Suppurative meningitis (infection)

This article has been translated from WikiSkripta; ready for the **editor's review**.

Suppurative meningitis belongs to the most serious *acute* diseases. Her **course is very fast**. It leaves **permanent effects** or ends in **death**. It is an acute infection of the subarachnoid spaces and meninges characterized by the presence of polymorphonuclears in the CSF.

Characteristics

This is an urgent condition in neurology accompanied by encephalitis. Typical manifestations are acute meningeal syndrome, cephala, vomiting, fever with photophobia and psychological changes.

Etiology

According to the method of origin:

1. **primary** - infection occurs *hematogenously*
2. **secondary** - transition from the *surrounding bearing* (middle ear, petrosal axis, paranasal sinuses)
    - insertion in *infective endocarditis*
    - predisposing factors – head injuries, subdural barrier disorder, etc.

The bacterial spectrum differs significantly in newborns, children and adults:

1. **neonates**: G- rods: *E. coli*, *Klebsiella*, *Haemophilus influenzae*
2. **children**: *Haemophilus influenzae*, pneumococcus, meningococcus
3. **adults**: pneumococcus, meningococcus

The causative agents are diverse - Listeria monocytogenes, Streptococcus pyogenes, Staphylococcus aureus, fungal agents or amoebas (rarely) also play a role here.

Risk factors: ethylism, diabetes mellitus, hyposplenism, AIDS.

Pathogenesis

Bacteria penetrate the meninges hematogenously (usually from a distant site of inflammation), or porogenically from inflammation in the surroundings (otitis, sinusitis, etc.). Another mechanism of formation is trauma with a breach of dura mater - communication between the external and intracranial space is created. Rarely, the source of infection is iatrogenic (lumbar puncture, infected shunt).

Pathological-anatomical picture

- Congestion of the meninges with polymorphonuclear infiltration;
- blood-brain barrier broken;
- purulent exudate from the basal cisterns into the convexity sulci;
- arteritis and venous thrombophlebitis of subarachnoid vessels;
- brain itself is not affected (an intact pia mater prevents the formation of abscesses);
- edema and ischemia of the brain;
- the condition can be complicated by thrombosis and subsequent infarcts of the brain;
- enlarged cerebral ventricles, involvement of cranial nerves in cisterns frequent (oculomotor, n. VII, n. VIII);
- healing is accompanied by scarring, with hydrocephalus.

Diagnosis

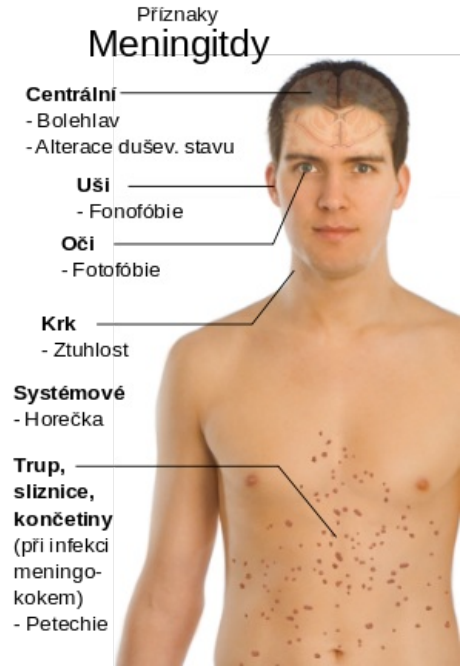
The diagnosis must be made **as quickly as possible**, after admission to the hospital, the diagnosis should be determined and the treatment started **within 30 min—1 hour**. Anamnesis will help with **secondary** - repeated inflammations of the middle ear or sinuses.

- blood collection for hemoculture;
- inflammatory cerebrospinal fluid during lumbar puncture (intracranial hypertension must first be ruled out!);
- fluid colored green-yellow during lumbar puncture, cells are 100–10,000/mm^3 (80–90% polymorphonuclears), sugar level reduced (0.3 g/l), protein increased (0.5 g/l), increased lactate dehydrogenase, chlorides normal;
- sediment stained according to Gram will be examined microscopically;
- serological + immunological tests determine capsular antigen in the cerebrospinal fluid by immunoelectrophoresis;
- a **latex agglutination test** is also performed at the bedside - detection of antibody in fluid (quick orientation about the causative agent);
- with the aim of examination of the collected fluid, proof of the infectious agent directly or by culture + apply

optimal treatment according to sensitivity, culture is negative in 10-20%;
- we choose the appropriate ATB according to: age, severity, sampling results.

Clinical course

Primary purulent meningitis is manifested by a very rapid worsening of the condition, in contrast to **secondary**, which has a more protracted course. It progresses from full health to a typical image within 24-36 hours. The patient is hypersensitive to light and sounds, has severe headaches, rising temperature. meningeal symptoms, confusion and malaise occur. Concomitant impairment of consciousness occurs in about 90% of patients, bradycardia occurs with brain edema. Focal cerebral symptoms include hemiparesis and epileptic seizures. There is damage to the cranial nerves, hl. oculomotor and n. VII and VIII. Other complications include septic ones (pyogenic arthritis, acute bacterial endocarditis), failure of vital functions, development of shock and disseminated intravascular coagulation (DIC) may occur within a few hours. . The development is so rapid that it may resemble stroke (CMP), aggression and disorientation may occur. In meningococcal and hemophilic meningitis, we find petechia and suffusion on the skin. Newly formed petechiae, larger than 2 mm, confluent and located on the thighs and abdomen are typical for meningococcal disease. Differentiation from ordinary urticaria is possible by pressure (exanthem under pressure below with a glass it fades to disappear, petechiae persist ("slide method")[1] (ht tps://www.khszlin.cz/wcd/pages/extranet/organizacni-struktura/odbor-proti epidemicky/legislativa/vest_10_2006.pdf).



