DILDORA BAHODIROVNA MIRZAKARIMOVA

PROTOZOOSIS AND HELMINTHOSIS OF THE REPUBLIC OF UZBEKISTAN

STUDY GUIDE

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DILDORA BAHODIROVNA MIRZAKARIMOVA

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TUTORIAL

on the topic:

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Prepared for "Therapeutic faculty" - 5510100

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This textbook is intended for clinical residents and master's students studying in medical institutes, and is reinforced with drawings, tables, instructions on the techniques of performing practical skills.

The textbook was discussed in the Central Methodological Board of ADTI and recommended to the Board of the Institute.

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Secretary of the University Council,
Associate Professor **N.A. Nasirdinova**

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PROTOZOOSIS AND HELMINTHOSIS
REPUBLIC OF UZBEKISTAN

ABSTRACT

- The manual contains information on modern aspects of parasitic diseases. A detailed description of the most common protozooses and helminthiases that make up the regional pathology of Uzbekistan is given. Possible ways of penetration into the human body, pathogenesis and clinical picture of diseases are outlined, the diagnostic value of laboratory verification methods is shown. The latest information about the methods of complex therapy of parasitosis is presented. The manual is intended for students of medical universities, general practitioners: pediatricians, therapists, infectious disease specialists, parasitologists.

INTRODUCTION

Parasitic diseases remain one of the most common types of pathology. To date, out of 1415 known human pathogens, 353 cause protozoal diseases and helminthiases. Expectations that most parasitic diseases would be under control by the end of the 20th century have not materialized. On the contrary, they continue to affect the population of many countries of the world, and primarily the inhabitants of developing countries located in tropical and subtropical climatic zones. In the industrialized countries of Europe, the situation has also deteriorated over the past two decades due to the growing importation of parasitic diseases from endemic countries.

The mass spread of parasitic diseases is recorded in all regions of the world. Parasitic infections in the world infect 4.3 billion people.

WHO expert assessment shows that helminthiases are in third place among all the most significant infectious and parasitic diseases in terms of the number of patients in the world. For comparison: the annual number of patients with influenza and SARS in the world is in sixth place. About 2 billion people of the planet are infected with helminths. In terms of economic damage to the health of

the world's population, intestinal helminthiases are among the 4 leading causes among all diseases (after diarrhea, tuberculosis and coronary heart disease). Morbidity and mortality in helminthiases in relation to infested persons is low. However, every year 135 thousand people die from helminthiases in the world.

The child population in the world accounts for 80-90% of all infested. Approximately 800 million children of preschool age suffer from helminthiases, while in 74% of cases a combination of two or more types is noted.

PARASITIC DISEASES

The actual parasitic diseases include:

- **protozoosis** caused by pathogenic unicellular organisms (protozoa);

- **helminthiases (helminthic infestations),** the causative agents of which are parasitic worms, or worms,

- **arachnoentomoses** - diseases caused by arthropods (sarcoptic mange, demodicosis, pediculosis, myiasis). Arachnoentomoses are not covered in this manual.

SECTION I. _ PROTOZOIAN DISEASES

GENERAL CHARACTERISTICS OF PROTOZOOSES

Protozoa, or Protists (*Protozoa, Protista*) in accordance with modern taxonomy represent the kingdom of unicellular eukaryotic organisms. The name "Protozoa" is now considered obsolete. Their body consists of one cell, functioning as a full-fledged independent organism due to the presence in it of special organelles (organelles), which perform separate functions corresponding to the functions and tissues of multicellular animals. The cell is bounded by an outer membrane. In most species, under the membrane there is a dense, elastic shell - the pellicle. Sometimes the pellicle is absent and its functions are performed by a denser homogeneous surface layer of the cytoplasm - ectoplasm, surrounding a more liquid and granular endoplasm. In a number of other unicellular species, in addition to the pellicle, a thicker outer shell is formed, which performs protective and supporting functions. The endoplasm contains the nucleus (or several nuclei), cellular organelles (ribosomes, endoplasmic reticulum, Golgi complex, mitochondria, etc.), as well as some special organelles and inclusions. Protozoan cells have sizes from 3 microns to 3 mm(average 50 - 150 microns). In most cases, the shape of their body is asymmetrical, some, having a denser skeletal structure, are built according to radial-beam, spiral or bilateral symmetry.

