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Treatment of a Tuberculosis by Preparation Sangviritrins without Resistance Occurrence Mycobacteruim

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Abstract

Immunomodulators a vegetative at treatment of the infections caused Mycobacteria tuberculosis (cases of clinical supervision). Today with a tuberculosis more often the other inhabitants of developing countries are ill: annually there die of this illness about two millions persons. In the Great Britain every year it is registered about 8 thousand cases of disease by a tuberculosis. In 2007 the record quantity has been cured of a tuberculosis the person - 2, 3 million is 87 % of patients. Then for the first time it was possible to surpass the plan in recover (85 %). In 2008 it has been registered 9, 8 million new cases of a tuberculosis, 1, 8 million persons have died, and at 500 thousand victims this illness has developed against a HIV-infection. Steady against medicines sctamms activators have infected last year a half-million the person, but only 6 thousand from them have received treatment under all standards of the World Organisation of public Health services (WHO).nces.

Keywords: Immunomodulators; Inhabitants; Mycobacteria; Tuberculosis

Introduction

Immunomodulators a vegetative at treatment of the infections caused Mycobacteria tuberculosis (cases of clinical supervision).

Today with a tuberculosis more often the other inhabitants of developing countries are ill: annually there die of this illness about two millions persons. In the Great Britain every year it is registered about 8 thousand cases of disease by a tuberculosis. In 2007 the record quantity has been cured of a tuberculosis the person - 2, 3 million is 87 % of patients. Then for the first time it was possible to surpass the plan in recover (85 %). In 2008 it has been registered 9, 8 million new cases of a tuberculosis, 1, 8 million persons have died, and at 500 thousand victims this illness has developed against a HIV-infection. Steady against medicines sctamms activators have infected last year a half-million the person, but only 6 thousand from them have received treatment under all standards of the World Organisation of public Health services (WHO).

Unfortunately, many sick tuberculosis makes not the correct diagnosis or it is put late, correct schemes of treatment of inadequate duration are appointed not, corresponding dynamic supervision is not spent.

The tuberculosis causes conditionally allocated complex in the person; *M. tuberculosis*, including Mycobacterium tuberculosis (a human kind), Mycobacterium bovis (the bull kind), Mycobacterium africanum, Mycobacterium bovis BCG (BtSzH-shtamm), Mycobacterium microti, Mycobacterium canetti. Recently to it are carried Mycobacterium pinnipedii, Mycobacterium caprae, filogenetic concerning to Mycobacterium microti and Mycobacterium bovis. Mycobacterium concern to procariots (in their cytoplasm is not present highorganization organells device Goldzhi, lizosoms). Are absent also characteristic for a part procariots plazmids, providing for microorganisms dynamics genom's. *Mycobacterium tuberculosis* - aerob, Mycobacterium bovis and Mycobacterium africanum - aerofils. Activators of a tuberculosis do not allocate any exzotoxin which could stimulate fagocitoz, therefore there is no necessity to do inoculation as it causes functional insufficiency fagocitoz. Possibilities fagocitoz Mycobacterium at this stage are limited, therefore presence at fabrics of a small amount of the activator is shown not at once. Mycobacterium are out of cages and breed slowly and fabrics some time keep normal structure. This condition is called «latent microbizm».

Nevertheless, in a place of a congestion of the big number Mycobacterium begins fagocitoz. At first activators begin fagocitoz and to destroy polynuclear leukocytes, however unsuccessfully - all of them perish because of weak bactericidal potential, having come into contact with Mycobacterium. Irrespective of initial localisation Mycobacterium with a lymph current get in regionar lymph nodes then lymfogen extend on an organism - occurs primary (obligat) mycobacteriemia. Mycobacterium are late in bodies with the most developed microcircular a channel (lungs, lymph nodes, crust a layer of kidneys, epifizs and metafizs tubular bones, ampullar-fimbrional departments uterus pipes, uveal an eye path, causing uveits). As the activator continues to breed, and immunity was not generated yet, activator population considerably increases.

The basic specific sign Mycobacterium a tuberculosis - pathogenicity which is shown in virulent. Virulent can essentially change depending on factors of an environment and differently be shown depending on a condition of a macroorganism which is exposed to bacterial aggression. The tuberculosis at people arises at infection human and bull activator kinds more often. Allocation M. bovis it is marked mainly at inhabitants of a countryside, where a transfer way basically alimentary. Peasants can be imparted inoculations, as schtamm identical vacinal - at will, certainly.

The bird's tuberculosis which meets mainly at Immunodeficits carriers is marked also. The breath system is protected from penetration Mycobacterium mucociliar by a road clearance (allocation glass-forms cages of respiratory ways of slime which sticks together arrived Mycobacterium, and further elimination Mycobacterium by means of wavy fluctuations vibrating epitelii). Infringement mucociliar a road clearance at a sharp and chronic inflammation of the top respiratory ways, a trachea and large bronchial tubes, and also under the influence

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of toxic substances does possible penetration Mycobacterium in bronchiols and alveoluses then the probability inficiroval and diseases by a tuberculosis considerably increases.

Possibility of infection with the alimentary is by caused by a condition of a wall of intestines and its soaking up function. Then to fagocitoz are connected macrofags. However Mycobacterium synthesise ATF-POSITIVE protons, sulphates and factors virulent (cords-factors) therefore function lizosoms macrophages is broken. Formation fagolizosoms becomes impossible, therefore lizosomal enzymes of macrophages cannot influence on absorbed Mycobacterium. Micobacterium settle down inside cell, continue to grow, breed and more and more damage a cage-owner. The macrophage gradually perishes, and Micobacterium again get to intercellular space. This process is called «as not finished fagocitoz». The Same effect is caused by inoculation as strengthens infection Micobacterium a tuberculosis.

The got cellular immunity

At the heart of the got cellular immunity effective interaction of macrophages and lymfocits lays. Special value has contact of macrophages with T-helpers (CD4 +) and T-supressors (CD8 +). Macrofags, absorbed Mycobacterium, expression on the surface antigenes Mycobacterium (in a kind peptids) also allocate in intercellular space interleukin-1 (IL-1) which activates T-limfocits (CD4 +). In turn T-helpers (CD4 +) co-operate with macrophages and perceive the information on genetic structure of the activator. Sensibility T-lymphocyte (CD4 + and CD8 +) allocate chemotaxins, gamma interferon and interleukin-2 (IL-2) which activate migration of macrophages towards arrangement Mycobacterium, raise fermentative and the general bactericidal activity of macrophages. Activated macgofags intensively develop active forms of oxygen and hydrogen peroxide. It is so-called oxygen explosion; it influences on fagocitar the tuberculosis activator. At simultaneous influence L-arginin's and the factor necroz tumours the alpha is formed oxid nitrogen (NO) which also possesses antimicrobic effect. As a result of all these processes destructive action Mycobacterium on fagolizosoms weakens, and bacteria collapse lizosomal enzymes. At the adequate immune answer each subsequent generation of macrophages becomes more and more Immunocompetent. Allocated with macrophages mediators activate also B-lymphocyte, responsible for synthesis of antibodies, however their accumulation in blood on stability of an organism to Mycobacterium does not influence. But development B-lymphocyte opsoniroval antibodies which envelop Mycobacterium and promote their pasting, is useful for further fagocitoz.

Increase fermentative activity of macrophages and allocation by them various mediators can conduct to occurrence of cages of a hypersensibility of the slowed down type to antigenes of bacteria. Macrofags are transformed in epiteliod huge cages Langchans's which participate in restriction of a zone of an inflammation. It is formed exssudative-productive and productive tubercular granuloma which formation testifies to the good immune answer to an infection and about ability of an organism to localise micobacterial aggression. At height granulomatous reactions in гранулеме are T-lymphocyte (prevail), B-lymphocyte, macrofags (carry out fagocitoz, carry out affector and effector functions); macrofags are gradually transformed in epiteliod cages (carry out pinocitoz, synthesise hydrolitic enzymes). In the centre granulem's there can be a small site cazeous necroz which is formed of bodies of the macrophages which were lost at contact with Mycobacterium.

