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LEPROSY AND ACTINOMYCOSIS DESCRIPTION AND DIAGNOSIS OF THE DISEASE CAUSED BY THEM

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Abstract: In this article, air-droplet infections are studied on the example of leprosy and actinomycosis causative agents, their description and detailed information about the diseases they cause, diagnosis of these diseases, and treatment measures are provided.

Key words: Leprosy (leprosy), Steele-Nielsen, diffecoloxidase, lepromatosis, actinomycosis, Saburo medium,

The causative agent of leprosy

Leprosy is a chronic infectious disease that affects the whole body, especially the skin, nervous system and internal organs. The causative agent of the disease is acid-resistant leprosy mycobacterium (Mycobacterium leprae Hansen).

Morphology. Leprosy mycobacteria are straight or slightly curved rods, 1–8 μm long, 0.2–0.5 μm wide, and one end may be thicker than the other. They penetrate into the cell, form tight spherical nodules and are located in close contact with each other. In damaged tissue, leprosy bacilli are spherical, filamentous, nodular and other forms are found. Mycobacteria contain 9.7–18.7% lipids and up to 2.25% phosphatides, so they are stained red by the special Steel-Nielsen method. In addition, they are resistant to acid because they contain a lot of oil pigments, various waxes and leprosin mycolic acids. Spores and capsules do not form, inactive.

Growth. Leprosy bacilli do not grow in the same nutrient medium as the causative agent of tuberculosis. The test material was grown by injecting it under the feet of white mice. Storrs (1974) was able to develop a method for growing mycobacterium leprosy in the nine-banded armadillo (Dasypus novemcinctus). After 15 months, mycobacteria multiply in the armedilla organism in various forms, mainly in the cell cytoplasm.

Methods have now been developed to infect bronenos (in Texas and Louisiana) and mangaboy monkeys. Fermentative property. It was found that there are diffecoloxidase, peroxidase, stetochromoxidase, dihydrogenase, and other enzymes involved in the reproduction of leprosy mycobacteria.

Pathogenesis of the disease in humans.

3 clinical types of leprosy are distinguished:

1. Lepromatous type is very severe, epidemiologically dangerous. A lot of lepromas appear on the patient's face, wrist, calf and other parts, they can merge with each other and form large infiltrates.

Later, lepromas are punctured and non-healing wounds appear in their place. Sensibility is lost in the foci of the disease, hair and feathers in this area fall out, especially eyebrows and eyelashes fall out. In this type of leprosy, the mucous membranes of the nose, mouth, and eyes are also damaged.

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Fingers and toes are mutilated and fall off, infiltrating corneal lesions sometimes make patients completely blind.

2. The tuberculoid type of leprosy (in the form of a skin tubercle) is relatively harmless and passes easily. In patients suffering from this type of leprosy, the lepromin allergic reaction is positive, due to the lack of elements of the rash, it is more difficult to find mycobacterium leprosy.

3. In undifferentiated, i.e., unclear type of leprosy, the resistance of the macroorganism is different, often stronger. When the material from the damaged area is examined bacterioscopically, mycobacteria are not always found. They have a negative or weakly positive allergy test.

The disease is chronic. Children aged 8-14 are very susceptible to leprosy, and they are infected mainly from their sick parents. The disease occurs 3 times more often among men than among women.

Immunity. Immunity in leprosy is poorly understood, its mechanism is similar to tuberculosis immunity and is cellular. Genetic factors also play a role in the disease of patients with leprosy; for example, people with haplotype HLA-DR2-DQW1 are more likely to have lepromatous leprosy, and people with HLA-DR2 or HLA-DR3 are more likely to have tuberculoid leprosy.

Laboratory diagnosis. If mycobacterium leprosy is not found in the laboratory examination, the doctor makes a diagnosis based on the clinical symptoms of the disease. However, the diagnosis made through laboratory examination is more accurate and reliable.

In the lepromatous type of leprosy, more mycobacteria are found than in other types of the disease. The preparation is prepared from smears taken from the upper respiratory tract, for example, the nasal mucosa. For this, the nasal cavity is thoroughly cleaned, and the patient can do it himself. Then, swabs are taken from the inner wall of the nose with sticks wrapped in gauze tampons prepared in advance and applied to the glass of several items with the same thickness. Examination of smears prepared from the fluid of the affected skin tissue gives good results for the detection of leprosy mycobacteria. First, the skin of this area is cleaned with alcohol or ether, thoroughly wiped, firstly, aseptic technique is followed, and secondly, some acid-resistant saprophytic microorganisms are cleaned from mycobacteria. Then, while pinching the intended skin level with the fingers, a sterile sharp surgical knife (scalpel) is cut 5 mm long and 2-3 mm deep. By scraping the separated liquid with a scalpel, several smears are made on the glass of the object. Tissue fluid is obtained from lepromas in the area of the eyebrow, forehead, auricle, back and buttocks. Smears are painted by the Steele-Nielsen method. But mycobacteria of leprosy are acid-resistant compared to mycobacteria of tuberculosis, and care should be taken when decolorizing the drug.