Protozoa reproduce by simple or multiple fission, as well as by budding or cyst formation. Some have a sexual process - *copulation* or *conjugation* . When two sexual individuals (gametes) copulate, they merge completely, including their nuclei, resulting in the formation of a zygote. During conjugation, which is observed in ciliates, two of their individuals exchange particles of the nucleus formed after its mitotic division, and, therefore, containing a haploid set of chromosomes. In some groups (for example, sporozoans), there is an alternation of generations that reproduce sexually and asexually (a life cycle with a change of generations).

The biological cycle of many unicellular organisms includes two life forms: an active or vegetative form called a *trophozoite* , and a dormant, resistant form called *a cyst* .

In humans, protozoa parasitize in the gastrointestinal tract, in the blood and tissues of various organs. They have both local and general pathogenic effects on the human body. Diagnosis of protozoal diseases is often based on the identification of pathogens by microscopic methods. Depending on the localization of the parasite, the material for research can be blood, sputum, feces, tissue samples (biopsy specimens), cerebrospinal fluid, etc. Sometimes they resort to culturing protozoa on special media or by passage on laboratory animals (biological test). In recent years, the range of immunological methods for diagnosing protozooses has expanded. In some parasitic diseases, the PCR method is used to identify parasite DNA in biological materials.

Treatment of some protozoal infections is hampered by the development of drug resistance in pathogens. A number of medicinal antiprotozoal drugs have side effects on the human body.

Protozoal diseases are widespread throughout the world, especially in the tropics and subtropics. They hurt millions of people. Malaria has the greatest socio-economic importance among protozoal diseases. In recent years, the incidence of opportunistic protozoal infections among HIV-infected individuals (toxoplasmosis, cryptosporidiosis, leishmaniasis) has increased.

Prevention of protozoal diseases consists mainly in the application of a set of measures aimed at strengthening sanitary supervision of environmental objects, food, water supply, vector control and the organization of chemoprophylaxis. Specific prophylaxis methods (vaccines) for protozoosis have not been developed, with the exception of leishmanization (see the *leishmaniasis section*). Several malaria vaccine prototypes are under testing.

AMEBIASIS

Amoebiasis (ICD10 - A06.0 - 9) is an anthroponotic invasion caused by *Entamoeba histolitica* , characterized by chronic recurrent colitis and extraintestinal manifestations .

Etiology. The causative agent is the dysenteric amoeba (*Entamoeba histolytica)*, which was first discovered in 1875 gr. military doctor, Privatdozent of the Military Medical Academy F.A. Lesh (1840 - 1903).

In the life cycle of a dysenteric amoeba, 3 stages are distinguished: a cyst, a small vegetative form (forma minuta) and a large vegetative form (forma magna).

The cyst is round in shape, has a size of 10-15 microns, is protected from the outside by a dense membrane, contains 4 nuclei inside and is an invasive stage.

The small vegetative form has a size of 15-20 microns, there is one core inside. This commensal form feeds on bacteria and lives in the upper colon (Fig. 1).

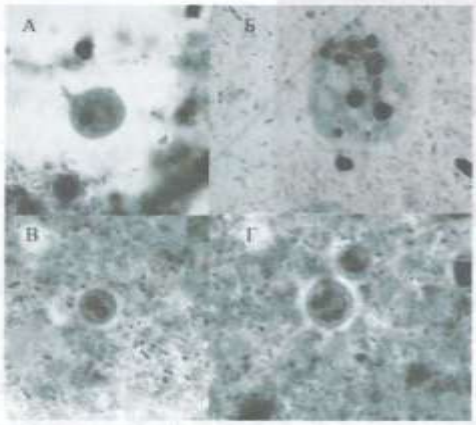

Figure 1. Entamoeba histolytica, Heidenhain stain. A – translucent form, B – tissue form with phagocytosed erythrocytes, C – binuclear cyst, D – quadrinuclear cyst.

The large vegetative form is an obligate luminal endoparasite 20–40 μm in size. Its cytoplasm is clearly divided into an outer light layer (ectoplasm) and an inner layer (endoplasm). Inside the endoplasm are digestive vacuoles with red blood cells. In the nucleus, when stained with iron hematoxylin, it can be seen that the karyosome occupies a central position, and clumps of chromatin are located radially, along the periphery. When moving, the amoeba forms a massive pseudopodia. This form of cysts does not form, but is able to transform into a small, translucent form.

Some experts also distinguish the tissue form of the amoeba. It differs in smaller sizes in comparison with a large vegetative form and is localized in the submucosal layer of the intestine. This form does not phagocytize erythrocytes and causes the development of extraintestinal lesions in amebiasis. (Fig. 2).