Reaction Hypersensibility of the slowed down type appears in 2-3 weeks after inficiroval, and expressed enough cellular immunity is formed in 8 weeks. After that reproduction mycobacterial diseases is slowed down, their general number decreases, specific inflammatory reaction calms down. But full liquidation of the activator from the inflammation centre does not occur. Remained Mycobacterium are localised inside cell (L-forms) and prevent formation fagolizosoms, therefore are inaccessible for lyzosomal enzymes. Such antitubercular immunity is called as unsterile. Remained in an organism Mycobacteruim support population sensibility T-lymphocyte and provide sufficient level immunological activity. Thus, the person can keep Mycobacteruim in the organism long time and even all life. At immunity easing there is a threat of activization of the remained population Mycobacterium and diseases by a tuberculosis.

The got immunity decreases at AIDS, a diabetes, a stomach ulcer, abusing alcohol and long application of drugs, and also at starvation, stressful situations, pregnancy, treatment by hormones or Immunodepressants. As a whole the risk of development of a tuberculosis at for the first time infected person makes about 8 % in the first 2 years after infection, gradually decreasing in the next years.

Occurrence of clinically expressed tuberculosis

In case of insufficient activation of macrophages fagocitoz it is inefficient, reproduction mikobaktyerii is not supervised by macrophages and consequently occurs in a geometrical progression. Fagocytar cages do not cope with volume of work and in large quantities perish. Thus in intercellular space the considerable quantity mediators and proteolitic enzymes which damage at laying fabrics arrives. Occurs original « swill» fabrics, the special nutrient medium promoting growth and reproduction out of a cage located Mycobacterium is formed.

The big population micobacterium breaks balance of immune protection: quantity T-supressors (CD8 +) grows, immunological activity T-helpers (CD4 +) falls. At first sharply amplifies, and then weakens PCHZT to antigenes Mycobacterium. Inflammatory reaction gets widespread character. Permeability of a vascular wall raises, in a fabric fibers of plasma, leukocytes and monocyts arrive. Are formed tubercular granulems in which prevails cazeos necroz. Amplifies infiltration an external layer polyneclear leukocytes, macrophages and limfoid cages. Separate granulems merge, the total amount of tubercular defeat increases. Primary inficiroval it is transformed to clinically expressed tuberculosis.

Symptoms of the tubercular intoxication at children and teenagers

In clinic of a tubercular intoxication distinguish two periods - early and chronic.

Early tubercular intoxication at children and teenagers: Symptoms of an early tubercular intoxication are shown in infringement of balance of the nervous system, expressed in change of behaviour of the child: irritability, excitability, attention fall, in infringement of a dream and headaches. At children for whom were available in katamnez's kists a brain with blood circulation infringement, at them appear psychological stigms in the form of occurrence pathological burns. By 3 years such children become aggressive, at them develops nailneurosis ($p < 0, 01$).

Quite often during this period mark bad appetite, the pallor of integuments alternating small subfebril temperature, some swelling of external lymph nodes. At thin children it is not difficult to probe increased liver, a spleen. There can be a digestion infringement: indulgences of action of intestines or locks. In 4-6 weeks after primary inficiroval children have positive tuberculin a test (a bend tuberculin

tests). It is quite often found out knotty eritema (erythema nodosum). Its occurrence is preceded by a heat, in some days after which, mainly on forward surfaces of shins, appear dense infiltrat, hot to the touch, very painful, red colour with cyanotic a shade. More often eritema arises at preschool children and younger schoolboys, being allergic, paraspecific reaction, instead of tubercular defeat of a skin.

Chronic tubercular intoxication at children and teenagers

At a chronic tubercular intoxication characteristic signs become backlog of the child in development, pallor, micropolyadenia (6-9 groups of the increased lymph nodes - from elastic consistences to stones are palpated). At a chronic tubercular intoxication that fact is important, that after a bend tuberculin tests there has passed 1 year and more, and tuberculin tests remain positive, or accrue. At a chronic tubercular intoxication morphological changes of tubercular character in one or several bodies are found out: in a bone brain, lymph nodes and sometimes in parenchematoz bodies.

Unlike an early tubercular intoxication, at a chronic intoxication all symptoms are expressed more brightly and keep more with firmness. At patients are marked chronic conunctivits, flictens (a rash on conunctivits or a cornea) which that appear disappear. Appetite is sharply lowered. Sometimes take place dispeptic the phenomena or locks. Depending on duration of a chronic tubercular intoxication backlog of physical development, growth and especially weight of a body of the child is observed. As a rule, fall turgor all fabrics, a skin, hypodermic clenchatka is marked. Small periodic rise in temperature with fluctuation from 37 to 37, 5 is observed.

Reaction of the ill child to noisy games, hobbies, dialogue with children sharply varies. As a rule, children quickly get tired, aspire to retire and, that is not peculiar to children's age, quite often are early put to bed. Schoolboys become disseminated. At patients some morbidity around muscles of a humeral belt, on that party where illness extends is possible. Tuberculin tests are moderately expressed more often. In initial phases usually at a tussiculation of the patient it is possible to hear small, individual damp rattles and rigid breath.

Blood research on a subject centre a tuberculosis yields following results: The Phase infiltration - is observed small lymfopenia, and the left shift of formula SSE (speed of subsidence eritrocits) can reach 12 or 15 percent stick a kernel forms. Little expressed the fresh form - blood indicators basically normal. When illness in the productive form (a chronic current of illness) the centres in the sizes of 3-6 mm having the wrong or round form and sharp or average intensity are defined.

The roentgenogram can show the centres reaching of the sizes 1 sm in diameter. The centre the tuberculosis at some patients can not show almost any symptoms for a long time. But in most cases centre the tuberculosis is accompanied by appetite and work capacity decrease, sweat, weakness. Also there can be a short-term fever, heat in palms or cheeks and small subfebril temperature in the afternoon. In some cases even at initial stages there can be a dry and changeable cough with pains in a side. In due time to diagnose a tuberculosis in some cases difficult enough as at early stages of a current of illness symptoms can be very similar to symptoms of other diseases, it is similar on virus infection or a sharp pneumonia more often.

Clinically, rhentgen in lungs it is not possible to reveal obvious pathological changes. At «senior» children who are carriers of a long chronic tubercular intoxication, it is possible to find out the begun to live primary complex: centre Gon's and cicatricial changes of a root with petrificat in it.

The chronic tubercular intoxication can be prevented correctly and it is long spent treatment. Treatment of already developed chronic tubercular intoxication represents the big difficulties. In connection with formation of a fibrous capsule and unvascular a zone round defeat antitubercular preparations do not sate to the full the tubercular centre, and Mycobacterium, being in it, do not lose viability.

At schoolboys clinically the tuberculosis is characterised by rise in temperature to subfebril figures, appetite deterioration, occurrence neurovegetative frustration, a headache, a tachycardia, small increase in lymph nodes.

Especially hard proceeds dessiminiroval a tuberculosis. Allocate tifoid, pulmonary and minengial disease forms. At tifoid the long fever, the general intoxication are usually expressed the form, the liver and a spleen are increased. At the pulmonary form prevailing symptoms are a short wind and cianoz. At minengial to the form symptoms of a tubercular meningitis join.

The primary tuberculosis can proceed and chronically. Disease differs long torpid the period proceeding with symptoms of a chronic intoxication: rise in temperature to subfebril values, fatigue, weakness, appetite decrease.

The secondary tuberculosis meets at children of advanced age is more often. To features of a clinical current of a secondary tuberculosis at children and teenagers concern bent generalisation, high sensitivity of fabrics to tuberculin's, reaction of lymph nodes. In reнeзe a secondary tuberculosis the residual phenomena of a primary tuberculosis, a superinfection and such factors, as intercurrent diseases, overfatigue, insolacia, corticosreroid therapy matter, etc.

Current of a secondary tuberculosis at the majority of children smooth, not complicated. In 2-3 months after the begun antibacterial therapy the positive radiological picture is observed, there comes a phase resolve, consolidations or calcination.