Symptoms of meningitis

Complications

Complications of purulent meningitis are numerous. The **acute stage** can be accompanied by *cerebral edema* which can cause visual or auditory impairment or central palsy. In the **recovery phase**, *parainfectious arthritis, myocarditis, headaches and fatigue* appear. Children may then have *psychomotor retardation or hydrocephalus*.

Prognosis

Symptoms usually 3-5. day of treatment subsides and the patient gradually improves. Early diagnosis, adequate therapy and resistance of the organism are decisive. In practice, the disease has a twofold course:

1. **mild:** unremarkable symptoms, quick correction of findings in the cerebrospinal fluid
2. **severe:** sick from the beginning in a coma, death can occur within 24 hours under the guise of shock, a frequent complication of bleeding into the adrenal glands (Waterhouse-Fridrichsen) or DIC

Differential diagnosis

- if there is no anamnesis and the impairment of consciousness progresses → rule out CMP, subarachnoid hemorrhage, metabolic comatose states (DM), poisoning, etc.
- other meningitis (serous, tuberculous, mycotic), brain abscess, epidural empyema/abscess (intracranial or spinal), subdural empyema, infective endocarditis with CNS embolizations, sinus thrombophlebitis, dermoid cyst rupture, brain tumors
- the diagnosis will be determined by examination of the cerebrospinal fluid

Treatment

1. **causal**: antibiotics
2. **symptomatic**: antiemetics, analgesics, antiedema preparations, infusion

- early ATB treatment (IMMEDIATELY!, control CSF in 1-2 days)
- a doctor can save a life by administering penicillin before transport to the hospital
- **Third-generation cephalosporins** are optimal today (e.g. cefotaxime, ceftriaxone)
- the chosen ATB must penetrate the blood-brain barrier well (not tetracycline, partly aminoglycosides), dosage in children is based on weight and age
- chloramphenicol can rarely lead to aplastic anemia, moreover not suitable for children under 5 years
- the source of meningitis can be another inflammatory focus in the body = **secondary meningitis**: start treatment in time and look for a possible cause
- correction of the internal environment with regard to ADH secretion
- we correct possible brain edema, administer vitamins and other symptomatic therapy
- ATB is planted no earlier than 10-14 days after the normalization of the temperature
- meningococcal meningitis is contagious (vaccination, prophylaxis in family members)

Types of purulent meningitis

Pneumococcal purulent meningitis

Pneumococcal purulent meningitis occurs at any age, and is usually of secondary etiology. **Pathogenic** are **encapsulated strains**. It has the most severe course in weakened persons (alcoholics, cirrhotics) and in splenectomized persons (fulminant course). It can be *primarily transmitted through the air* (mainly in winter), but most often occurs **secondarily**. The infectious agent **multiplies in the respiratory tract**, from there it spreads through the *blood* to the brain. It can also get into the brain by transferring from the *sinuses or ear*. The formation of **abscess** is not rare either. In the CSF there is a typical picture for purulent meningitis, '*microbiology is necessary*.

Meningococcal meningitis and sepsis

Meningococcal meningitis is associated with manifestations of a **systemic response of the organism**. **In our country**, the disease is caused mainly by **serotypes**: B, C, A, Y, it is transmitted through **the air**. It often occurs **after exhaustion** (sport, disco, late night, etc.). Manifestations:

- sometimes initially 1-2 days symptoms of pharyngitis, fatigue and paineven stomach
- followed by fever, vomiting, impaired consciousness
- petechiae and suffusion appear on the skin
- development of DIC and shock - **multiorgan involvement**

The most severe cases end within a few hours under the image of **Waterhouse-Friderichsen syndrome** (bleeding into the adrenal glands).

Sepsis has a higher mortality rate than meningitis. In sepsis, the finding in cerebral fluid is normal.

Shunt Meningitis

Shunt meningitis occurs in children with hydrocephalus ([Hydrocephalus|hydrocephalus)] having drain plugs installed (infection can occur).

Other neurological damage

One of the possible damages can be palsy cranial nerves or motor disorders (e.g. hemiparesis or quadriparesis, ataxia). Furthermore, cerebral ischemia, diencephalohypophyseal syndrome or SIADH (syndrome of inappropriate secretion of antidiuretic hormone) can occur, which is manifested by oliguria and CSWS (cerebral salt wasting syndrome) manifested by polyuria.

Links

External links

Standard of effective clinical care – invasive meningococcal disease Bulletin of the Ministry of Health of the Czech Republic 2006 (https://www.mzcr.cz/wp-content/uploads/wepub/3674/9337/V%C4%9Bstn%C3%ADk%2010-2006.pdf) Page 18

Related Articles

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Source

- ws:Hnisavá meningitida (infekce)
- BENEŠ, Jiří. *Study materials* [online]. [cit. 2010]. <http://jirben.wz.cz>.

References

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