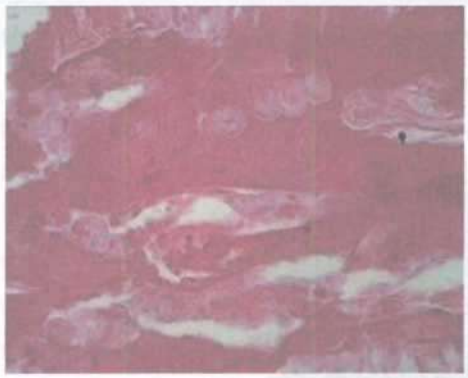

Figure 2. Entamoeba histolytica in the submucosal layer of the intestine. Hematoxylin-eosin staining.

Life cycle. The invasive stage of the parasite - a 4-nuclear cyst enters the human gastrointestinal tract with water or food. In the duodenum, a four-core amoeba emerges from the cyst, which divides and forms 8 small vegetative forms. They descend into the caecum, where they feed on bacteria and multiply. As the luminal forms of amoebas move through the pores of the large intestine, they become encysted and, together with feces, are excreted into the environment.

When the body's defenses are weakened, caused by hypothermia, hypovitaminosis, stress, chronic diseases, etc. some translucent forms (forma minuta) are transformed into hematophages (forma magna). At the same time, amoeba increase in size and begin to actively absorb red blood cells and lyse the intestinal epithelium. Penetrating into the submucosal layer, and then into the vessels, amoeba can enter the liver, lungs, brain and other organs through the portal vein system, forming abscesses in them.

Epidemiology. Amoebiasis is a ubiquitous invasion. About 10% of people in the world are infected with amoebas. The annual incidence of intestinal amebiasis is about 50 million cases, the mortality rate reaches 100,000 cases. Among all infectious diseases, amoebiasis, together with other diarrheal infections, ranks first in the world in terms of the frequency of deaths. The most unfavorable for amoebiasis are the countries of Southeast Asia, Africa, South and Central America. The regions of Georgia and Armenia, the republics of Central Asia (Tajikistan, Kyrgyzstan) are also considered endemic. Amoebiasis occurs sporadically in Uzbekistan. In countries with a temperate climate, sporadic incidence is also characteristic.

The wide spread of amoebiasis is facilitated by the low level of sanitary conditions, the low socio-economic development of the country, crowding of people and some features of cultural traditions.

Amebiasis is an anthroponotic disease. The source of infection is a person excreting *E. histolytica cysts in the faeces* . Mechanism transmission is fecal-oral. The intensity of the release of cysts is up to 580 thousand per 1 grfeces. One carrier (cyst excretor) can excrete tens of millions of cysts per day with feces.

Vegetative forms of dysenteric amoeba remain viable in feces for no more than 15-30 minutes. Cystic forms have significant resistance in the external environment. In faeces, they remain alive for up to 30 days, in the water of natural reservoirs - 9-60 days.

Factors transmission of amoebiasis can be soil, sewage, water from open reservoirs, household and industrial equipment, fruits, vegetables, food products, hands contaminated with dysenteric amoeba cysts.

The incidence is characterized by spring-summer seasonality. There are more carriers than sick people. In endemic areas, the ratio of patients to carriers is approximately 1:10.

natural susceptibility people to amoebiasis high. As with all other parasitoses, immunity is non-sterile. After recovery, specific antibodies in high titers are determined in the blood within 4-8 months, so the likelihood of repeated invasions is high.

Pathogenesis. The transformation of the luminal (commensal) form of the amoeba into a hematophage (parasitic form) serves as a starting point for the development of the infectious process. These amoebae are capable of producing trypsin, which destroys mucosal epithelial cells, as well as hyaluronidase, collagenase, and phospholipase-A, which destroy intestinal wall tissue down to the muscular layer, forming ulcers. Ulcers are irregularly shaped with undermined edges (the shape of a bottle with a narrow neck or flask), they are located on an unchanged background of the surrounding mucosa.

Perforation of ulcers leads to peritonitis. With the healing and scarring of deep ulcers, intestinal strictures can form, which cause the development of intestinal obstruction. A long, chronic course of amoebiasis leads to the development of amoeba - tumor-like formations in the intestinal wall. Ulcerative process leads to bleeding. Amoebas are able to penetrate into the vessels and spread throughout the body with the blood flow, and form amoebic abscesses in various organs.

