

Tuberculosis meningitis

Tuberculosis (tbc) affects the brain in 1% of patients^[1]. The maximum number of pathological changes is in tanks and meningeal base brain – therefore **basilar meningitis**.

- course of subacute
- always a secondary disability
- thickening meninges, exudate, vasculitis

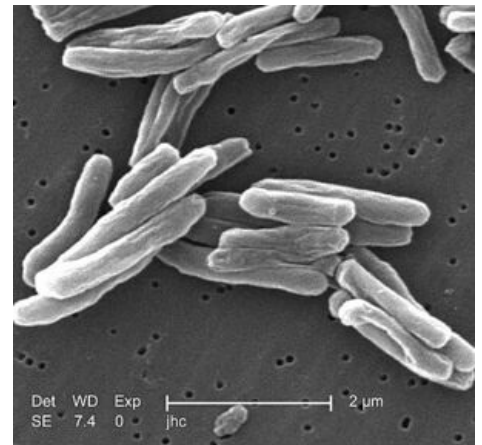
preview *Mycobacterium tuberculosis* in an electron microscope

Etiology and pathogenesis

Mycobacterium tuberculosis (Koch's bacillus) or *Mycobacterium bovis* penetrates hematogenously into the CNS, where it forms encapsulated deposits. Bacteremia is primary in **children**, pulmonary tbc. Adults **become** ill even years after the primary infection, often from a lesion located in the nodule or vertebra. Mycobacteria are released from encapsulated foci and the infection spreads subarachnoidally and into the basal cisterns, directly into the tissue brain or spinal cord or into the choroid plexus. Older people today are more ill

Clinical signs

Clinical signs are due to the following: self-infection, exudation with obstruction of the basal cisterns and hydrocephalus, vasculitis causing heart attacks and mission from perivascular inflammation. The greatest is brain-based meningitis in the form of meningitis (arachnoiditis). The onset of the disease is **usually inconspicuous**, lasts a few days to 2 weeks. Manifestations include mood disorders, drowsiness, disorders of concentration, temperature (subfebrile, may be absent)^[2], perspiration, headache (alternating or permanent) and abdomen. In the **2nd phase**, the night confusion begins, paresis cranial nerves, acute-delirious conditions, obliterative endarteritis → paresis, ataxie, dysarthria, epileptic seizures and meningeal symptoms, such as typical headache and neck pain. Untreated will go **to phase 3**, which is coma. Sedimentation and leukocyte they are in blood increased. Serious consequences of basilar meningitis are adhesions, it is often affected e.g. n. II with consequent blindness. Not treated progressively cachectizing. alteration consciousness over time results in coma, usually within days to weeks.^[2] Sick people usually die on failure even 3 weeks from the first manifestations. The treatment of basilar meningitis takes weeks to months, the patient is able to return to normal life in 8-12 months. Among the most common **permanent consequences** belongs to blindness, hydrocephalus and paraplegia from spinal cord endarteritis. A rarer form of the disease is **tubercle**, which is a limited site of infiltrative inflammation in the CNS containing caseous necrosis, crisis, calcification, lympho- and leukocyte infiltrate.



Mycobacterium tuberculosis in an electron microscope

Diagnostics

Diagnostic indicators of basilar meningitis include increased sedimentation, anemia and leukocytosis. Hyponatremia is accompanied by the syndrome of inappropriate secretion ADH. The diagnosis is determined **liquor**, in which lymphocytes and polymorphonuclear cells increase up to 500 / mm³, protein increase to 1–4 g / l with globulin growth, glucose below the surface to 2/3 glycemia. Chlorides are reduced. **Microscopically, the detection of Koch's** cerebrospinal fluid bacillus is positive in 20% in Ziehl – Nielsen staining. It is possible to cultivate mycobacteria on Lowenstein-Jensen soil from cerebrospinal fluid. Direct ID is possible with DNA – **PCR**. A X-ray of the lungs in 50-70% shows signs of recent or old TB. The **skin tuberculin test** (Mantoux more than 10 mm) is 90% positive, it can be negative during steroid treatment or in case of fresh primary tuberculosis. On **CT a MRI** we can observe wider chambers, heart attacks, after contrasting coloring meninges, possibly also tuberculomas.

Tuberculosis treatment

Tuberculosis treatment lasts for weeks to months and is **combined**. We start with a triple combination:

- isoniazid (INH)
- rifampicin
- pyrazinamide (2 months)

The following is a double combination:

- isoniazid
- rifampicin (7 months)

Assuming possible resistance to previous anti-tuberculosis treatment (developing countries, history of tuberculosis), we add as the 4th drug streptomycin or ethambutol. **Corticosteroids** we serve at: deterioration of consciousness or worsening of neurological symptoms. The treatment quickly normalizes the sugar level in moku, protein and cells return to normal within 3-4 months. The **prognosis** depends on age and condition, the development of arachnoiditis and vascular complications. **Mortality** in the case of early treatment, it is around 10%, late to around 50%. In about 30%, tuberculosis meningitis **leaves permanent consequences**, such as hemiplegia, blindness, deafness, hypothalamic-pituitary dysfunction, demencia, epilepsy^[1].

Links

related articles

- Tuberculosis (pneumology) • Tuberculosis (pathology) • Tuberculosis (pediatrics) • Potts disease
- Meningitis • Meningitis (pediatrics)
- Viral meningitis • Serious meningitis a meningoencephalitis • Herpetic meningoencephalitis
- Purulent meningitis (infection) • Purulent meningitis (pediatrics) • Hemophilic meningitis
- Infectious brain disease • Neuroinfection, CNS / PGS inflammation • Encephalitis

References

1. https://www.wikiskripta.eu/w/Tuberkul%C3%B3zn%C3%AD_meningitida#cite_ref-Seidel_1-0
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2. https://www.wikiskripta.eu/w/Tuberkul%C3%B3zn%C3%AD_meningitida#cite_ref-Herchline_2-0
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1. **Cite error: Invalid <ref> tag; name "Seidel" defined multiple times with different content**
- 2.

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