The basic complications of this period: involving in pleura process, limfo-tropogennal dissemination tubercular process, destruction a pulmonary fabric, spontaneous pneumotorax, blooding spit, a bleeding, generalisation with out of lung tuberculosis localisations.

Tuberculosis diagnostics

Tuberculosis diagnostics is based on carrying out of reaction Mantu (skin test with cultivations tuberculin's). Dependence of intensity of reaction Manty on size postvaccinal sign is revealed. The more postvaccinala hem, the above sensitivity to tuberculin.

At helminthic invasias, hypertireoz, sharp diseases of bodies of breath, a virus hepatitis, the chronic centres of an infection sensitivity to tuberculin is raised. Besides, till 6 years of news agency (the infectious allergy) and antigene interaction is more intensively expressed at more senior children.

Antigene interaction (tuberculin) with receptors on a surface lymphocyte-effektors therefore there is an allocation mediators cellular immunity, involving macrofags in process of destruction of an antigene. The part of cages perishes, allocating proteolitic the enzymes, having damaging an effect on a fabric. Other cages accumulate round the defeat centres. Time of development and morphology of reactions at any ways of application tuberculin essentially do not differ from those at intraskin introduction. The peak of reaction Foreign aid - 48-72 when its nonspecific component is minimum, and specific reaches a maximum.

Sensitivity strengthening to tuberculin observe at statement of

test Manty in terms from 1 day till 10 months after carrying out of vaccination against children's infections (against a measles, against a parotitis vaccines). Earlier negative reactions become doubtful and positive, and in 1-2 years they become again negative, therefore carrying out tuberculindagnostic plan or before carrying out of preventive inoculations against children's infections, or not earlier than in 1 month after inoculations. It is even better not to do any inoculations.

Less expressed reactions on tuberculin register in the summer. Intensity tuberculin reactions decreases at feverish conditions, oncological diseases, virus children's infections, during time mensis, at treatment glucocorticoid hormones, antigistamin preparations.

The second diagnostic method radiological

For preventive maintenance of a tuberculosis at children inoculation, and preventive maintenance of a tuberculosis at pets have great value not. For this purpose it is necessary to co-operate with veterinary surgeons.

Ways of a transmission of infection: Infection with a tuberculosis arises through respiratory ways (aspiration a way) at contact to the infectious patient which allocates in an environment mikobaktyerii, containing in drop kernels and dust particles more often.

Only less than 10 % of these particles in the size are no more 5 microns get to alveoluses and cause infection. The overwhelming majority of other, larger, particles settles on a mucous membrane of the top respiratory ways, and leave therefrom thanks to function mucociliar a road clearance. At enteral infection soaking up function of intestines has certain value.

For inficiroval and developments of disease by a tuberculosis major importance massiveness of an infection, a dose and duration of receipt Mycobacterium in a human body, secondly, have, first, a condition of nonspecific and specific factors of protection in influence of the infectious agent. Mechanisms of protection of bronho-pulmonary system operate at level of spending and respiratory departments and are carried out for the account kondensiroval air, mechanical clearing, endocytoz bronchial endotelii, by cellular nonspecific protection. The intact mucous membrane of an oral cavity, nasopharynx and the top respiratory ways is an impenetrable barrier for Mycobacteruim. Here are carried out mechanical clearing of air at the expense of a filtration and sedimentation of alien particles, their removal owing to sneezing and cough.

The most significant part of mechanical clearing is mucociliar a road clearance at level of bronchial tubes and bronchiol. The road clearance is provided with a secret, containing lizocim, proteazas, surfactant, antibody A (IgA) which onsoniroval Mycobacterial, and movement pec nichek bronchial epitelii deduce them from an organism [1-4]. Infringement of an integrity of a mucous membrane of an oral cavity and nasopharynx, caries a teeth, aftoz a stomatitis, paradontoz, a sinusitis, a chronic tonsillitis, and also the periods of age change of a teeth at children can be entrance collars for a tubercular infection.

In a trachea and large bronchial tubes Mycobacterium, as a rule, are not late and settle in the bottom respiratory ways and alveoluses. Some part Mycobacterium remains on a penetration place, another with a lymph and blood current is transferred to an introduction place - to regional lymph nodes. Depending on localisation of entrance gate of an infection axillary intrachest and other regional lymph nodes can be amazed submaxillary, cervical, over and under clavicle.

In lymph nodes Mycobacterium cooperate with cages of immune system, causing the whole cascade of the reactions conducting to immunological to reorganisation of an organism to a tubercular infection and formation of specific cellular immunity. Local changes in an introduction place Mycobacterium are caused by nonspecific reaction polynuclear cages (neutrofils), which fagocitoz the activator.

Under the influence of toxins Mycobacterium neutrofils collapse, causing inflammatory reaction which is replaced by more perfect protective reaction with participation of the macrophages which are carrying out fagocitoz and destruction of the activator. Efficiency of all system of antitubercular protection of an organism in many respects depends on activity fagocitoz. These possibilities destroy inoculation.

The macrofags in the centre of an inflammation take the form epitelioid of cages which merge together, form huge cages, however at a tuberculosis macrofags independently destroy Mycobacterium can't. Activating influence on them sensibility T-lymphocyte, in particular is necessary for subpopulation T-helpers (CD4 + cages) and T-supressors (CD8 + cages).

At aerogene infection micobacterium, reached alveoluses, are exposed fagocitoz alveolar macrophages. The macrofags fix micobacterium on a cellular membrane, then immerse them in cage cytoplasm. Mechanisms with which help these cages destroy Mycobacterium already well-known; antimicobacterial effector functions of macrophages: formation fagosomo-lizosomnyh complexes, generation of active forms of oxygen (AFO) at so-called respiratory explosion and formation of the is free-radical intermediaries, carried out on L-arginin to a dependent cytotoxic way.

The first of these mechanisms is connected with nonspecific function of macrophages; two second are carried out at influence sensibility T-lymphocyte. In a trachea and large bronchial tubes Mycobacterium, as a rule, are not late and settle in the bottom respiratory ways and alveoluses. Some part micobacterium remains on a penetration place, another with a lymph and blood current is transferred to an introduction place - to regional lymph nodes. Depending on localisation of entrance gate of an infection axillary intrachest and other regional lymph nodes can be amazed submaxillary, cervical, over - and under clavicle.

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Formation fagosom-lizosomal complexes occurs as a result of merge fagosoms, containing Mycobacterium, with lizosoms. The lizosoms represent rather difficult complex organells, containing in the membranes the big number of the enzymes, capable to destroy the most various macromolecules. Optimum conditions for functioning of these fermental systems are provided thanks to weak acidity of environment inside lizosom (pH nearby 5); in maintenance pH the important role belongs to the ATF-dependent ionic pump.

The Mycobacterium, fagocits macrophages, collapse in fagosom's these cages under the influence of various lizosomal enzymes as a result of merge fagosom-lizosome a complex. However Mycobacterium, getting in macrofags, can remain in fagosoms and even to continue reproduction; thus fagocitoz has not complete character. It is established, that Mycobacterium can produce ammonia which, on the one hand, is capable to ingibitor merge fagosom's with lizosome's, and with another - by alkaline contents lizosom's to reduce it fermentative activity. In cases when digestion process mycobacterium is blocked, macrofags collapse, and activators leave the cages which have absorbed them. It is proved, that it is connected with the toxic substances released at destruction Mycobacterium; it first of all concerns the cord-factor (the factor virulent).

The cord-factor plays a key role in development of a secondary immunodeficiency and sharp inflammatory process in bodies and organism fabrics

Cord-factor, first, it is destructive operates on a power metabolism of cages of a macroorganism, causing defeat mitochondrium and carrying over infringement electrons on a respiratory chain between Koenzim's Q and cytochrome; secondly, synthesis lizosomal enzymes that protects in cages located Mycobacterium from destruction brakes and, thirdly, oppresses synthesis CD4 + linfocits interferon-scale (INF-SCALE) which concerns the important factor of activation of macrophages in their ability to strengthen production H202. It has toxic an effect on macrofags at fagocitoz micobacterium.