Clinic. Currently, in accordance with the recommendations of the WHO expert committee (1997), it is customary to distinguish between:

- invasive amebiasis (penetration of amoebae into the intestinal mucosa and other organs);

- asymptomatic carriage (only commensal forms of amoebas live in the intestine).

According to the clinical course, they differ:

- intestinal amebiasis (amebic dysentery);
- extraintestinal amoebiasis.

Intestinal amebiasis is divided into the following forms:

- asymptomatic infection;
- amoebic dysentery;
- fulminant colitis with intestinal perforation;
- toxic megacolon;
- chronic amoebic colitis;
- amoeba;
- perianal ulceration.

In 90% of cases infection *E. histolytica* is accompanied by asymptomatic carriage.

Manifest intestinal amebiasis can occur in acute and chronic forms.

Acute intestinal amoebiasis . The incubation period lasts from several days to several months. The general condition and well-being in the initial period of the disease remain satisfactory: there is no fever, working capacity is maintained.

Initially, there is bloating and pain in the right iliac region. The chair is plentiful, mushy, 3-5 times a day with a small amount of mucus and blood. Then it becomes more frequent up to 15-20 times a day and becomes liquid, with a large amount of transparent vitreous mucus, which is saturated with blood, taking the form of "raspberry jelly". The sigmoid colon is spasmodic and palpable in the form of a compacted cord. Body temperature is often subfebrile.

In weakened people, against the background of pronounced symptoms of intoxication, severe forms of acute amoebic dysentery can develop: multiple acts of defecation, loose stools mixed with blood and mucus, the temperature rises to 39 ° C.

In intestinal amebiasis, reactive amoebic hepatitis is often observed. with a benign course, accompanied by periportal inflammation and an increase in the size of the liver. Rarely, fulminant amoebic colitis develops. as a result of perforation of the intestinal wall and the development of peritonitis, which can be fatal.

Without specific treatment, amoebiasis lasts for years (up to 10 years), acquiring a chronic form, which can occur in the form of recurrent (cases where remissions last from several weeks to several months, alternating with exacerbations lasting from 2 to 6 weeks) or continuous (without remissions), which is observed in patients with concomitant bacterial bowel diseases: salmonellosis, shigellosis, etc. With a continuous course of chronic amoebic dysentery , the ulcerative process spreads to the entire large intestine, anemia progresses, nutrition decreases, up to cachexia, hypopolyvitaminosis develops, and the functions of the digestive glands are disturbed. Complications may develop.

Extraintestinal amoebiasis accounts for about 10% of all cases of intestinal amoebiasis. It is manifested by the development of amoebic abscesses in any organ, more often in the right lobe of the liver. With a superficial abscess there are symptoms of irritation of the peritoneum, sometimes adhesions are formed with the diaphragm and surrounding organs.

The size of abscesses varies from barely visible to the eye to 10 cma diameter or more (Fig. 3).

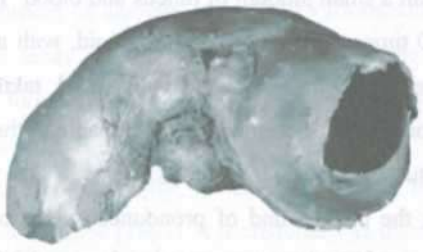

Figure 3. Giant amoebic abscess of the left lobe of the liver.

During the formation of a ***hepatic abscess*** , patients develop weakness, pain in the epigastric region or in the right hypochondrium, radiating to the right shoulder or shoulder blade. The size of the liver increases, soreness appears on palpation of its lower edge.

With the acute development of a hepatic abscess with hectic-type fever, repeated chills, signs of intoxication, after 1-2 weeks the disease may end in the death of the patient.

When a secondary microflora enters an abscess, it may suppurate and break through into the lungs and pericardium (through the diaphragm), into the abdominal cavity and even out through the anterior abdominal wall with the formation of a skin-hepatic fistula.

Abscesses may occur in the lungs (sometimes with the development of pleural empyema with peribronchial fistula), rarely in spleen and brain.

Amebiasis of the skin develops as a secondary process in debilitated and debilitated patients. Erosions and ulcers are localized mainly in the perianal region, perineum, buttocks.

Cases of ***urogenital amebiasis are described*** , which usually develops in women, due to the direct entry of pathogens through the ulcerated surface of the rectal mucosa into the genitals.

Complications. ***Perforation of the intestinal wall with the development of peritonitis*** is characteristic of severe forms of the disease and is manifested by the clinic of "acute abdomen". Most often, it develops during the height of the disease and is noted in 2-6% of cases. This complication is the most formidable and causes high mortality (20-45% of the total number of deaths from amoebiasis).

