



## WOUND INFECTIONS. DESCRIPTION AND LABORATORY DIAGNOSIS

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### ABSTRACT

*Microbiology, virology and immunology laboratories are of great importance not only for diagnosing the patient, but also for preventing the disease.*

*Students distinguish anaerobic bacteria that cause wound infections by their infectivity, pathological conditions, and toxic properties. Every doctor should know the epidemiology, pathogenesis, clinical symptoms, diagnosis and prevention of these diseases. If the general and special measures taken in the event of these diseases are not carried out in time, if the doctors cannot choose the right material to take from the patient for examination, this disease can spread among the population and cause severe complications.*

The causative agent of convulsions, *S. tetani*, was discovered by N. D. Monastirsky (1883) and A. Nikolai (1884). In 1889, S. Kitazato isolated its pure culture.

**Morphology.** (*Cl. tetani*) is gram-positive, rod-shaped, mobile, 4-8  $\mu\text{m}$  long, 0.3-0.8  $\mu\text{m}$  wide. Inclusions are located in the middle and edges of the cell, the spore is mainly located on the edge, giving the cell the shape of a drumstick. The causative agent is a peritrix and does not form a capsule.

**Cultivation.** *Cl. tetani* strictly anaerobic on sugar or blood agar with pH 7.0-7.9 at a temperature of 37C forms a thin membrane, some of which have a dense center and an irregular shape around the colony. In the Kitt-Taroststi environment, this clostridium grows, producing a thick and peculiar smelling gas.

**Durability** The causative agent of Kaqshol dies when it is heated in a vegetative state at a temperature of 60-70°C for 30 minutes. Clostridium spores are very resistant to the external environment. Spores are stored in the soil for up to 1 year, and in various objects.



**Pathogenesis of the disease in humans.** The source of the disease is animals and people. Bacteria of the disease can fall into the soil through their excrement and remain in the form of spores for many years. Spores of the disease enter the damaged skin or mucous membrane through the soil. The causative agent of the disease enters babies through the umbilical wound, and women who have given birth through the inflamed mucous membrane of the uterus. The spore changes to a vegetative form in the body, multiplies and begins to release exotoxin. Exotoxin enters the blood and affects the motor nerve center of the spinal cord through the blood. As a result of damage to the nervous system by the tetanus bacteria toxin, various muscle groups in the body contract.

The latent period of the disease is 4-14 days. The muscles in the place where the stimulus entered, and then the muscles of the face, chewing and facial expressions contract. Then the neck, back and leg muscles tighten. The patient's head goes back, the body is bent, arched behind the head and supported by the waist, head and heels. The patient dies as a result of failure of the respiratory center and other vital organs. Mortality in this disease is 35-70%.

**Immunity.** After the disease, weak antitoxic immunity is formed, so it is possible to get sick again.

**Laboratory diagnosis.** Because the clinical symptoms of the disease are clear, the material, which is less often tested in the laboratory, is divided into two parts: one part is planted in a special nutrient medium to isolate a pure culture; the second part is injected into the back leg muscle of a white mouse for biological testing. If a tetanus pathogen is present, an infected white mouse will develop the disease and die within 1-3 days. White mice do not develop the disease when the toxin is administered together with tetanus immune serum.

**Treatment and prevention.** After surgical treatment of the wound, tetanus anatoxin, antitoxic serum is administered, which creates active immunity in the body. If a special immunoglobulin is given against tetanus, it will give a good result. The patient is treated in separate, quiet, noiseless rooms.

Tetanus anatoxin is used for special prevention. According to the plan, AKDS and ADS-M vaccines are given to children and adults at 2, 3, 4, 16 months and 7, 16-17, 26, 46 years.

### **Gas gangrene**

Wound anaerobic infections include *Cl. perfringens*, *Cl. novyi*, *Cl. septicum*, *Cl. histolyticum*, *Cl. difficile*, *Cl. sordellii*, *Cl. bifermentans*, *Cl. fallax*. In the pathogenesis of the disease, along with pathogenic microorganisms, non-pathogenic clostridia *Cl. aerofaetum*, *Cl. tertium*, *Cl. sporogenes* are also involved.

Each of *C. perfringens*, *C. novyi*, *C. septicum*, *C. histolyticum* can cause the disease separately, but it is often caused by other species together. The disease is mainly caused by *Cl. perfringens*

The pathogen (*Clostridium perfringens*) was discovered by M. Welch and G. Netall (1892). It lives as a normal microflora in human and animal intestines. In the external environment, it lives in the form of spores in the soil.