Raised virulent also it is connected with activity katalaza/peroksidaza which raises endocellular survival rate of the activator, protecting it from mechanisms lizis's in a macrophage. The important antimicrobic mechanism of the activated macrophages directed against endocellular activators, is connected with cytotoxic action L-arginin's. The cytotoxic effect of this mechanism is mediated oxid nitrogen (N0) and connected with AFO which are generated with participation L-arginin's and by means of factor action necros the tumours, synthesised by the activated macrophages.

Thus, macrophages play a double role, providing not only protection against a tuberculosis, but also creating favorable conditions for a survival mycobacterial diseases. Thus fagocitoz is the unique natural mechanism of destruction mycobacterium in a human body. At a tubercular infection macrofags play a key role in representation T-lymphocyte Mycobacterium, formation of specific immunity with the subsequent destruction of the activator. All depends on that, how much macrofags during the given concrete moment are functionally high-grade for destruction fagocitar them of activators.

The organism tries to localise an infection therefore it is formed so-called GZTc, being the indispensable mechanism in formation of cellular antitubercular immunity. The antigens mediates development of the cellular immunity directed on localisation of a tubercular inflammation in the infected organism, and creation of the got immunity directed on destruction Mycobacterium.

At primary inficitar immunity formation occurs in parallel reproduction Mycobacterium in cages and organism fabrics.

Morphological equivalent of protective cellular reactions of an organism against a tubercular infection is specific granulema

In granulema there are three kinds of cellular elements. The centre and its main weight make epileoid cages, on huge multinuclear cages Pirogova-Langhans's. The result of interaction depends on a condition of functional system of cellular immunity macro-and a microorganism which can end with formation of relative immunity, or at its inconsistency conducts to development of disease and tuberculosis progressing. In these cases fabric cellular reactions not in a condition to delimit and localise a specific inflammation and its distribution in the amazed body and then disease develops.

In natural conditions of infection antigens develops in 2-3 weeks after inficiroval, and expressed enough immunity is formed approximately in 6-8 weeks.

The leading part in resistibility of an organism of a tubercular infection is taken away to the got cellular immunity. In its basis activation of macrophages and effector their influence on T-lymphocyte lays. Thus macrofags represent actually effectors, and T-lymphocyte carry out a role inductors antigens. In the same way as macrofags, T-lymphocyte are not only a necessary component of antitubercular immunity, but also are the factor defining patogenez diseases. Interaction coordination between macrophages and T-cages consider as the central link in formation of antitubercular immunity.

Formation of antitubercular immunity is provided with all populations T-lymphocyte, but the basic role belongs CD4 + (T-limfosis helpers) and CD8 + to cages (T-lymphocyte supressors). CD4 + cages are capable to distinguish antigenes micobacteruim, fagocitiroval macrophages, and play the important role in antitubercular immunity. Accordingly it explains, why reduction of quantity CD4 + cages as a result of a HIV-infection often leads to a tuberculosis aggravation. CD4 + limfocits in a significant amount produce Interferon-gamma. The same interferon allocate sensibility Mycobacterium CD8 + limfocits. The Interferon-gamma is the main thing mediator resistance to a tuberculosis, raising digesting abilities of macrophages.

The tuberculosis amazes lungs: The body which is the basic collars of an infection, more often. More often, it is 1 and 2 shares (supraclavicular and subclavian) the right lung. The activators which have got to alveoluses, fagocitoz the alveolar macrophages which have been obviously insufficiently prepared for destruction Mycobacterium.

Together with macrophages these activators are transferred in pulmonary parenchima and further in regionar lymph nodes where there is their reproduction. Infected macrofags, fagocitoz Mycobacterium and carrying out their digestion, fragments destroyed micobacterium, proteolytic allocate in extracellular space enzymes, mediators, including interleukin-1 (IL-1) which activate T-lymphocyte, in particular CD4+ cell. The last allocate limfokins, in particular IL-2 and Interferon-gamma under which influence there is a migration of new macrophages to a place of localisation of the activator and their activation. Activated macgofags in addition is secreted the factor necros

tumours and (FNO), initiating formation productive granulems, that limits distribution Mycobacterium and interferes with their reproduction.

However similar granuloma not in a condition completely to eliminate the activator. In particular, huge multinuclear cages Pirogova-Langhans's become storehouse Mycobacteruim and not in a condition to destroy the endocellular inhabitants. Full elimination of the activator at a tuberculosis is not reached even at well coordinated interaction of macrophages and T-lymphocyte therefore the organism remains infected though active pathological process develops far not in all cases.

Any infringement of immune balance of the next years creates conditions for reacrivation the remained population Mycobacteruim and developments of clinically expressed forms of disease. In the subsequent productive granulema's it's centre necroz is surrounded with a dense fibrous capsule, and. The important role in incapsulation granulem's and in development central it becroz's plays FNO. Border stay zones mycobacterium and central necroz reduce P02 and create adverse conditions for growth Mycobacterium.

At intensive reproduction mycobacterium in a human body owing to ineffective phagocytosis the big number of the toxic substances causing infringements of an endocellular metabolism of macrophages and T-lymphocyte is allocated. These infringements develop of oppression of the power metabolism shown in deep depression of activity of enzymes mitochondrial of oxidation and anaerob glicoliz's, infringements of synthesis ATF, DNA, RNK, amino acids and fiber.

It conducts behind itself aggregation and labilization lizosom's, to their exit contained in citozol, to damage of endocellular structures and the cage; especially these changes are expressed in subpopulation CD4 + cages. Thus arises not only their quantitative reduction, but also decrease in level of synthesis IL-2 by them and Interferon-gamma. Such cages are little life ability and little active and at activation in a significant amount are exposed apoptoz's (the programmed destruction), that conducts to formation of a secondary immunodeficiency. It promotes development exudative a component of an inflammation with development kazeos necroz's and can lead to fusion of fabrics and break necrotic weights in a gleam of bronchial tubes or vessels.

Presence of cellular disintegration and high sizes PO2, this environment create optimum conditions for reproduction Mycobacterium. Break kaseoz a bronchial tube contained in a gleam leads dissemination process at laying lung sites, and break in a vessel - to generalisation and distribution of process in the remote bodies.

3.8.2. The vicious circle is formed: Toxins Mycobacterium cause metabolic infringements in macrophages and cages of the T-cellular link of immunity which conduct to system membrano-damaging effect and raised them apoptoz's. It, in turn, creates the extremely negative influence on cooperation Immunocompetent cages in system «a macrophage - CD4 + limfocit - a macrophage.

The expressed immunodeficiency when Immunocompetent cage not in condition to render due resistance of an infection is formed and perish in a considerable quantity (necrobioz), that, in turn, conducts to rough and massive reproduction of population Mycobacterium and to progressing of tubercular process. Along with macrophages and T-lymphocyte necrobioz's at a tuberculosis are subject and neutrofils. It is necessary to notice, that raised necrobioz, the leader to quantity reduction Immunocompetent cages, is accompanied by considerable decrease in synthesis IL-2 and Interferon-gamma. The adequate specific chemotherapy conducting to destruction of population Mycobacterium in amazed body, essentially reduces intensity necrobioz and conducts to increase in synthesis IL-2 and Interferon-gamma [5].

At lungs sick of a tuberculosis with an immunodeficiency of infringement of a metabolism with membrano-damaging effect have system character and are observed in cages of various bodies and systems, that substantially defines variety of clinical displays at the given pathology. These changes have paraspecific toksiko-allergic character and their morphological equivalent is infringement of function of concrete bodies and organism systems. It leaves an essential mark on clinical displays of disease. Such patients have relative insufficiency of function of a bark of adrenal glands, decrease synthetic and detoxic liver functions, dystrophic and functional changes of a myocardium, kidneys, peripheral and central nervous system (CNS), etc.