Some patients develop ***adhesive fibrinous peritonitis*** . in the place of localization of deep ulcers of the intestinal wall. The main clinical sign is a painful infiltrate 3 15 cmin diameter, accompanied by fever, local muscle tension of the anterior abdominal wall, neutrophilic leukocytosis. This complication does not require surgical intervention and responds well to antiparasitic therapy.

Amoebic appendicitis can occur in the form of acute or chronic appendicitis. The defeat of the appendix is associated with the penetration of the pathogen into it from the caecum. Surgical intervention can lead to generalization of invasion.

Intestinal obstruction develops due to the formation of cicatricial strictures of the colon. At the same time, patients have a clinic of low dynamic intestinal obstruction with a typical pain syndrome. On palpation, a painful dense infiltrate is determined, bloating and asymmetry of the abdomen are noted.

Amoeba (amoebic tumor) is a rare complication. It develops in individuals who have not received specific therapy. Palpation of the abdomen reveals a tumor-like formation. With amoeba, the development of intestinal obstruction is possible. The complication usually does not require surgical treatment and responds well to anti-amoebic therapy.

Other complications - intestinal bleeding, colonic polyposis, prolapse of the rectal mucosa - are rare.

For extraintestinal amoebiasis the most formidable complication is perforation of the ***amoebic abscess,*** which the registered in 10-20% of cases of amoebic liver damage, and is accompanied by 50-60% mortality.

Diagnostics. The diagnosis can be considered established when pathogenic forms of dysenteric amoeba, hematophagous (forma magna). The material is viewed no later than 15-20 minutes after its receipt. If it is impossible to conduct a study immediately, the material is placed in a special preservative (Turdyev, Safaraliyev, Burrows, etc.).

If the results of the study are negative in patients with the corresponding epidemiological history and clinical picture, the study is repeated up to 5-6 times a day. To stimulate the excretion of amoebas, a saline laxative is sometimes prescribed.

Instrumental research methods are essential in diagnostics . Rektoromanoscopy and fibrocolonoscopy allow to detect characteristic ulcers of the intestinal mucosa with undermined edges, surrounded by a zone of hyperemia.

Radiography, computed tomography and ultrasound can identify amoebic abscesses, determine their location, shape and size.

Serological methods. The informativeness of serological tests based on EIA for intestinal amebiasis is extremely low and amounts to 65-70 % , with extraintestinal it rises to 95% or more. The determined level of antibodies does not correlate with either the extent of the lesions or the severity of the course of the disease.

Control of the effectiveness of therapy can be assessed by the results of serological studies in paired sera. In those who recovered after 6-12 months, antibody titers gradually decrease, reaching the average population values.

Molecular biological method: PCR makes it possible to identify DNA nucleotide sequences in faeces and contents of abscesses *E. histolytica* and conduct differential diagnosis with a non- pathogenic counterpart of the dysenteric amoeba - *E. dispar* .

In the general blood test , hypochromic anemia, leukocytosis with a stab shift, and increased ESR are often determined .

Differential diagnosis of intestinal amoebiasis should be carried out with other infectious and parasitic diseases: shigellosis, balantidiasis, intestinal schistosomiasis, trichuriasis, hymenolepiasis strongyloidiasis, campylobacteriosis and non-infectious diseases: ulcerative colitis, Crohn's disease, diverticular disease and neoplasms in the colon.

amoebic Liver abscesses are most often differentiated from liver echinococcosis, hepatocarcinoma, and bacterial abscesses.

Treatment of patients with amebiasis should be carried out in a hospital, taking into account the possibility of complications and the need for clinical and laboratory monitoring of the effectiveness of therapy.

For the sanitation of decreed contingents with non-invasive amoebiasis (asymptomatic "carriers"), translucent amebocides are used. ***Etofamide*** is prescribed for adults at 20 mg / kg / day in 2 doses for 5-7 days or ***paromomycin*** - 1.0 / day in 2 doses for a course of 5-10 days

Systemic tissue amebocides are used to treat invasive amoebiasis.

metronidazole, orally 30 mg / kg / day in 3 divided doses. The course is 8-10 days.

Tinidazole, up to 12 years - 50 mg / kg / day (max. 2 gr) in 1 dose for 3 days; over 12 years old - 2 g / day in 1 dose for 3 days.

Ornidazole, up to 12 years - 40 mg / kg / day (max. 2 gr) in 2 divided doses for 3 days; over 12 years old - 2 g / day in 2 divided doses for 3 days.

