Leprosy (infection)

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae* (*Hansen's bacillus*). The disease is not very contagious (less than 1% of the population that comes in contact with it) and has a long incubation period (1-8 years). It is treatable, but there is no vaccine for it. Between 500,000 and 700,000 new cases are reported each year.

History

The Bible already writes about leprosy. Leprosy was known in ancient China, India, Egypt. It is a disease that appeared in antiquity. The skin was wet and the smell of festering wounds spread to the surroundings. At present, the disease still occurs. It is estimated that 10-11 million people currently suffer from leprosy, mainly in Central Africa and South-East Asia, but cases from southern Europe, especially Spain and also Romania, are also known. It has been present in Romania since at least the 1950s, but this was kept secret by the Ceausescu regime.

Basic division

Leprosy has two basic forms - nervous (*English) Lepromatous leprosy (LL*) and cutaneous or nodular (*English Tuberculoid leprosy - TT*).

- The cutaneous form is characterized by a severe but delayed, immune response in the skin and nerves mediated by T-helper 1 (Th1) cells and hypersensitivity to *M. leprae* antigens .
- In the nerve form, T-helper 2 (Th2) cells are active and there is an inability to respond to *M. leprae* antigens , so that the bacterium is abundant in the skin and nerves. Patients treated with this form of antibiotics are often (50% of cases) affected by inflammation of the subcutaneous tissue (or fat cells), in which hard, red, painful deposits with a diameter of 1-5 cm (*erythema nodosum leprosum - ENL*) appear. .

Most patients have symptoms of both forms (borderline forms are described: *borderline tuberculoid* - BT, *borderline-borderline* - BB and *borderline lepromatous* - BL forms). All variants eventually result in damage to the peripheral nerves in the limbs, which causes a loss of sensory and motor nerve function. Research into the disease has been hampered by the fact that leprosy affects virtually only humans (other infectious species include only the armadillo and three species of monkeys - *chimpanzee, smoky mangabey and cynomolgus macaque*), as well as the fact that *M. leprae bacteria can* not - *it in "extreme" conditions*) are cultured in vitro. Armadillos and immunocompromised mice are used for research purposes. Genome*M. leprae* was sequenced at the beginning of the 21st century as one of the first.

Manifestations and consequences

Leprosy usually does not show more pronounced symptoms and is also not highly contagious in most of its phases. The exception is the skin form , which creates ulcers on the skin that tend to be highly infectious . Under adverse circumstances, a destructive form of leprosy can develop and endanger the patient's life. Leprosy mainly attacks Schwann cells and macrophages in the peripheral nervous system. It destroys the human immune systemand causes deformities on the limbs and face. The skin and parts of the limbs remain dull. Lesions (hands, feet, testicles) form on the skin that look like an ulcer. The sebaceous glands stop working and the skin cracks. The integrity of the skin is violated, causing a gateway infection. Due to the fact that the sensation is disturbed, the affected person does not feel the high temperature, for example, burns occurred in the fire in the Middle Ages. Another consequence of leprosy is blindness or visual impairment. The eyelid stops moving and the eye remains open.

Symptoms

Skin form

It is manifested by redness of a part of the skin most often in the face . This area is often symmetrical and appears around the nose, gradually darkening to a gray-black color as its center fades, eventually being replaced by a white spot resembling vitiligo . Leprechauns begin to form in the affected area. This is often accompanied by fever and swelling of the local lymph nodes. Leprechauns are formed not only on the face, but also on the limbs, where there is a loss of sensitivity in the affected areas - therefore the skin is easily injured, ulcerated and necrosis occurs . As these nodules grow, they change the appearance of the face (so-called *facies leonina* or *leontiasis*).

Nervous form

It starts with the same rash , but the peripheral nerves in the trunk and limbs are also affected , where palpable thickening forms. Over time, hyperesthesia develops , which is later replaced by anesthesia , first for heat and pain, and later for touch. Polio and skeletal muscle atrophy appear . In rare cases, leprosy also occurs in internal organs (mainly lungs and spleen). The spleen is often enlarged due to the presence of amyloid .

Diagnostics

Lepromine test

It is not used to diagnose leprosy, but to distinguish whether a patient has an LL or TT form. The application consists in subcutaneous injection of a suspension of whole autoclaved (heat-killed) bacteria. If a granulomatous reaction develops in the skin within 4 weeks, it is a TT form (APC and T-cell response). A negative reaction is associated with the LL form (APCs and T-lymphocytes do not respond). As already mentioned, this test is not used for diagnosis, because even a healthy person can have a positive reaction to the lepromine test.
Bacteria were once used for this test directly from human leprosy, today they are obtained from infected armadillos. Work is currently underway to identify protein antigens that would be used as reagents instead of whole bacteria, but so far no specific or sufficiently sensitive ones have been found.

Skin biopsy, smear (or nerve biopsy)

Skin biopsy and subsequent histological analysis still play an irreplaceable role in the diagnosis of leprosy. Classical hematoxylin-eosin staining and carbolfuchsin staining are used.
PCR Complementary tests include blood counts , liver and renal function tests .

Therapy

Until 1871, when the pathogen was discovered by the Norwegian microbiologist GH Armauer Hansen , it was believed that the disease was either hereditary or considered a punishment by God. The sick (so-called lepers) were considered unclean and were concentrated in leprosariums. Since the description of this bacterium, treatment began. In the first half of the 20th century, chaulmoogran oil (a type of tree) began to be used, which was injected into the skin, but its effectiveness was debatable. In 1941, *Promin* began to be used , which was effective but required significant doses of anesthetics. Another drug, *Dapsone* , came on the market in the 1950s, but soon (1960–1970) bacteria developed resistance to it. Around 1970, a combination of three ATBs was switched (*dapsone, rifampicin and clofazimine [Lamprene]*) and this concept is still used today.

Etiology

As already mentioned, the causative agent of leprosy is *Mycobacterium leprae* , an immobile, microaerophilic, undisputed, Gram positive, acid-resistant rod. The bacterium is contained in lepromas (nodules that form in the affected skin) intracellularly .

The route of the infection is certainly not clear to this day, but the mucous membranes of the nasopharynx and skin are most often mentioned. Water transfer has also been described. The source of the infection are decayed ulcers, nasal mucus or the stool of the patient. Long-term close contact is required for transmission, which is why children are most at risk. The bacterium thrives best at lower temperatures than body temperature - that's why it mainly attacks the skin and peripheral nerves.

In the 1970s, at least two genes were thought to control the immune response to leprosy and were subsequently substantiated by subsequent research. Today, there is ample evidence for the existence of various genes controlling the immune response against *M. leprae* . This reaction is carried out on 2 levels. The first "package" of genes controls the innate immune response mediated by monocytes . If this innate immunity is not effective enough, the infection will "take root".

This is followed by a second set of genes that control specific cellular immunity (antigen presenting cells (APCs) and T-lymphocytes) and possibly delayed hypersensitivity to *M. leprae* antigens .

If leprosy has already stabilized in the body, it depends on the "quality" of the cellular immune response what form of leprosy will develop. When T-cell-mediated immunity is effective, a less severe TT form develops, manifested by white patches on the skin. This form can heal spontaneously. If this immunity is less "powerful", there is usually a more severe LL type of leprosy, in which symmetrical skin lesions, nodules, plaques, thinned dermis and frequent involvement of the nasal mucosa (bleeding) occur.

There are known cases where one of the family members has leprosy, but other close relatives do not become infected because they have a "good ability" to fight the infection.

Links

related articles

- Leprosy (pathology)

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