In tuberculosis development it is possible to track two periods when cellular reactions of an organism to introduction Mycobacterium carry accurately differentiated Immunological, morphological and clinical displays.

These periods are connected with reaction of an organism on primary exogen the infection defined as a primary tuberculosis, and on the reaction caused by secondary infection (exogen a superinfection) or reactivation already begun to live postprimary changes (endogen an infection), carrying the name a secondary tuberculosis.

Depending on entrance gate of primary introduction Mycobacterium the inflammatory centre, or the primary centre, can be formed in lungs, a mouth, tonsils, intestines, etc. the Subsequent reproduction of the activator occurs both in lungs, and in lymph nodes, and the organism reacts formation specific granulem's (hillock), the primary effect is formed.

Thus, from the moment of primary infection the tubercular infection carries generalize and the system character giving in subsequent possibility of the development inside lungs of forms of a tuberculosis.

The primary tuberculosis as a result exogen infections develops only at 7-10% of the infected persons, the others transfer a primary tubercular infection without clinical displays and spontaneously recover. The come infection is shown only in transition negative tuberculin reactions in positive (a bend tuberkulynovoy sensitivity).

Absence of clinical displays of a primary tubercular infection can be explained high level of natural resistance of a human body to a tuberculosis, and also can be a consequence of the artificial immunity got as a result of vaccination. The inoculation can mask a tuberculosis. But does not protect from its occurrence.

After a primary tuberculosis there can be the form of the tubercular defeat shown extended hematogen or limfogen dissemination. Such kind of tubercular defeat defined as dissemiroval a tuberculosis, is genetically connected or with progressing of a primary infection, or with reactivation after the primary centres.

The primary tuberculosis can come to the end with treatment with the minimum (small) or expressed enough changes, for which are characteristic calcification or kalcination specific changes in lungs (centres Gona's and Simona's) and intrachest lymph nodes (petrificats). At such people the got immunity is formed.

Preservation in the residual centres persistiring Mycobacterium not only supports immunity, but simultaneously creates risk endogen reactivation tubercular process owing to reversion the changed forms of the activator (Z-forms, etc.) in the bacterial form and reproduction micobacterial populations. The mechanism consists in it endogen

reactivation at development of a secondary tuberculosis.

Other way of development of a secondary tuberculosis - exogen, connected with new (repeated) infection Mycobacterium (superinfection) is possible also. At the same time both for endogen, and for exogen it is not enough way of development of a tuberculosis only penetration micobacteruim in already infected organism even at a massive superinfection. Set of some conditions and the risk factors reducing immunity is necessary.

In development of a secondary tuberculosis by a necessary condition for disease immunity decrease, including specific which "break" does not provide the sufficient control over breeding population

mycobacterial diseases is thus, as a rule, at 90 % of patients clinical displays of disease develop and practically there is no tendency to spontaneous treatment that is characteristic for a primary tuberculosis.

Application of antitubercular preparations can lead completely to treatment from a tuberculosis. At the same time crucial importance belongs to a macroorganism, a condition of its protective mechanisms, ability to resist to activator action, and also development high-grade reparation processes.

At the heart of a tubercular inflammation three classical kinds of fabric reactions lay: Alteration (necroz), exudation (inflow of cages and liquids from vessels), proliferation (reproduction of cages in the inflammation centre). Depending on immunologiacal conditions of an organism and pathogenicity degree Mycobacterium this or that reaction prevails, all variety of forms of a tuberculosis of lungs is based on it and their current.

Various combinations patomorfological displays create preconditions for extremely big variety of tubercular changes, especially at a chronic current of disease with change of the periods of an aggravation and silent process. To it it is necessary to add and the various complications connected with features of specific process, such, as distribution micobacterium with a lymph or blood current, destruction of the amazed body with cavity formation, defeat of vessels with development blood to spit and pulmonary bleedings, infringement of exchange processes with development amiloidoz internal bodies, etc.

A variety of morphological reactions in bodies and fabrics at a tuberculosis depends from patogenez, forms, stages, localisation and prevalence of pathological process and first of all is connected with a condition and functional activity of immune system of a human body at the moment of development of a specific inflammation.

In morphological reactions of a specific inflammation at a primary and secondary tuberculosis in experiment R.Kokh has shown distinctions still. After a primary hypodermic inoculation in this place in 10-14 days there is not healing ulcer, and are necessarily amazed regionar lymph nodes. Repeated introduction Mycobacterium also leads to ulcer formation, but thus is not observed defeats regionar lymph nodes (Kokh's phenomenon).

The general for the majority of forms of a tuberculosis are specific changes in a combination to nonspecific or paraspecific reactions

The tubercular inflammation which is expressed in three basic forms of inflammatory reactions concerns specific changes: Mainly productive (with development granulems), mainly exssudative (with development exssudat) and necrotic, or curdled (with development primary necroz fabrics).

Development of a productive inflammation and formation granulems is characteristic for high level nonspecific and Immunological reactance of an organism. Thus in granulema's there are three kinds of morphological elements. The centre is presented by a site kaseoz necroz, surrounded with cellular elements, basically epileoid cages and huge multinuclear cages Pirogova-Langhansa, on periphery settle down limfocits and plasmatic cages, and also neutrofil leukocytes.

Thus caseous changes are rather limited and are exposed slow to resolve with the subsequent sclerosis, fibroz and incapsulation on periphery most necroz. Further at specific treatment granulema is exposed to fibrous transformation.

These processes are observed at central, limited dissimination and infiltrat a tuberculosis of lungs and tuberculema's, and also at limited specific out of lungs defeats in lymph nodes, a brain, bones, joints and other bodies. Immunologichesky researches at the given category of patients do not reveal infringements, both in a quantitative parity, and in decrease in functional activity Immunocompetent cages. Such forms of a tuberculosis, as a rule, come to light at patients at regular preventive inspections, proceed without a symptom, or little a symptom.

At process progressing the increase kaseoz necroz is observed, amplifies infiltration granulation fabrics macrophages, limfiod cages and neutrofils. Specific process contact and limfogenic by extends. In the morphological plan: exudative the tubercular inflammation in lungs has character of a nonspecific pneumonia and is characterised sharp alveolit's and centre or diffus allocation fibrin, leukocytes, sometimes eritrocits. The so-called phase of a nonspecific inflammation thus takes place.

Gradually on periphery appear specific granulems, characteristic for a tubercular inflammation. There are sites of a share specific pneumonia with defeat of bronchial tubes (bronho-share infiltrations) and with the tendency to merge and distribution of process within 1-2 segments, is more rare - lung shares. Tubercular infiltrations in lungs are exposed kaseoz to transformation. Kaseoz weights badly resolve and tend to fusion and formation of cavities. The formed cavity is a receipt source Mycobacterium in other departments of lungs and formation of the new centres, infiltrats and cavities.

The combination of nonspecific and specific phases of an inflammation, their alternation is one of the reasons of polymorphism of kliniko-morphological displays of a tuberculosis

At weight healing kaseoz necroz are condensed, in the subsequent adjournment of small grains of salts of calcium is marked. In granulation fabrics the quantity fibroblasts and fibrill the collagen, uniting in collagenic fibres which form connect a fabric a capsule round the tubercular centre increases. In the subsequent specific granulation the fabric is more and more replaced with a fibrous fabric.

These processes characterise infiltration a tuberculosis of lungs. In a clinical picture such patients on the foreground have an intoxication syndrome, and at disintegration of a pulmonary fabric and formation of cavities - bronho-pulmonary displays.The more volume of defeat with exssudative-kazeoz changes, the more sharply clinical displays of disease. At out of lungs localisations functions of the amazed body are broken. At patients with prevalence exssudative a component of an inflammation the immunodeficiency is characterised by moderately lowered quantitative and functional infringements in system of cellular immunity. In the presence of primary curdled necrozas the considerable functional infringements combined with expressed quantitative deficiency of T-cages and macrophages are marked; especially it is

characteristic for patients with sochetannym a current of a tuberculosis and a HIV-infection. Curdled necroz lung fabrics it is characterised by formation extensive share and lobular the defeats of a pulmonary fabric consisting of the centres merging with each other primary necroz's of a pulmonary fabric with very poor specific cellular reaction epileoid of cages, lymphocytes and prevalence neutrofils.

