Serous meningitis and meningoencephalitis

It is an inflammation of the brain tissue and adjacent structures - meningeal membranes, for which the term meningoencephalitis is collectively used. Its etiology is most often viral.

Classification

- acute encephalitis
  - enteroviruses
  - arboviruses
  - Herpesviridae
  - rabies
- post-infection encephalitis (on an immunological basis)
  - parotitis epidemica
  - varicella
  - morbilli
  - rubella
- subacute sclerosing encephalitis

Acute serous meningitis can also be caused by bacterial agents, most often Borrelia (in our conditions most often *B. garinii* and *B. afzelii*) and leptospira.

Signs and symptoms

- the meningeal syndrome is less pronounced; on the contrary, convulsions and topical neurological manifestations are observed more often
- respiratory disorders and bradycardia occur when brainstem structures are affected
- hypothalamic and pituitary involvement is associated with hypothermia and SIADH / diabetes insipidus
- the cranial nerves and spinal cord may also be affected

Diagnosis

- Lumbar puncture -> cerebrospinal fluid examination:
  - biochemical, cytological, microbiological, serological
  - PCR - the DNA of HSV-1, HSV-2, and the DNA of adenoviruses is detected by the so-called multiplex PCR, and the RNA of enteroviruses by RT-PCR

Cerebrospinal fluid characteristics in encephalitis / viral meningoencephalitis:

- in the order of 100-1000 cells with a predominance of lymphocytes
- proteinorrhachia usually up to 1 g/l
- glycorrhachia within normal limits
- auxiliary examinations:

CT, EEG, EMG, MRI, evoked potentials - for example, in herpetic meningoencephalitis, pathological EEG activity is demonstrated temporally, as well as evidence of necrosis on the CT on the third to fourth day of disease progression.

Treatment

- in HHV-1 and HHV-2, causal treatment with acyclovir
- anti-edematous
- anticonvulsant

Lyme neuroborreliosis

Pathogenesis

Lyme neuroborreliosis is a tick-borne multi-system infectious disease caused by the flagellate spirochete *Borrelia burgdorferi sensu lato*. In our conditions, the *B. garinii* and *B. afzelii* genotypes are most often used. Their reservoirs are mainly small mammals, and they are transmitted to humans by an infected tick bite. At the point of entry, spirochetes multiply, which manifests itself as erythema chronicum migrans in 50-80% of patients. If Borrelia is not eradicated, it spreads to the body through the blood and lymphatic system and the disease progresses to stage II and can also affect the nervous system.

Clinic

- stage I (days to weeks since infection)
  - exhaustion, fever, lymphadenomegaly, arthralgis – generally non-specific symptoms
  - erythema chronicum migrans
- stage II (weeks to months since infection)
  - borrelia arthritis
  - neuroborreliosis
  - borrelia carditis – impairment of the conduction system
  - benign borrelia lymphocytom
  - other symptomatology: ocular impairment, hepatitis, nephritis aj.
- stage III (months to years since infection)
  - chronic nervous system disorders
  - acrodermatitis atrophicans

Neuroborreliosis clinic

- diffuse permanent
- meningeal irritation
- Bell's palsy of the facial nerve; approximately in 50% of patients it is bilateral
- Banwarth syndrome: radicular pain, paralysis of cranial and limb nerves (this is the most serious form, fortunately rare in children)

Diagnosis

In addition to the clinical findings, the most beneficial diagnosis for Lyme neuroborreliosis is the CSF analysis, where signs of aseptic meningitis can be found, and a serological examination focused on the detection of antibodies against Borrelia will be helpful in a closer determination. Roughly indicative serology is performed by the ELISA method, but because of its high rate of false positives it is necessary to confirm the positive finding by Western blotting, which determines antibodies against individual Borrelia antigens and shows higher specificity. It is also necessary to remember that the amount of antibodies in the CSF depends on the permeability of the blood-brain barrier (BBB) and on the amount of antibodies in the serum - quantification of intrathecal production is possible on the basis of determining the ratio of antibodies and albumin in cerebrospinal fluid and serum, referred to as the **antibody index**. If this index is not determined, the findings of specific Ig in the cerebrospinal fluid must be evaluated with caution, as their presence can only be due to a larger transfer from serum with increased BBB permeability.

Treatment

- stay in the ICU depends on the intensity of clinical symptoms
- antibiotics: third generation cephalosporins (ceftriaxone, cefotaxime) for 14–21 days i.v.
- antipyretics, analgesics
- at the beginning, ev. treatment of cerebral edema

Links

Bibliography

- HAVRÁNEK, Jiří: *Serózní meningitidy a meningoencefalitidy*.