Seknidazole, up to 12 years - 30 mg / kg / day (max. 2 gr) in 1 dose for 3 days; over 12 years old - 2 g / day in 1 dose for 3 days.

After successful chemotherapy of a liver abscess, residual cavities usually disappear within 2–4 months, but cavities may persist for up to 1 year.

R convalescents are discharged after the end of the course of treatment with complete clinical recovery and complete sanitation of the intestine from the pathogen, which is established by a 3-fold study of feces. According to fibrocolonoscopy or sigmoidoscopy, the condition of the colon mucosa is assessed.

Forecast. With early diagnosis and timely treatment, a full recovery is possible. Without treatment, mortality is 5-10%, with the development of extraintestinal complications - 50%. With treatment, improvement usually occurs within a few days. In some patients, symptoms of colonic irritation persist for several weeks after successful treatment. Relapses are possible.

Prevention is carried out in accordance with the rules for intestinal infections:

- compliance with hygiene rules;
- drinking only filtered and boiled water, bottled drinks;
- obligatory washing of fruits and vegetables before use with safe water;
- examination of feces for amoebiasis during routine bacteriological examinations of workers in the water supply and nutrition system. In case of detection of cysts or luminal forms of dysenteric amoeba, asymptomatic carriers are sanitized with direct-acting amoebicides;

TOXOPLASMOSIS

Toxoplasmosis (ICD10 - B58) is a natural focal zoonotic disease caused by toxoplasma, characterized by a long course and frequent damage to the central nervous system and eyes.

The causative agent of toxoplasmosis was first discovered in 1908 by Nicole and Manso in African rodents - gondii (*Ctenodactylus gondii*).

Etiology. *Toxoplasma gondii* is an obligate intracellular parasite and exists in three forms: tachyzoite (endozoite or trophozoite), bradyzoite (cystozoite), and oocyst (Figure 4).

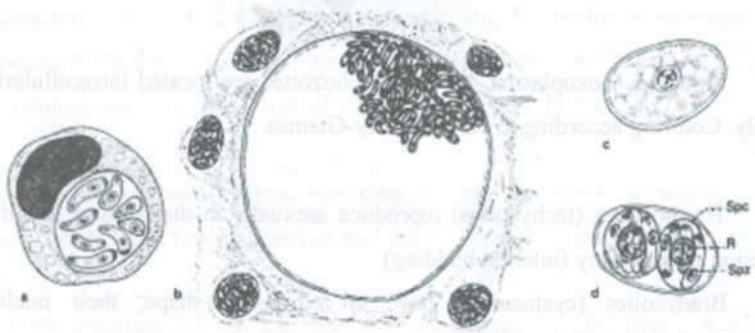

Figure 4. Toxoplasma gondii: a – trophozoites (tachozoites) in a monocyte; b – pseudocyst with bradyzoites in the brain tissue (the contents are only partially shown), c – immature oocyst from fresh cat feces; d – sporulated invasive oocyst; r is the residual body; Sps, sporocyst; Spz, sporozont (according to many authors).

Trophozoites (Greek toxon - arch, arc) in the form of a crescent or orange slices have dimensions of 4-7 x 2-4 microns. (Fig. 5).

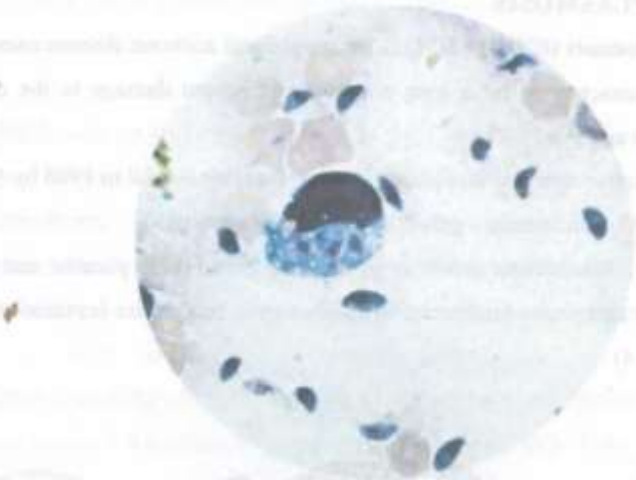

Figure 5. Toxoplasma gondii. Trophozoites are located intracellularly and freely. Coloring according to Romanovsky-Giemsa.