Curdled necroz it is observed in not changed fabric of a lung, sometimes with the subsequent development round sites necroz's specific granulomas. It distinguishes the given kind of specific defeat from exssudative inflammations where exssudat, instead of necroz, is a dominating component of a specific inflammation.

The given kind of specific defeat of a pulmonary fabric is allocated in the separate clinical form - kaseoz a pneumonia which is one of sharp and malignant versions of a current of illness. At out lungs defeats development curdled necroz's leads to full destruction of the amazed body. At a curdled inflammation along with development necroz's there is a system defeat microcircular channels of productive character, and also trombogemorragicheskih the changes conducting to an ischemia and fast некроз of amazed sites of a lung or other bodies.

Vascular defeats have universal character and concern both veins, and arteries: In one cases they are expressed perivescular cellular infiltration mononuclear character, in others inflammatory infiltration can grasp all layers of a wall of a vessel and then develops destructive vasculit, and are observed fibrioid necrozs vessels.

At this kind of a specific inflammation quickly there comes curdled regeneration of liquid and cellular elements inflammatory exssudat with formation in the beginning dry, and then liquid necrotic weights. Patogistologic research finds out a picture of a pneumonia of the mixed type in lungs. In one alveoluses there is a weight of fibrin and leukocytes, and in others the congestion liquid exssudat or mononuclear cages prevails. Very quickly there can come the fibrin organisation that gives a picture karnification (pathological change of a pulmonary fabric at which it gets a consistence and a kind of crude meat).

The pleura is necessarily involved in process with formation pleural kaseoz stratifications visceral and parietal. Increase curdled necroz's which quickly, sometimes within 2-3 weeks, extends on the increasing sites of a pulmonary fabric, is quite often accompanied secvestration necrotic lung sites. Are thus formed sekvestriral cavities of the wrong form with rough and it is indistinct conturing edges or a purulent softening kaseoz weights with formation of cavities of various size - from small to huge. At curdled necrosis changes begin in a respiratory part acinus's, and then very quickly amaze all acinus, that causes formation in the beginning kazeos endobrobchiolits, and then kaseoz endobronchits in larger bronchial tubes. Sharply developing and morfolog irreversible defeats of lungs at curdled necroz's are accompanied by the expressed syndrome of an intoxication, bronho-pulmonary displays and lead to development of respiratory insufficiency, and also to deep metabolic and haemodynamic to myocardium changes.

Healing at curdled necroz's goes the slowed down rates in view of extensiveness of morphological damages of a lung and the big population Mycobacteruim. As one of rare outcomes curdled necroz's at effective chemotherapy is marked cirrhosis development, however clinical treatment is possible only at operative treatment and removal of the basic centre of specific defeat of easy and other bodies. In cardiovascular and nervous system, blooding bodies, serous covers and other fabrics there can be so-called toksiko-allergic or paraspecific reactions as result antigenemia's and linkages of antigenes Mycobacterium with fibers of blood with formation of large specific immune complexes. Morfolog these changes are shown centre or diffuse macrophagal and limfocitar infiltration as a result of defeat by these complexes of corresponding bodies and systems.

The current and tuberculosis outcomes should be considered only in the conditions of specific chemotherapy which should be appointed all sick active tuberculosis of lungs. In the course of chemotherapy influence destroying it on the activator owing to what quantity Mycobacterium sharply decreases is marked and favorable conditions for development reparative processes [6-12] are created.

At the same time at chemotherapy application the different current of tubercular process is marked: Regress with the subsequent healing and clinical treatment; stabilisation without clinical treatment with preservation of a cavity or other changes; time leaving inflammatory process with the subsequent occurrence of an aggravation; development of chronic process or disease progressing [13-16].

Tubercular granulema in a lung

Defeat of shares of lungs at a tuberculosis: Usually right lung of 1 and 2 shares (over clavicle and under clavicle). Tuberculema amazes the same shares. Complexity of revealing tuberculema that shades on the roentgenogram that are visible are not visible (Figure 1). The capsule does not allow to consider a defeat zone. Are thus marked small cough with secret of bronchial tubes, short subfebrilitet, unsharply expressed leukocitoz, shift leukocitar formulas to the left, increase speed of subsidence eritrocits (SSE). Disintegration of a pulmonary fabric and dissemination in 30-40 % of cases are accompanied bacterial allocation. In the absence of treatment, illness, as a rule progresses. At children tuberculem's meet seldom. The analysis of blood does not represent special changes, in case of aggravations moderate acceleration SSE and leukocitoz moderate character is observed.

At stable tuberculem's Mycobacterium in secret of bronchial tubes are not found out. In case of disintegration in tuberculems' bacillas allocation meets in a case when there is a communication with a drainage bronchial tube. In most cases patients with tuberculemas lungs positively react on tuberculin, test Manty mainly has hyperergic character. By 20 years tuberculemas without corresponding treatment are formed more often at men of 20-29 years.

As the activator continues to breed, and immunity was not generated yet, activator population considerably increases.

The capsule tuberculem's consists of two layers (Figure 2). The inside layer formed by tubercular granulations, surrounds kaseoz a kernel tuberculem's. The external layer presented concentric by located fibrous fibres. Delimits tuberculem from at the laying not enough the changed fabric of a lung. Massive kaseoz a kernel and thin (1-1, 5 mm), well generated fibrous capsule - characteristic morphological signs of the most widespread type tuberculem's – kaseoma's. For infiltrative-pneumonic type tuberculems are characteristic alternation of sites kaseoz necroz's with epitelioid-cellular hillocks and weak development of a capsule. For children it is not characteristic.

Antimicrobic spectrum Sangvirintrin's (Table 1).

Sangviritrin - represents a mix of bisulphates quarter benzofenantridin alkaloids sangvinarin's and cholelitrin's

The action mechanism Sangviritrin's: suppresses bacterial nucleaza's: The new gene which plays the important role in occurrence of a tuberculosis of lungs is found. The gene has received name TLR8, is in X to a chromosome. Men are more sensitive to a tuberculosis as have only one X a chromosome and, accordingly, only one variant of a gene.

Women, thanks to two X to chromosomes, have two different variants of the same gene that promotes the raised firmness of an organism to an infection.

Third of inhabitants of a planet are infected by a tuberculosis, but only at 5 - 10% will arise disease because of presence of genetic defect. Unfortunately, in my research genno-molecular analyses in our conditions to make it is not obviously possible. At the majority the infection is in a latent condition, anything of not showing. «The genetic signature» signals that disease amazes lungs and disappears after successful treatment [1]. The pulmonary tuberculosis is connected with increased oxidant stress which is not connected with smoking and characterised by the increased levels peroxid lipids and low concentration of vitamin E in plasma, therefore for secondary preventive maintenance antioxidants and vitamin E are necessary to such patients. These means and for primary preventive maintenance of this terrible disease [2] are good.

The tuberculosis can catch through the cow milk. Micobacteruim a tuberculosis can appear both in milk, and in meat of sick animals. These products should be bought, either in shop, or at presence at the dealer of the inquiry that they are received from healthy cattle. But all the same unbottled milk should be boiled. And meat to cook or fry to full readiness. Fans of beefsteaks with blood risk to pay for the predilection with own health.

According to A.L.Chizhevsky's doctrine about geliobiology each 11, 30, 50 years on the sun are formed superprotuberanci, and on the earth after that to begin mutations first of all microorganisms, to appear new schtamms new kinds of illnesses. Mutation «Kokh's stick». To medicines already it is a lot of years, therefore medicines can lose efficiency against Kokh's sticks.

Density in prisons to Russia, absence there necessary medicines, is created by resistant forms of a tuberculosis which they bear to us in a society.

