Bacterial meningitis (infection)

Bacterial meningitis is one of the most serious *acute* diseases. Its **progress is very fast**. It leaves **permanent consequences** or ends in **death**. It is an acute infection of the subarachnoid spaces and meninges characterized by the presence of polymorphonuclear cells in liquor.

Characteristics

It is an urgent condition in neurology accompanied by encephalitis. Typical manifestations are acute meningeal syndrome, cefalea, vomiting, fever with photophobia and mental changes.

Etiology

According to the method of origin:

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

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

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

The causative agents are diverse - they include: Listeria monocytogenes, Streptococcus pyogenes, Staphylococcus aureus, mycotic agents or amoebae (rarely).

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

Pathogenesis

Bacteria penetrate the meninges hematogenously (usually from a distant site of inflammation), or porogenically from surrounding inflammation (otitis, sinusitis, etc.). Another mechanism of occurrence is trauma with disruption of the 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 meninges with polymorphonuclear infiltration;
- blood-brain barrier breached;
- purulent exudate from basal cisterns into convex sulks;
- arteriitis and venous thromboflebitis of subarachnoid vessels;
- brain is not affected by itself (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;
- cerebral ventricles dilated, involvement of cranial nerves in cisterns common (oculomotor, n. VII, n. VIII);
- Healing is accompanied by scarring, with hydrocephalus.

Diagnosis

Diagnosis should be made **as quickly as possible**; after admission to hospital, diagnosis should be made and treatment started **within 30 min-1 h**. In **secondary** , a history - repeated inflammation of the middle ear or sinuses will help.

- blood collection for hemoculture;
- Inflammatory cerebrospinalfluid at lumbar puncture (intracranial hypertension must be excluded before !);
- fluid stained greenish yellow on lumbar puncture, cells is 100–10 000/mm^3 (80-90 % polymorphonuclear cells), sugars decreased (0.3 g/l), protein increased (0.5 g/l), lactate dehydrogenase increased, chloride normal;
- Gram-stained sediment is examined microscopically;
- Serological + immunological tests determine immunoelectrophoretically the capsular antigen in cerebrospinal fluid;
- a **latex-agglutination test** is also performed at the bedside - detection of antibodies in the fluid (quick orientation about the causative agent);
- the aim of the examination of the collected fluid is the identification of the infectious agent directly or by culturing according to the sensitivity to use the optimal treatment, the cultivation is negative in 10–20%;
- we choose a suitable ATB according to: age, severity, result of samples.

Clinical course

thumb|Symptomy meningitidy **Primary** purulent meningitis manifests itself in a very rapid deterioration of the condition, in contrast to **secondary**, which has a more prolonged course. From full health, it progresses to a typical image within 24-36 hours. The patient is hyperpathic, has severe headache, is photophobic, rising temperature. There are meningeal symptoms, confusion and reluctance. Accompanying disorder of consciousness occur in about 90% of patients, bradycardia is seen in edema of the brain. Cerebral symptoms include hemiparesis and epileptic seizures. The cranial nerves, mainly the oculomotor nerves and n. VII and VIII, are affected. Other complications include septic ones (pyogenic arthritis, acute bacterial endocarditis), within a few hours, vital signs may fail, shock and disseminated intravascular coagulation (DIC) may develop. Development is so rapid that it may resemble stroke, aggression and disorientation may occur. In meningococcal and haemophilic meningitis, petechiae and suffusion are found on the skin. Newly formed petechiae, larger than 2 mm, confluent and located on the thighs and abdomen, are typical of meningococcal disease. Differentiation from ordinary urticaria is possible by pressure (exanthema at pressure below the petechiae persist ('slide method')) [1] (https://www.khszlin.cz/wcd/pages/extranet/organizacni-struktura/odbor-protiepidemicky/legislativa/vest_10_2006.pdf).