Trophozoites (tachyzoites) reproduce asexually in the form of longitudinal division or endogony (internal budding).

Bradyzoites (cystozoites) have an elongated shape, their nuclei are significantly displaced towards the posterior end of the body. Parasites are inside the cells, a dense shell forms around. These formations, called pseudocysts, are 100 microns or larger. Bradyzoites (cystozoites) are the main form of existence of Toxoplasma in the body of intermediate hosts.

Oocysts have a round-oval shape, their dimensions are 9-11 × 10-14 microns. Each mature oocyst contains 2 sporocysts. Each sporocyst contains 4 sporozoites. Oocysts are formed as a result of the sexual process occurring in the intestinal epithelial cells of the definitive hosts (the feline family).

The life cycle of Toxoplasma includes the stages of merogony, gametogony and sporogony (Fig. 6).

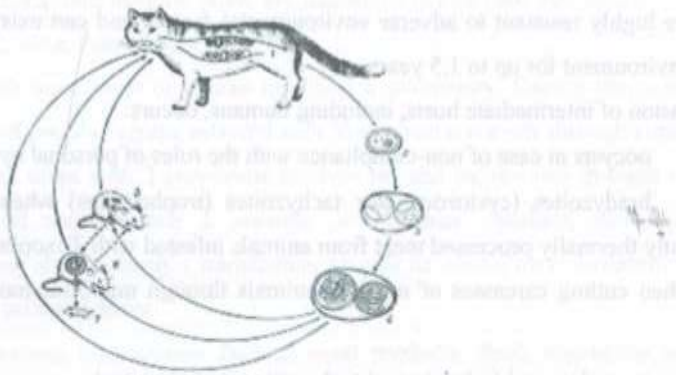

Figure 6. Scheme of the life cycle of toxoplasma: 1 - stages of development in the intestines of a cat; 2-4 - Toxoplasma oocysts; 5 - proliferative stages of development in the mouse body; 6 - toxoplasmic cyst in the brain of a mouse; 7 - a newborn mouse infected transplacentally (according to I.K. Frenkel, 1970).

The definitive hosts, feline mammals, become infected by eating meat containing bradyzoites (cystozoites) or tachyzoites (trophozoites), or by ingesting oocysts.

In the intestine of the cat, the cyst membrane breaks down, and the cystozoites penetrate the enterocytes, where they multiply by schizogony. As a result, merozoites are formed. Part of the merozoites, penetrating into enterocytes, gives rise to male and female germ cells, which, after transformation, exit the enterocytes into the intestinal lumen. Male cells (microgametocytes) divide many times to form microgametes. Macrogametes are formed from female cells (macrogametocytes). As a result of the fusion of micro- and macrogametes, a zygote is formed. The zygote is covered with a protective membrane, and an oocyst is formed, which enters the external environment with feces.

Sporogony (oocyst maturation) occurs in the external environment at a temperature of 4-37°C and sufficient humidity and lasts from 2 days to 3 weeks. The oocyst produces two sporocysts, each containing four sporozoites. Mature

oocysts are highly resistant to adverse environmental factors and can exist in the external environment for up to 1.5 years.

Invasion of intermediate hosts, including humans, occurs:

- oocysts in case of non-compliance with the rules of personal hygiene;
- bradyzoites (cystozoites) or tachyzoites (trophozoites) when eating insufficiently thermally processed meat from animals infested with Toxoplasma, as well as when cutting carcasses of infected animals through microtraumas of the skin;
- transplacental (intrauterine) tachyzoites (trophozoites).

When sporulated oocysts are ingested in the upper intestine, sporozoites are released from them, which are introduced into epithelial cells. Inside them, they grow, divide by endodiogenesis, forming rapidly multiplying tachyzoites. Most often, accumulations of Toxoplasma, including 10-30 or more tachyzoites, are formed in the cells of the lymph nodes, liver and lungs (macrophages). The membrane around them is the membrane of the parasitophoric vacuole. Such accumulations of generation parasites were previously often called pseudocysts.

As a result of the accumulation of parasites, the cell is destroyed and the tachyzoites released at the same time are introduced into neighboring cells, and are also carried hematogenously and lymphogenously to various organs. Proliferative forms of parasites (tachyzoites) rapidly accumulate in the host organism.

As the immune response develops, the number of Toxoplasma (tachyzoites) circulating in the blood decreases. In the future, they completely disappear from the blood, penetrating into the cells, where they are transformed into slowly multiplying bradyzoites (cystozoites). Their accumulations contain up to 10 thousand parasites and are surrounded by a dense shell. These intracellular cysts (pseudocysts) can be up to 200 μm in size. They are localized mainly in the brain and skeletal muscles. The formation of cysts begins 9 days after infection. Cystozoites in cysts remain viable for decades.