**Morphology.** *C. perfringens* is a large gram-positive rod, 8  $\mu\text{m}$  long, 1-1.3  $\mu\text{m}$  wide, it forms spores in the external environment, it is located subterminally in the cell, the diameter is larger than the width of the bacterium, it does not move



**Growth.** *C. perfringens* is strictly anaerobic, grows well at 37°C in nutrient media where all anaerobic bacteria with Pn 7.2–7.4 grow. In Kitta-Tarotsi medium, it forms thick, lentil-like colonies at the base of the agar column, and in Wilson-Blair medium, it forms black colonies after 1-3 hours. On blood agar it forms flat, gray, smooth disc-like S-colonies. Can form both R- and M-colonies.

**Pathogenicity to animals.** *C. perfringens* is pathogenic to all domestic animals and causes various serious diseases. Among experimental animals, guinea pigs, rabbits, pigeons and white mice are very susceptible. Other pathogens also differ from each other in their cultural, biochemical and serological characteristics.

**Pathogenesis of the disease in humans.** In a person, the spore falling into the wound from soil, clothing and other infected objects later changes to a vegetative form and begins to multiply, which in turn releases an exotoxin that rots and decomposes the tissues and produces a large amount of gas.

Necrotic or inflamed tissue is necessary for the reproduction of the pathogen. Aerobic microflora creates the necessary anaerobic condition at the site of damaged necrosis. This process develops especially quickly in muscle tissue, as a result of which glycogen is formed in large quantities, which, in turn, is a favorable environment for pathogenic anaerobes.

As a result of the development of anaerobic infection, swelling occurs in the first stage, and connective tissue and muscles undergo gangrene in the second stage. The body is strongly poisoned or intoxication occurs. Substances formed due to decay of tissues also have a toxic effect on the body.

Gaseous anaerobic infection is a disease of wounds, in which the wound tissues are often crushed, the infected wound becomes contaminated and anaerobic conditions are created, which leads to the development of gas gangrene. If pus-forming staphylococcus, streptococcus or putrefactive anaerobes appear on the surface of the wound, the disease is often severe.

The latent period of the disease sometimes lasts 4-6 hours, often 1-2-5 days. Clinical signs of gas gangrene are different. Swelling and reddening occur in the injured area, gas accumulates between the tissues. Muscles and other tissues undergo necrosis.

Since exotoxins have the property of constricting blood vessels, the skin of the gas accumulation area becomes fluid and shiny, gradually turns bronze, and the temperature of the injured tissues is lower than the temperature of healthy tissues.

*C. perfringens* causes food poisoning in addition to gaseous anaerobic infection. In the development of anaerobic wound infections, the condition of the wound, the immune status of the macroorganism, the number and type of the microbe, etc., play an important role.

**Immunity.** Strong, stable, long-lasting immunity is not formed after the disease. But antitoxic antibodies in blood serum can protect the body from infection to a certain extent.

**Laboratory diagnosis.** Microscopic, bacteriological, biological and neutralization methods are used to make a laboratory diagnosis of the patient. Crushed necrotic tissue, tumor fluid, bandage material, surgical suture, catgut, clothing, soil and other materials are taken for examination. The inspection is carried out in several stages.

After making a smear from the wound fluid, staining it and looking under a microscope, the causative agent and its capsule are found. The material to be tested is planted in a special nutrient medium, a pure culture is isolated and identified according to its morphology,



capsule formation, movement and enzymatic properties. To detect the toxin, a white mouse is injected with the filtrate of a broth culture or the patient's blood. The toxin is added to white mice with an antitoxin, a method of neutralization.

**Treatment and prevention.** In order to prevent the development of gas anaerobic infection, the patient will first be provided with full surgical assistance in time, and then, for the purpose of prevention, the patient will be given polyvalent antitoxin serum "Diaferm 3" against *C.perfringens*, *C.novyi*, *C.septicum*.

Streptomycin, penicillin, staphylosporin, anti-staphylococcal plasma, gamma-globulin are prescribed as biological drugs, and surgical method is also used in some cases. Special preventive methods against anaerobic infections have not yet been developed. After providing surgical assistance to the patient, a small amount of polyvalent antitoxic serum is administered for preventive purposes.

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