Complications

The complications of purulent meningitis are numerous. **Acute stage** may be accompanied by *brain edema* which may cause visual or hearing impairment or central palsy. In the **recovery phase**, *parainfectious arthritis, myocarditis, headache and fatigue* occur. Children may then have *psychomotor retardation or hydrocephalus*.

Prognosis

Symptoms usually subside on the 3rd-5th day of treatment and the patient gradually improves. Early diagnosis, adequate therapy and resistance of the organism are crucial. In practice, the disease has a dual course:

1. **mild:** insignificant symptoms, rapid correction of findings in the liquor
2. **difficult:** patient in coma from the beginning, death can occur within 24 hours under the picture of shock, a frequent complication of adrenal haemorrhage (Waterhouse-Fridrichsen) or DIC

Differential diagnosis

- if history is absent and consciousness disorder progresses → rule out stroke, subarachnoid haemorrhage, metabolic comatose states (DM), poisoning, etc.
- other meningitis (serous, tuberculous, mycotic), brain abscess, epidural empyema/abscesses (intracranial or spinal), subdural empyema, infective endocarditis with CNS embolizations, thrombophlebitis of the sinus, rupture of dermoid cyst, brain tumours
- the diagnosis is determined by examination of the cerebrospinal fluid

Treatment

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

- early treatment with ATB (RIGHT AWAY!, control CSF in 1-2 days)
- a doctor can save a life by administering penicillin before transport to hospital
- Optimal today are **Generation III cephalosporins** (e.g. cefotaxime, ceftriaxone)
- the selected ATB must cross the blood-brain barrier well (not tetracycline, partially aminoglycosides), dosing in children is guided by weight and age
- chloramphenicol may rarely lead to aplastic anaemia, also unsuitable for children under 5 years of age
- the source of meningitis may be another inflammatory deposit in the body = **secondary meningitis**: early treatment and search for a possible cause
- correction of the internal environment with respect to ADH secretion
- we correct possible brain edema, administer vitamins and other symptomatic therapy
- We discontinue ATB no earlier than 10-14 days after temperature normalization
- meningococcal meningitis is contagious (vaccination, prophylaxis in family members)

Types of purulent meningitis

Pneumococcal purulent meningitis

Pneumococcal purulent meningitis occurs at any age, usually of secondary etiology. **Pathogenic** are **encapsulated strains**. It has the most difficult course in debilitated people (alcoholics, cirrhotic patients) and in splenectomized people (fulminant course). *Primarily* it can be *airborne* (especially in winter), but is most commonly **secondary**. Infectious agents **multiply in the respiratory tract**, from there they spread *through the blood* to the brain. It can also enter the brain by transfer from the *sinuses or ear*. The occurrence of **abscess** is not rare. The CSF shows a typical picture for purulent meningitis, **microbiology is required**.

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*, transmitted **airborne**. It often arises **after exhaustion** (sports, disco, a sleepless night, etc.). Manifestations:

- sometimes initially 1-2 days symptoms of pharyngitis, fatigue and abdominal pain
- followed by fever, vomiting, disturbances of consciousness
- petechiae and suffusion appear on the skin
- development of DIC and shock - **multiorgan impairment**

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

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

Shunt meningitis

Shunt meningitis occurs in children with hydrocephalus who have a drainage shunt (infection can occur).

Other neurological damage

One of the possible damages may be paralysis of cranial nerves or motor disorders (e.g. hemiparesis or quadriparesis, ataxia). Cerebral ischaemia, diencephalohypophyseal syndrome or SIADH (syndrome of inappropriate secretion of antidiuretic hormone) manifested by oliguria and CSWS manifested by polyuria may also occur.

Links

Related articles

- Meningitis • Meningitis (pediatrics)
- Viral meningitis • Serous meningitis and meningoencephalitis • Herpetic meningoencephalitis
- Purulent meningitis • Purulent meningitis (pediatrics) • Haemophilic meningitis • Tuberculous meningitis
- Infectious brain disease • Neuroinfection, CNS / PGS inflammation • Encephalitis
- Meningococcal meningitis

Source

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Used literature

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