examine serous exudate from lesions, cerebrospinal fluid, amniotic fluid, etc.
  - PCR (polymerase chain reaction)
  - direct immunofluorescence antigen detection (DFA-TP) - expensive, not available in our country
  - histopathological examination (silver impregnation) - lengthy and burdened with a number of artifacts

Serological examinations

- **RRR** (Rapid Reaction Reaction) or RPR (Rapid Plasma Reagin) - a cheap *screening test*
  - microflocculation reactions where the antigen is cardiolipin mixed with cholesterol and lecithin
  - Cardiolipin is a phospholipid (hapten) contained in the membrane of Treponema pallidum, but also in other bacteria and also in mitochondria
  - anti-cardiolipin antibodies produced in syphilis are called "reagents"

        - clusters form when the antigen meets the antibodies
        - may be false positive (for tumors, pregnancy, malaria), therefore positivity needs to be confirmed eg by TPHA test
    - **VDRL** (Veneral disease research laboratory) microscopic test
        - can be used to test serum and cerebrospinal fluid
    - **TPHA** (Treponema pallidum hemagglutination) or MHA-TP (microhemagglutination of T. pallidum) - cheap ***screening test***
        - antigen, *T. pallidum ssp. pallidum Nichols* (so-called Nichols strain) is bound to chicken erythrocytes
        - agglutination occurs when this antigen meets serum antibodies
        - TPHA sensitivity in primary syphilis is 69-90%, in secondary syphilis 100% and later around 97%. Specificity is at least 98%
    - **FTA-ABS** IgG, IgM (fluorescent treponemal antibody - absorbed)
        - allows diagnosis from the 2nd to the 3rd week of syphilis
        - the antigen is the Nichols strain of T. p. pallidum
        - a so-called sorbent is used to remove cross-reactive antibodies from the patient's serum
        - the patient's antibodies are labeled with a fluorescent dye and fluorescent upon reaction with the antigen
        - evaluation of the immunofluorescence response is subjective
        - tends to be false positive in systemic lupus erythematosus
    - **ELISA**
    - **Westernblot** (WB)
    - Previously, Nelson's (-Mayer's) Treponema pallidum immobilization test (TPIT) and BWR (Bordet-Wasserman reaction) - complement fixation reaction - were used.

Laboratory diagnosis of neurosyphilis

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    - examination of serum and cerebrospinal fluid
    - positive treponemal (specific) serological tests (TPHA, ELISA, etc.) are a prerequisite
    - in cerebrospinal fluid there is lymphocytic pleocytosis , protein cytological association , proliferation of immunoglobulins
    - positive intrathecal antibody production and TPHA index
    - WHO (1982) neurosyphilis criteria: pleocytosis, cardiolipin test positivity (CSF-VDRL) and elevated cerebrospinal fluid protein levels.
    - The negativity of specific cerebrospinal fluid tests is sufficient to rule out neurosyphilis in a patient with positive seroreactions.

Clinical picture

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    - we most often encounter cranial nerve damage , which is typical for all stages
    - Argyll-Robertson pupils (anisocoric, conspicuously narrow, non-rounded, do not respond to exposure in a preserved response to convergence) - cause: lesion n. III or directly mesencephalus
    - papillary edema followed by atrophy n. II and blindness
    - deafness is a consequence of the lesion n. VIII

Therapy

-
    - Penicillin is a causal treatment for all stages of the disease
    - All forms of syphilis are treatable, the better the results the sooner penicillin is administered
    - Penicillin G iv 2–4 MIU after 4 hours 10–14 days
    - Procaine penicillin G im 2-4 MIU (and probenecid 500 mg / 6 h) after 24 hours - total 15 days
    - Pendepon (benzathine benzylpenicillin) im 2-4 MIU every 7 days for 3 weeks
    - Jarisch-Herxheimer reaction - fever and tachycardia at the beginning of treatment, the body's response to endotoxin from decayed spirochetes, responds favorably to corticoid treatment
    - in case of hypersensitivity to penicillin → to erythromycin or tetracycline for 30 days
    - tobacco crisis (see below) will affect atropine in part

Acute syphilitic meningitis

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    - The first manifestation of untreated neurolues, has 3 forms:

1. asymptomatic - most, shows only an accidental finding in the cerebrospinal fluid after lumbar puncture
2. aseptic - fatigue, meningeal syndrome , febrile rash
3. acute basal - hydrocephalus , cranial nerve lesions (n. VII, VIII), papillary edema n. II
    - fluid: lymphocytosis 100–1000 cells / mm 3 , increased protein 0.5–2 g / l, sugars decreased, RRR +
    - treatment: 1st and 2nd stage - penicillin

Late neurological consequences of untreated syphilis

-
    - rare
    - in those not treated at the earliest 5 years after the infection during the latency period

[1]

Meningovascular syphilis (5-10 years)

- obliterative endarteritis causes stroke
- brain-based granulation causes cranial nerve palsy

Spinal syphilis (10-15 years)

- the spinal cord is affected by subpiically chronic meningitis
- radicular pain, upper limb muscle atrophy, paraplegia