In stained smears, leprosy mycobacteria are red or pink in color, in groups, and sometimes singly, they are slightly elongated and parallel to each other. 1 ml of liquid to be tested for leprosy bacilli should contain at least 10,000-100,000 mycobacteria. This requires examining 60–100 fields of view in one swipe. Finding 1-2 mycobacteria does not confirm the diagnosis. The number of mycobacteria in the field of view is determined according to Hort's scheme as follows: 0 - no mycobacteria; + suspicious, there are 1-2 mycobacteria in the visual field; ++ there is a lot of mycobacteria in the field of vision; +++ there is a lot of mycobacteria in the field of vision. To distinguish leprosy from tuberculosis, the pathological material is infected with a guinea pig in a 0.85% sodium chloride solution. If the patient has tuberculosis, then the guinea pig quickly develops tuberculosis and dies, or vice versa, guinea pigs do not get leprosy. When 0.1 ml of lepromin is injected between the skin of the patient's wrist, after 48-72 hours this area becomes red and swollen, the Mistuda reaction is considered positive. KBR, BilGA reactions are used to detect antibodies formed in the patient's blood. Treatment and prevention. To treat the patient, dapsone, rifampistin, lampren,

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oflaxastin, minosticline, in addition, cortisone, prednisolone and other corticosteroid drugs are used to reduce sensitivity.

Pathogenic actinomycetes

Morphology. Actinomycetes consist of branching, rod-shaped, cocci-like, thin, filamentous mycelia that are easily divided into parts. Grammusbate, without septa, forming spores. (Fig. 73).

Growth. Actinomycetes are facultative anaerobes, and 35-37°C is a favorable temperature for their growth. After 24 hours, small colonies form on the surface of the solid medium, and after 7-14 days, large polymorphous, smooth or rough, grayish-yellow, soft, uniform white, duchoba-like colonies are formed. Colonies can be inside or outside the nutrient medium. Colonies are purple, brown, red, green and other colors. Actinomycetes form aerial mycelia in solid nutrient environments (Saburo medium). They are divided into cylindrical and round pieces. Spores are formed at the tips of myceliums, which give color to the colonies.

Pathogenicity to animals. Actinomycetes cause chronic diseases in sheep, goats, cattle, pigs, horses, dogs, rabbits and other animals. In this disease, the animal's skin, neck, lungs, tongue, lips, and in some cases, bones and udder are injured.

Pathogenesis of the disease in humans. Actinomycetes are found in a variety of places, including mountainous terrain, valleys, warm seas, water or underwater mud. It is also abundant in soils rich in organic matter.

Actinomycosis affects 3 times more men aged 20 to 30 than women.

Actinomycetes are similar to propionate bacteria, mycobacterium tuberculosis, and corynebacteria, but differ from them in terms of their development.

The source of the disease is sheep, goats and cattle, wild animals, dogs, pigs, horses, rabbits, as well as soil, plants, air, even infected wheat ears, etc. Actinomycosis occurs as a result of endogenous entry of actinomycetes from the gastrointestinal system into various organs. A. israilii is often present in the oral cavity of a healthy person and causes endogenous infection when the body's reactivity decreases. Actinomycosis also occurs exogenously. For example: when a person chews the ear or stalk of cereal plants, the actinomycetes in it cause the disease. When actinomycetes enter the body from the external environment, exogenous infection develops.

The occurrence of the disease is influenced by dental caries, "stones", gum disease, caecum and others. In addition, injuries to the mucous membranes of the skin, surgery, and bone fractures play a major role in the development of actinomycosis.

Actinomycetes, having entered the body, spread through connective tissues under the skin, spaces between muscles, blood and lymph. As a result, inflammation develops, a large, hard swelling similar to phlegmon appears, an infiltrate occurs, this swelling becomes necrotic and softens, and pus begins to leak out or into the body. In the pus, "druze" consisting of a set of actinomycetes are formed.

Immunity. A patient who has experienced the disease does not develop a strong, stable, long-lasting immunity, so a person can get sick again. Agglutinin, prestipitin, and complement-binding antibodies are formed in the blood of a recovered person and animal, but they cannot protect the macroorganism from re-infection. During the course of the disease, an allergic condition occurs in the macroorganism, so the skin-allergic test with actinolizate is positive in 87.5% of cases.

Laboratory diagnosis. 1. In actinomycosis, a smear is prepared from the pus from the wound, it is examined under a microscope in its native state with or without staining, and the presence of drusen is determined. 2. Pus is cultured on sugar broth (pH 6.8), blood, serum, meat-peptone agar, Saburo's medium under aerobic and anaerobic conditions, and a pure culture is isolated and identified according

to cultural, biochemical characteristics and sensitivity to streptomycin, chloramphenicol . 3. KBR is placed with the patient's serum to detect antibodies in it. 4. A skin-allergic test is performed with extracts of actinomycetes.

Treatment and prevention. In the special treatment of this disease, actinolysates, polyvalent actinomycete vaccine prepared from 6-8 strains are used. Antibiotics, sulfanilamide and iodine drugs are given that act on actinomycete and additional microorganisms. In some cases, the patient is treated with surgery and X-ray. Penicillin, tetrastichlin, erythromycin and clindomistin give good results.

To prevent the disease, it is necessary to strictly observe personal hygiene, protect the skin and mucous membranes from various injuries, and protect the throat, oral cavity, and teeth from diseases. There is no special prevention against this disease.

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