Clostridium tetani

Clostridium tetani is an anaerobic, slender, non-invasive, Gram-positive rod with flagella. It produces neurotoxin **tetanospasmin** inducing tetanus. It spreads to the CNS mainly through the neuromuscular connection. The toxin **blocks** the release of inhibitory **neurotransmitters**, clinically manifesting itself as a spasm of striated muscle. It forms very resistant **spores**, which can survive for a very long time, while vegetative forms perish rapidly under the influence of oxygen. Spores survive boiling and are resistant to most disinfectants.

History

In 1884, A. Nicolaire isolated a strychnine-like toxin from anaerobic soil bacteria. In the same year, A. Carle and G. Rattone clarified the etiology of tetanus by provoking tetanus in a rabbit that was inoculated with an injection of pus from human tetanus. Kitasato Shibasaburo isolated *C.tetani* in humans in 1889. He later demonstrated the formation of tetanus in animals after injecting bacteria, but also neutralizing the toxin with specific antibodies. In 1897, E. Nocard demonstrated the ability to induce passive immunity in humans with tetanus antitoxin. P. Descombey developed in 1924 a vaccine against tetanus based on toxoid.

Occurrence

Found in the **intestines** of mammals (often in horses), it may be in the intestine of humans. It is usually found in soil that is fertilized with a horse or cow manure.

Morphology

C. tetani is a slender Gram-positive rod 0.5x5–7µm in size. It contains a **terminal spore**, the shape resembles a mallet.

Cultivation

It grows well on **blood agar**. It shows **hemolysis**. Requires an **anaerobic** environment.

Antigens and toxicity

Tetanus toxin is made up of three components: **tetanospasmin,** *tetanolysine'* with hemolytic properties, and **renin enzyme**. The most important is tetanospasmin, a homogeneous polypeptide of one antigenic type. Depending on flagella antigens and tetanospasmin production ability, 11 types of *C. tetani* have been identified.

Tetanus

C. tetani causes a disease called tetanus traumaticus. Infection occurs by **injury** and contamination from the soil, the incubation period is about one week. This disease does not leave immunity, since the toxin does not get to immunocompetent cells due to rapid uptake by nerve tissue.

Pathogenesis

The toxin absorbed from the injury into the blood and lymph gets **to the neuromuscular discs** and through them to the nervous system. After penetration into the cytosol, the neuron blocks the release of inhibitory mediators (gamma-aminobutyric acid, glycine). It reduces the threshold of irritability of motor neurons and tonic-clonic **convulsions** occur.

Manifestation

Initially, the contraction of the chewing muscles, **trismus**. Spasms of mimic muscles, which are supplemented by salivation (risus sardonicus). Stiffens the neck and back to form a typical arcuate deflection of the body opisthotonus. When trying to drink, laryngospasm occurs. Gradually, the respiratory muscles are also affected, the patient dies of asphyxia or heart failure.

Laboratory diagnostics

The disease is recognizable by the clinical picture. The excision from the injury under examination would probably give a negative result of a microscopic examination, due to the low number of clostridia in the wound. It is advisable to supplement the culture with evidence of toxin on a mouse, which is injected to the root of the tail, and in the case of positive evidence, its sigmoid solidification will occur.

Epidemiology

Tetanus is a characteristic sapronosis (a disease provoked by putrefactive bacteria), the source of infection is the external environment. It is not transferable to another person. Thanks to the vaccination system, the disease is rare in our country, it occurs mainly in elderly immunocompromised people.

Prevention

The most significant is the regular **toxoid vaccination**. It provides a protective effect for more than 10 years, so after every ten years, a reminder dose (booster) is recommended. It is part of a six-valent mixed vaccine (hexavaccine) together with diphtheria, active cough, invasive hemophilic infection, viral hepatitis B and children's cerebral palsy.

Treatment

Treatment consists of administering **antitetanical immunoglobulin** (it has little chance of success due to the fact that the toxin is already present in the CNS, where immunoglobulin does not reach). Previously, equine IgG was used, which was supposed to show higher efficiency, but due to side effects, it was replaced by human IgG. Non-specific treatment means proper surgical treatment of the wound, support of breathing and reduction of irritability by medications.

Treatment with penicillin G or metronidazole is also often initiated. Antibiotic therapy is of lower importance, however, clostridium is sensitive to it. However, the administration of penicillin may lead to amplification of tetanospasmin effects.

Links

Related Articles

- Tetanus
- Gram staining
- Active immunization
- Bacterial toxins
- Repetitorium microbiology

External links

- www.tetanus.cz (http://tetanus.cz/)

Literature used

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