Infectious diseases in intensive care

Bacterial meningitis

Clinical picture

The three most important signs of bacterial meningitis

1. altered level of consciousness;
2. fever;
3. meningeal signs.

A characteristic sign is the development of a disorder of consciousness (within 24 hours). 90% of patients will develop somnolence, sopor, or coma as a result of impaired consciousness. The condition is accompanied by severe sepsis and progresses fast to organ failure (distal organs are affected, i.e., distant from the site of infection).

Treatment in case of suspected bacterial meningitis

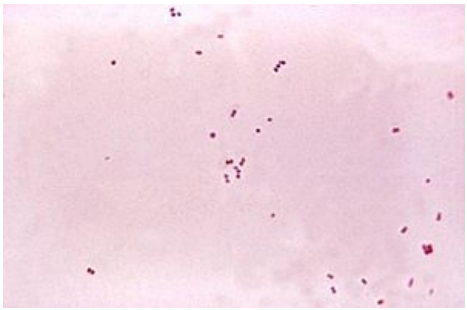
1. secure the airways;
2. IV access;
3. mechanical lung ventilation;
4. anti-edematous treatment (diuretics, mannitol);
5. immediate transport to the hospital (ICU);
6. antibiotics are administered only in the hospital, after blood cultures collection. Delayed ATB treatment does not worsen the prognosis of bacterial meningitis. An exception is in case of the meningococcal meningitis (invasive meningococcal disease), when third generation of cephalosporins is also administered during transport to the hospital (3 g of cefotaxime).

Diagnostics

In critically ill patients, lumbar puncture is performed while lying on one's side. A minimum amount of cerebrospinal fluid is taken. Intracranial hypertension should be ruled out before removing fluid to minimize the risk of the occipital conus. Ophthalmological examination is used for this purpose, where edema of the papilla of the optic nerve (optic disk) would indicate intracranial hypertension. Papillary edema indicates CNS involvement with a 24-hour delay. A CT scan of the brain is more accurate, but it often leads toa a long delay. Relative contraindications for lumbar puncture are focal brain process, papillary edema (or CT findings), and immunodeficient patients over 60 years of age (CT examination is preferred).

Finding in cerebrospinal fluid in bacterial meningitis

- morphology: elevated leukocytes levels (leu > 1000/mm^3), of which polymorphonuclear cells make up > 60 % of cells;
- biochemistry: increased lactate (> 3,5 mmol/l), decreased glycemia, glucose coefficient (cerebrospinal fluid / blood) < 45 % (standard: 50–60 %), increased proteins (> 1,3 g/l).



Finding of gram-negative Neisseria meningitidis in cerebrospinal fluid in Gram staining

Gram staining has a sensitivity of 80% in cerebrospinal fluid, while a standard cultivation has a sensitivity of 90% (cerebrospinal fluid, unlike blood, is a more favorable environment for bacteria because it does not contain immunoglobulins, which protect the CNS from autoimmune processes). PCR examination of cerebrospinal fluid is a backup examination, and is expensive. It is focused on the detection of bacterial (possibly viral) DNA.

Loss of consciousness in bacterial meningitis

Pathophysiology

Loss of consciousness is caused by edema, which is cytotoxic (pneumococcus). The blood-brain barrier through which solutes enter the CNS is compromised. This increases intracranial pressure, worsens perfusion, and worsens hypoxia.

Treatment of cytotoxic edema

- antiedematous drugs: mannitol, diuretics;
- adjuvant therapy: controlled hypocapnia (causes vascular spasms), corticosteroids (stabilize membranes).

Sepsis

Sepsis is defined as SIRS (systemic inflammatory response syndrome) associated with an infection. Bacteria may not be present in the blood (although it is in most of cases). Fungi, viruses, parasites, endotoxins, or inflammatory agents can also cause sepsis. Sepsis is the leading cause of death in the ICU. This is related to higher morbidity of patients (old age, invasive treatment or diagnostic procedures, immunosuppression,...). The most common etiological agents are bacteria. In regards to viral agents, viruses in Africa responsible for hemorrhagic fevers, influenza viruses in Europe, and cytomegalovirus in immunosuppressed individuals may be responsible.