Contactors with a tuberculosis

1. Doctors, pharmacists, medical personnel, members of their families.
2. Persons till 30 years.
3. Persons, having chronic diseases of lungs, diabetes.
4. Persons, long time accepting hormonal preparations.
5. Smokers.
6. Emloyers colonies, prisoners.
7. Vagabonds.

Usually top shares of lungs (overclavicle and underclavicle) are amazed! The basic way of infection of a tuberculosis - air-drop!

Diagnostics of a tuberculosis at children

1. Reaction on tuberculin - Manty - is not specific. Shows presence of any inflammation and an allergy in an organism of the child.

2. Usually diagnostics of the pulmonary form of tuberculosis is based on the analysis secret of bronchial tubes. It is possible to carry complexities. To minuses of this method with gathering of enough of a material, difficulty in revealing Mycobacterium because of density of slime. Besides, the patient at gathering secret of bronchial tubes should cough, that increases risk for medical workers. For children the method is not necessary.

3. Earlier attempts of definition Mycobacterium in other biological liquids were unsuccessful, their sensitivity did not exceed 50 %. In the previous researches it has been shown, that DNA Mycobacterium can be grasped by cages epitelium a mouth of humanoid and other primacies and then come to light by means of polycepomeraz reaction (PCR) in dabs from a mouth. In the given work the test based on this method, has been estimated in clinical conditions at patients with pulmonary forms of tuberculosis.

New method PCR: To analyze presence of a unique complex in DNA Mycobacterium - IS6110. From 20 patients with a pulmonary tuberculosis at 18 at least in two dabs from three DNA fragments Mycobacterium, even have been found out at the analysis of a small part of volume of test. DNA revealing Mycobacterium can become an effective method of diagnostics of a tuberculosis among risk groups, and also at patients whom not in a condition to hand over secret of bronchial tubes on the analysis, for example, at small children. Scientists hope, that perfection of technology of a fence of a material can raise sensitivity of a method which in the given research has made 90 % [14-20].

Diagnostics at adults

1.Diascin-test (similar, but more sensitive test).

2.Fluorographiya (once a year).

3.Computer tomographya lungs.

3.16. Immunity at a tuberculosis has the features

It not sterile, is supported by bacteria, persistiroval in an organism and providing a condition inficiroval. Immunity unstable [20- 22].

CD4 T-lymphocyte play the important role in formation of antitubercular immunity. Especially their role became clear in connection with growth of number sick of a tuberculosis among the persons infected with a virus of AIDS. Among this group of patients especially high growth of number of patients with reactivity a latent tuberculosis is marked. The pathogenetic role of this population limfocits is predetermined by their ability to distinguish antigenes of the big complex histocompatibility (major histocompatibility complex, or in the reduced form - MHC II a class). So are distinguished dendrit cages and macrofags, in vacuols which is antigene peptid.

Contrary to these Immunological - I molecules of antigenes which activate CD8 T-lymphocyte are presented mechanisms MHC. This Immunological mechanism supervises transport of an antigene from cytoplasm in endoplasmic reticulum. As Mycobacterium tuberculosis initially lives in vacuols more than in cytoplasm of cages the role of the given population limfocits in formation of antitubercular immunity, appear, is improbable. However in researches which have been spent [23], participation CD8 T-lymphocyte in mechanisms elimination *M. tuberculosis* has been shown from a spleen fabric.

Antibacterial activity CD8 T-limfocits can reach some ways. Lymphocyte this population can be production source such citokins, which are Interferon-gamma (IFN-g) and TNF-a. CD8 T- lymphocyte can render protective effect direct action, directed against macrophages, being in fabrics and grasped *M. tuberculosis*. Prodaction citokins games important role in activation macrofags. CD4, CD8 T-limfocits allocate a secret INF-g and TNF-a, which concentration increases in the inflammation centre. Other mechanism, in helping which inficiroval macrofags CD8 T-limfocits connect with ability cages to kill macrofags within help perforin's. Perforin is protein, which sintez of granulas

CD8 T-limfocits. In helping specified protein's perforid membrana of macrofags, and via it inside cage's get such toxic peptids, which are granzims or granulizin, which accelerate process apoptoz's macrofags. Apoptoz macrofags can be carry out via machanism Fas-ligand's too, which brings to activation CD8 T-limfocits (7, 8, 9).

CD8 T-limfocits has some machanizms antibacterial actions, among which direct cytotoxic effects, participation in production proinflammatory citokins, variety synthesis peptids with the expressed toxic properties are more studied. These cages can compensate functional inferiority CD4 T-lymphocyte substantially.

Researches [24] the direct cytotoxic action CD8 T-lymphocyte directed against *M. tuberculosis* has been shown, being in a cage. This mechanism of direct defeat Mycobacterium ability human limfocits prodaction granulizin.

Everyone T-limfosit has specific epitop, or a short chain of amino acids in antigene structure. Identification of antigenes, or epitops is the important stage in decoding of mechanisms of protection as this information is necessary for using in designing of new generation of medicines. In classical variant CD8 T-lymphocyte are distinguished peptids, entering into structure MNC Ia. This population limfocits is capable to distinguish also antigenes MNC I, thus are available in view of such molecules, as CD1 or MNC Ib. Research genom's Mycobacterium dictates necessity to receive the information on a role and a place classical and not a classical way of its interaction with immune system of the owner. These data matter today in treatment of different clinical displays of a tuberculosis. So, the characteristic classical and nonclassical MNC has allowed to allocate clones limfocits with raised and lowered production IFN-g. Specific antigenes *M. tuberculosis* which influence anti-inflammatory activity T-lymphocyte, are secretor antigenes and include such, as 6, Ag85A, Ag85B, 38 kD, shock fiber 65 and lipoprotein 19 kD. CD8 T-lymphocyte have specific epitops for early secretor an antigene 6 which is absent in M. bovis, Bacille Calmette-Guerin (BCG). With the given antigene connect activation limfocits and the beginning of production of interferon. Inoculation does not activate limfocits and does not give start of production of interferon.

Now ability limfocits to production of interferon G (gamma) depending on activity of tubercular process and participation as classical way MNC-Ia, and not classical MNC-Ib [25] have established, that at healthy individuals who had a positive reaction on tuberculin CD8, T-limfocits synthesise lower concentration scale - interferon, than at healthy people at whom reaction on tuberculin was negative. It has been thus established, that more than in 96 % of cases it was observed not classical MNC-Ib.

I always surprised, if reaction Manty was negative imposed to do inoculations to children and adults, if the organism itself consulted not to be ill, the met infection to such people was not terrible.

It is necessary to underline their low efficiency of vaccines in struggle against a tuberculosis at children and adults. To tell more fairly, vaccines not preventive tuberculosis occurrence. It recognises also officials from medicine. This fact is of great importance in planning of new approaches in treatment of preparations of new generation. It is necessary to give great value and to such factor, as ability CD8 T-lymphocyte to distinguish macrofags, which fagocitar *M. tuberculosis* and to lead to death of them it is direct in the centre of inflammatory reaction.

Early clinical signs of a tuberculosis at children

1. Quickly fatigue

2. Increase body temperatures at 12-14 o'clock, instead of at 16-18 o'clock - some weeks, then rise in temperature from 15-17 o'clock in the afternoon, slight increase of temperature to 37-37. 5 degrees

3. Micropolyadenit (increase in lymph nodes behind nod muscles).

4. Night sweats.

5. Not the steady appetite.

6. Decrease in weight of a body 7.specific (feverish) shine in eyes

Diagnostics of a tuberculosis at children

1. Crops a secret of bronchial tubes it is problematic - children cannot from to spit and spit out a secret of bronchial tubes.

2. Sharp positive reaction Manty or full absence of reaction at test Manty in cultivation 1:1000.

3. Tussi or the tussiculation with allocation a secret of bronchial tubes, is possible with blood (at tuberculem's (incapsular a tuberculosis) - there is no cough and a secret of bronchial tubes, and Kokh's sticks are not sowed)!

Diagnostics methods

Analyses were made in Immunological laboratories of the Federal Center of health of a family of of Ivanovo. Crops did in microbiological laboratory of the Center of hygiene of of Ivanovo. Data on innoculation the companies took in children's polyclinic N of 6 of Ivanovo.