Optical atrophy (in 10-15 years)

- chronic meningitis and subpilar necrosis exclusively around n. II unilaterally and bilaterally
- the field of view narrows
- cerebrospinal fluid is always the same in untreated syphilis with late neurological consequences: lymphocytic pleocytosis 100 / mm 3 , increased protein and gamma globulins, positive serology
- penicillin is the drug of choice

Progressive paralysis (in 15-20 years)

- **subacute meningoencephalitis** with predominantly frontal involvement
- very rare today
- neurasthenia → affective disorders (mania), concentration, behavior, judgment and memory → progressive dementia - ***preparalytic stage***
- ***paralytic stage*** - + corticospinal, extrapyramidal, myoclonus, or Argyll-Robertson pupil , dysarthria, epilepsy → ends with personality breakdown and general marasmus, sudden death - ictus paralyticus
- most often - megalomaniac form - giant delusions, depressive form, simplex form - picture of progressing dementia, Lissauer form - neurological symptomatology, cerebrospinal fluid paralysis - cerebrospinal fluid finding, without clinical manifestation
- dg: seropositivity - treponemal reactions (TPHA, TPI)
- fluid: lymphocytic pleocytosis 50 / mm 3 , protein 0.5–2 g / l, gamma globulins +++, RRR +
- autopsy: brain atrophy. cortex, diaper thickening, lymphocyte and plasma cell infiltrates around blood vessels, microglia proliferation, neuronal degeneration
- penicillin in 40% of patients stops progression (formerly Wagner-Jauregg - malariotherapy)

Tabes dorsalis (in 15-20 years)

- **atrophy of the posterior cords and spinal roots**
- Inflammation occurs on the spinal cord as chronic progressive meningitis
- very rare
- it also affects the optic nerves (20% have papillary atrophy)
- ataxia , balance disorders, hyporeflexia (up to patellar and Achilles tendon areflexia), lanceic lower limb pain, trophic and pupil changes (90% miotic unresponsive to exposure, positive Romberg's symptom
- **tobacco crises** = visceral colic that faithfully mimics a sudden abdominal event - pain, nausea, vomiting, tenezms, diarrhea
- bouts of burning paresthesia often affect the sciatic nerve and the ulnar nerve
- incontinence, impotence, genital numbness
- painless joint deformities, trophic ulcers on the leg (malum perforans) are the result of trauma from a lack of proprioception , with no hope of improvement
- cerebrospinal fluid is closer to normal than in progressive paralysis, IgG, lymphocytic pleocytosis to several dozen elements is increased. moderate hyperproteinosis, RRR is up to 30% negative
- syphilitic gum is extremely rare, as infiltrative limited inflammation affects other organs in addition to the brain, such as the liver

Bone syphilis

- rare
- **adrenal syphilis**: osteochodritis of the growth plates of long bones, reactive periostitis (doubling of the bone line)
- **acquired syphilis**: negumous periostitis with skull and tibia involvement / gummy syphilitic periostitis and destructive osteomyelitis (defects in the skull, perforation of the palate , compression fractures of the vertebrae)

Links

related articles

- Congenital syphilis
- Tabes dorsalis (preparation)

External links

- Syphilis (Czech wikipedia)
- Syphilis (English wikipedia)

Source

1. POLÁČKOVÁ, Z. Sexually transmitted diseases I. part. *Dermatology for practice* [online] . 2008, vol. 2, pp. 74-76, also available from < http://www.solen.cz/pdfs/der/2008/02/06.pdf >.
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10. ↑Jump up to:a b http://www.szu.cz/tema/prevence/syfilis-tradicni-choroba-soucasny-problem-ii
11. ↑Jump up to:a b http://mikrobiologie.lf3.cuni.cz/mikrobiologie/bak/uceb/obsah/lues/lues.htm
12. ↑Jump up to:a b http://www.medmicro.info/portal/syfilis/lvl3/ch09s05.html
13. ↑Jump up to:a b c http://www.medmicro.info/portal/syfilis/lvl3/ch05s03.html
14. ↑ POVÝŠIL, Ctibor and Ivo ŠTEINER, et al. *Special pathology.* 2nd edition. Prague: Galén-Karolinum, 2007. pp. 297-299. ISBN 978-80-7262-494-2 .

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Inflammation	
Specific inflammations	leprosy • tuberculosis • sarcoidosis • syphilis • and others
Non-specific inflammations	alternative inflammation: -
	exudative inflammation: surface: skin: fibrinous • putrefactive • purulent • lymphoplasmocytic • serous; mucous membranes: fibrinous • putrefactive • purulent • catarrhal (serous) • catarrhally purulent • lymphoplasmacytic • ulcerative; serous membranes: fibrinous • putrid • purulent • serous
	exudative inflammation: deep (interstitial): fibrinous • gangrenous • purulent • lymphoplasmocytic • serous
	proliferative inflammation: -
Portal: Pathology	

Category :

- Neurology
- Microbiology
- Dermatovenerology
- Infectious diseases
- Pathology

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