Current definition of sepsis from 2015: **Sepsis** is a life-threatening organ dysfunction caused by dysregulated response to infection.

Criteria for the diagnosis of sepsis

1. temperature > 38 °C or < 36 °C;
2. heart rate > 90/min;
3. respiratory rate > 20/min;
4. p_aCO_2 < 32 kPa;
5. leukocytes > 12 × 10^9/l or > 10 % rods.

Clinical severity of sepsis

1. **sepsis**;
2. **severe sepsis** (MOFS = multiorgan failure);
3. **septic shock** – the worst prognosis, circulatory failure occurs. Unlike other types (hypovolemic, cardiogenic, anaphylactic types of shock), during this phase of septic shock, the patient is pink, well-perfused, has warm limbs and has increased cardiac output. The arteriovenous shunts (A-V Shunts) are open, the blood goes to the internal organs, and deep organ hypoxia develops, which is caused by increased lactate.

Sepsis of nosocomial origin

Several factors contribute to nosocomial sepsis in ICU patients, such as intubation, cannulas, urinary catheters, analgesia, which cause muscle relaxation and the accumulation of secretions, bedsores, and poor general state with the production of cortisol (stress hormone) and IL-10.

Most common types of nosocomial sepsis

1. **ventilator-associated pneumonia** – intubation cannulas can be primarily infected, secondarily infected by the hands of hospital staff, or endogenously infected by the penetration of gram-negative bacteria from the intestines. The cranial progression of bacteria is facilitated by reduced intestinal motility, increased pH in the stomach (administered by proton-pump inhibitors to prevent stress gastric ulcers) and gastroesophageal reflux. The limit for pneumonia to be considered nosocomial is 48 hours after admission. Earlier development indicates that it is community-acquired;
2. **catheter sepsis**;
3. **urosepsis**;
4. **bedsores**.

Causative agents of nosocomial sepsis

The causative agents include almost exclusively gram-negative bacteria.

- Pseudomonas aeruginosa (treatment: carbapenems, in case of colistin resistance);
- Klebsiella;
- Proteus mirabilis.

Polyradiculoneuritis

Polyradiculoneuritis is a post-infectious inflammatory process that affects peripheral nerves. There is a rapid development of sensory disturbances and motor impairment of the lower limbs. These disorders are symmetrical and motor disorders are also referred to as "glove-and-stocking" paresthesias.

Characteristics of polyradiculoneuritis

- peripheral nerve impairment;
- progression to ventilation failure;
- typical association with certain pathogens: Campylobacter jejuni, Borrelia burgdorferi, Cytomegalovirus, HIV, Influenza Virus.

Diagnostics

- **lumbar puncture**: discrepancy between cerebrospinal fluid cytology and proteinorachia: proteins are elevated (> 1 g/l) and pleocytosis is minimal.
- **MRI**: without any signs of spinal compression;
- **serology**: borreliosis, CMV, HIV.

Treatment and prognosis

The basics include monitoring the condition in order to detect possible ventilation failure, secure the airways, and initiate mechanical lung ventilation. This is often accompanied by swallowing and micturition disorders, which should be noted. Hospitalization is necessary. In addition, high doses of immunoglobulins are administered or plasmapheresis is performed. Immunoglobulins appear to be a safer alternative. They are preferred in children. On the other hand, plasmapheresis is just as effective, but cheaper; however, it is often associated with a higher incidence of complications.

Complications

Serious complications of polyradiculoneuritis include:

- progression to cranial nerve palsies;
- respiratory failure;
- persistence of residual paresis.

References

- HOLUB, Michal. *Infekční nemoci v intenzivní péči* [lecture for subject Infekční lékařství, specialization všeobecné lékařství, 1.LF UK]. Praha. 2011-04-27.