Data on patients

In research took children from 3 till 10 years which imparted and have been not imparted, only 39 children.

Results

If the inoculation is made to any child till 3 years fagocitar activity CD8 T-lymphocyte will be low always. It is checked up by me on the big population of children (reliability p<0, 05). During postnatal lives insufficiency fagocitar a link is traced and the secondary immunodeficiency with insufficiency gumoral immunity develops. It conducts to frequent illnesses, a syndrome of strengthening of an infection, a respiratory distress-syndrome. The death of such kids often comes within 1-2 years after inoculation. But in official medicine persistently do not connect deadly cases with inoculations. Moreover, it is cases disappear in Russia.

M antibodies have been raised at 10 children (3-14 years) more often at children-boys with blond hair - till 1 g/l c the complicated current of tubercular process. More decrease T-lymphocyte has been noted at children with for the first time revealed infiltrative processes. According to NST-TESTS decrease functional fagocitar activity was marked.

It testifies that antiteloformed at a tuberculosis - result of long antigene influence and a consequence of changes of a parity regulator cages (transition with Th1 on Th2). Accurate dependence between concentration of antibodies and an immunity condition it is not established. Still [26] noticed, that antibodies at a tuberculosis are only witnesses of development of cellular reactions. They will not neutralise neither tuberculin, nor Mycobacterium [27,28]. But inoculations do till now though, it is proved, that the antibodies formed after inoculations, do not protect from illness development. Antibodies develop with the kavernoz-fibrous form of illness is more often. Antibodies only show

duration of antigene influence. Antibodies strengthen activity

phagocytic reactions, but the inoculation causes fagocitar recession. The contradiction unsoluble till now. Absorbing function of phagocytes is kept, and here functional activity of phagocytes considerably is exhausted.

How one of founders of a vaccine of A.Kalmet could create a useless vaccine? Though, writes truly about antibodies and not finished fagocitoz. The second author of creation of vaccine Geren, that obviously too suffered paralogic thinking. Otherwise, they would not create this vaccine. If at inficiroval fagocitoz not finished by manufacture of inoculations it will be not even more complete. From here, and uselessness of a vaccine, and many children's death, and allocation of the whole population of children, as often ill children (long-term supervision of the author).

Higher production mieloperoxidaza's prevails at exssudat an inflammation at a tuberculosis. Definition mieloperoxidaza's needs to be included in diagnostics standards exssudative tuberculosis phases [17,29].

Indicators of immunity at imparted children with not complicated and complicated current of illness

Indicators of immunity at imparted children with not complicated and complicated (Table 2). Localisation Immunological insufficiency at patients a tuberculosis of 2-3 severity levels (Figure 3). Features gumoral immunity at children in groups with the complicated and not complicated current (Figure 4). Features of indicators of system of cellular immunity at children in groups with the complicated and not complicated current (Figure 5). Features of indicators of immunity at children in groups with the complicated and not complicated current (Figure 6). Features of the maintenance Lizocim's at children in groups with the complicated and not complicated current (Figure 7).

Results of the statistical analysis of features of system of cellular immunity during the periods of the complicated and not complicated current of illness at children at the age of 3 - 10 years. Note: (O) – Complicated current; (HO) – Not complicated current. Diagrammes of dispersion for indicators cellular and gumoral immunity in the complicated current the illnesses having authentic correlation communications ($p<0,05$) (Table 3).

Now often there is a HIV-infection and a tuberculosis

Treatment of these two infections is considered incompatible. But I have an application experience Ribavirin's at treatment of a tuberculosis at adult young faces. The preparation of a wide spectrum antivirus actions as Ribavirin can clean additional clinical signs of a tuberculosis, as a chronic rhinitis (reliability $p < 0, 001$). It testifies from volume, that combinations of preparations are possible, in particular, combination Sangviritrin + Ribavirin. Considering and that moment, that till 1 year at a HIV-positive of children the infection tends to self-healing.

However there is a considerable quantity of not studied questions to which it is necessary to carry interaction of various populations limfocits, stability of immunity, especially in the remote terms of supervision, an estimation of efficiency of the developed strategy in working out of medicines of new generation (Figure 8a-h).

Conclusions

1. To make inoculations till 3 years categorically it is impossible.
2. Until inoculations it is necessary to spend more careful inspection.
3. Persons with an immunodeficiency of inoculation should be counter-indicative not to rise generation of is frequent-ill children and not to cause resistance of sticks of Kokh.
4. At tuberculosis the secondary immunodeficiency develops, it is necessary to write it in diagnoses. Inoculations are counter-indicative to such patients any.
5. It is necessary to study and find more widely a medicine for a tuberculosis from phytoraw materials, because their action softer, than action of toxic pharmacotherapy from a combination of the preparations which efficiency is doubtful for today [30-33]. Besides, negative action on a liver, often crosses out advantage of treatment. There is one more moment: at slow acetilators (slow metabolisators medicines) - are shown kumulation chemiopraparations and the toxic effect is shown, therefore it is better to appoint preparations vegetative.

For today there is a Phytopreparation of a wide spectrum of action without occurrence of resistance of microflora Mycobacterium a tuberculosis to it - Sangviritrin which combines properties Immunomodulator's and an antibiotic. Besides, they can treat it is long without fear of occurrence of resistance. It is in parallel necessary to conduct search of new preparations.

For preventive maintenance and tuberculosis treatment the special diet is necessary

The success in treatment of this disease to no small degree depends on an immunity condition. One of ways it to support - an albuminous food. Eggs by all means should enter into a daily diet cottage cheese, dairy products, meat and fish, but not fried, a hen whom it is necessary to skin together with fat adjoining to it. Sausage products are excluded, as fats entering into them are badly acquired. Vegetables and fruit accelerate deducing of toxins. It is not obligatory to choose from them the most expensive. It is quite possible to manage cabbage, a beet, carrots, bunch greens, apples. Juice, a cranberry berry juice are rather useful. In the morning on an empty stomach it is desirable to eat a honey spoon, having washed down with its half of litre of water. It helps an organism to get rid of the harmful substances which have accumulated for night. At a tuberculosis all patient recommend to accept vitamin complexes (A.E.C) with minerals. As an additional source of vitamins of group B in food add wheaten bran. The mix of the crushed dried apricots and raisin with honey helps to compensate for the deficiency Kalium and other mineral substances. The onions, garlic are useful, garlick oil (contains Amicin), grapes (Amelotherapy) is even more effective.

Comb honey: It is recommended to chew it throughout several hours for 15-20 minutes with small breaks. It liquidates disease process.

Uterus the milk: Renders bacteriostatic and bactericidal action, ability to stop reproduction and growth of many bacteria and even to kill them.

But here that is surprising: the action Spectrum Uterus a milk is various - at its cultivation in a proportion 1:1000 uterus the milk detains growth of many bacteria.

Course of treatment: Sangviritrin tablets - to drink strictly in 20-40 minutes after meal. Course of treatment - 2-3 months. Such course has essential advantage before toxic therapy in 9 months the usual scheme the treatments consisting of 3-4 preparations.

Plants with antitubercular properties make the big group. It is desirable to adjust manufacture of phytopreparations.

At a tuberculosis of lungs it is necessary to eat pair milk, eggs, fat (dog, bear, pork), fruit, apples grated with honey, the guelder-rose with honey to drink fresh squeeze out juice. Tuberculosis treatment long also is necessary to support immunity. Grasses or gathering should be changed in 1-2 months, doing having rummaged 10-14 days, therefore it is impossible to stop on one recipe. In prisons it is necessary to contain in separate chambers of prisoners, to use bactericidal lamps in chambers (for processing of premises), there should be no a mental and physical violence.

6. Scheme: Sangviritrin + Immunofan (pricks) = racional!

7. Treatment of the HIV-infected patients with a tuberculosis, I consider, that should be individual. Disease needs to be treated and it is better herbal medicine. Children should appoint sparing therapy.

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