Tuberculous meningitis

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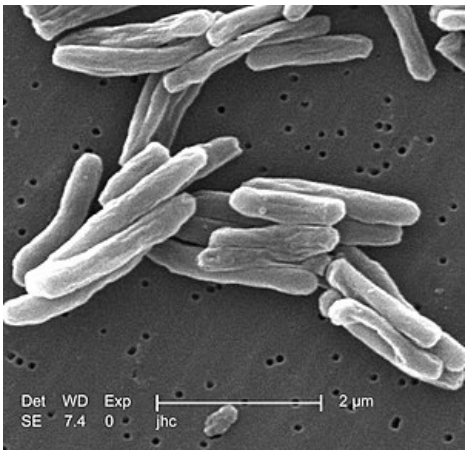
Tuberculosis (TB) affects the brain in 1% of patients[1]. The maximum of disease changes is in the cisterns and membranes of the base of the brain - hence **basilar meningitis**.

- course subacute
- always a secondary disability
- meningeal thickening, exudate, vasculitis



Mycobacterium tuberculosis in the electron microscope

Etiology and pathogenesis

Mycobacterium tuberculosis (Koch's bacillus) or *Mycobacterium bovis* penetrates hematogenously into the CNS, where it forms encapsulated foci. Bacteremia belongsto primary pulmonary tuberculosis in **children**. **Adults** get sick even years after the primary infection, often from a deposit located in a node or vertebra. Mycobacterium is released from the encapsulated deposits and the infection spreads subarachnoidally and into the basal cisterns, directly into the tissue of the brain or spinal cord or into the choroid plexus. Older people are more likely to be sick today

Clinical signs

Clinical symptoms are given by the following: own infection, exudation with obstruction of the basal cisterns and hydrocephalus, vasculitis causing infarctions in the brain and spinal cord from perivascular inflammation. The greatest involvement of the meninges at the base of the brain is in the form of meningitis (arachnoiditis). The **onset** of the disease is usually inconspicuous, lasting a few days to 2 weeks. Symptoms include mood disorders, sleepiness, concentration disorders, temperature (subfebrile, may be absent[2]), sweating, headaches (alternating or permanent) and abdominal pain. In **the 2nd phase**, nocturnal confusion, cranial nerve paresis , amento-delirious states, obliterating endarteritis → paresis, ataxia ,dysarthria , epileptic seizures and meningeal symptoms, which include, for example, typical headache and neck muscle pain. Untreated, they go into **stage 3,** which is a coma. Sedimentation and leukocytes in the blood are increased. Adhesions are serious consequences of basilar meningitis, it often affects, for example, n. II with subsequent blindness. Untreated, they progressively cachectate . Alteration of consciousness eventually results in coma, usually within days to weeks.) Patients usually die from circulatory failure even 3 weeks after the first symptoms Treatment of basilar meningitis takes weeks to months, the patient is usually able to return to normal life in 8-12 months. Among the most common **permanent consequences** are blindness, hydrocephalus and paraplegia from spinal vascular endarteritis. A rarer form of the disease is **a tubercle** , which is a circumscribed focus of infiltrative inflammation in the CNS containing caseous necrosis , calcification, lympho- and leukocyte infiltrate.

Diagnostics

Diagnostic indicators of basilar meningitis include increased sedimentation , anemia , and leukocytosis . Hyponatremia accompanies the syndrome of inappropriate ADH secretion . The diagnosis is established by the cerebrospinal fluid, in which there is an increase in lymphocytes and polymorphonuclear cells up to 500/mm 3 , an increase in protein to 1–4 g/l with an increase in globulins, glucose below the level of 2/3 of glycemia . Chlorides are reduced. **Microscopically**, proof of Koch bacillus from the cerebrospinal fluid is positive in 20% in Ziehl–Nielsen staining. From the cerebrospinal fluid, mycobacteria can be cultured on Lowenstein's medium. Direct evidence is possible using DNA - PCR. A chest x-ray will show signs of recent or old TB in 50-70% of cases. The **skin tuberculin test** (Mantoux more than 10 mm) is positive in 90%, it can be negative during treatment with steroids or with a recent primary TB infection. On **CT and MRI** we can observe wider ventricles, heart attacks, staining of the meninges after contrast, or even tuberculomas.

Treatment of tuberculosis

Tuberculosis treatment lasts 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 TB), we add streptomycin or ethambutol as the 4th drug. Corticosteroids are given in case of: deterioration of consciousness or worsening of neurological symptoms. The treatment quickly normalizes the level of sugar in the fluid, protein and cells return to normal within 3-4 months. **Prognosis** depends on age and condition, development of arachnoiditis and vascular complications. Mortality is around 10% in the case of early treatment, up to 50% in late treatment In about 30%, tuberculous meningitis leaves **permanent consequences**, e.g. hemiplegia, blindness, deafness, hypothalamopituitary dysfunction, dementia, epilepsy.

Links

Related Articles

- Tuberculosis (pneumology)
- Tuberculosis (pathology)
- Tuberculosis (pediatrics)
- Pott's disease
- Meningitis
- Meningitis (pediatrics)
- Viral meningitis
- Serous meningitis and meningoencephalitis
- Herpetic meningoencephalitis
- Purulent meningitis (infection)
- Purulent meningitis (pediatrics)
- Haemophilic meningitis
- Infectious diseases of the brain
- Neuroinfections, CNS/PGS inflammations
- Encephalitis

References

1. SEIDL, Zdeněk – OBENBERGER, Jiří. *Neurology for study and practice.* 1. edition. Praha : Grada Publishing, 2004. 363 pp. ISBN 80-247-0623-7.
2. HERCHLINE, T – AMOROSA, J.K. *https://emedicine.medscape.com/* [online]. ©2009. [cit. 27.6.2009]. <https://emedicine.medscape.com//article/230802-overview>.