Epidemiology. The definitive hosts of *T. gondii* are representatives of the cat family (*Felidae*), excreting oocysts of pathogens with faeces into the external

environment, intermediate hosts are mammals (more than 300 species, including humans), more than 60 species of birds.

The main route of human infection is alimentary. During life, a significant number of people become infected with Toxoplasma oocysts through contaminated food, less often with Toxoplasma bradyzoites and tachyzoites through raw meat. The blood contact route is possible (in veterinary workers, workers in meat processing shops), rarely - transfusion, as well as during transplantation of organs from an infected donor.

Leading transmission factors: meat products, fresh vegetables and fruits, unpasteurized milk and dairy products, etc. Young children are easily infected by contact with soil and sand contaminated with cat feces.

The second route of transmission is transplacental. Transplacental damage to the fetus can occur when the mother is infected during pregnancy. The risk of transmission increases dramatically with increasing gestational age (from 6% at 13 weeks' gestation to 72% at 36 weeks' gestation).

Pathogenesis. In the infectious process of toxoplasmosis, two stages are distinguished: acute and chronic (persistence). The gates of infection are the digestive organs. The introduction of the pathogen occurs in the small intestine, mainly in its lower sections, where inflammatory changes occur.

Pathogens penetrate into the mesenteric lymph nodes, resulting in the development of specific mesadenitis. Overcoming this barrier, Toxoplasma disseminates (hematogenously and lymphogenously) and invades target cells (brain, striated muscles, liver, etc.). In these organs, local inflammatory reactions develop and specific granulomas form. Active proliferation of Toxoplasma is accompanied by the release of various toxins and allergens, leading to delayed-type hypersensitivity.

With an adequate immune response, most trophozoites (up to 90%) die, and the rest, having invaded cells, transform into bradyzoites and form cysts. At this stage, the ability of the immune system to recognize "friend or foe" is somewhat reduced due to changes in the composition of surface antigens in bradyzoites.

Bradyzoites slowly multiply inside the cysts and continuously secrete immunogenic metabolites against which specific antibodies are produced. The formation of specific immunity leads to the attenuation of the acute stage of the development of the disease (the dissemination of toxoplasmosis stops) and toxoplasmosis passes into the *chronic stage,* which in most cases proceeds latently.

Allergic restructuring of the body is of great importance in the pathogenesis of invasion.

Toxoplasmosis is an AIDS marker infection indicating the presence of cellular immunodeficiency. In persons with impaired immunity (HIV infection, long-term use of immunosuppressants, etc.), toxoplasmosis acquires a malignant course. As a result of constantly growing parasitemia, a specific "parasitic sepsis" or necrotizing meningoencephalitis can develop, often with a fatal outcome.

Congenital toxoplasmosis is the result of transplacental transmission of the infection to the fetus. In acute toxoplasmosis during pregnancy, as a result of inflammatory changes in the placenta and a violation of its barrier function, tachyzoites affect the myometrium, allantoid mesenchyme, followed by damage to the tissues of the embryo. All this can lead to both intrauterine death of the fetus (when infected in the early stages of pregnancy), and the birth of a child with various clinical forms of congenital toxoplasmosis (when infected in the later stages of pregnancy).

Clinic. The incubation period lasts from 5 to 23 days (average about 2 weeks). The acute stage of acquired toxoplasmosis in persons without immunodeficiency proceeds, as a rule, in a latent form. Clinically, the disease often does not manifest itself.

The manifest form of the acute stage of acquired toxoplasmosis is rare and is characterized by a gradual onset, the development of cervical or axillary lymphadenitis, and a moderately pronounced syndrome of general intoxication. Sometimes the disease begins with chills, fever above 38.5 ° C, headaches, myalgia, manifestations of acute enteritis or gastroenteritis with diarrhea.

By the end of the first week of illness, the liver and spleen are enlarged. Acute chorioretinitis develops in 20% of patients and is manifested by a sudden "loss" of part of the visual field. Relative lymphocytosis is revealed in the peripheral blood, in some cases - single atypical mononuclear cells.

From the second week of the disease, the manifestations of general intoxication significantly decrease, the diarrheal syndrome disappears, signs of damage to the musculoskeletal system, cardiovascular system, and organs of the reticuloendothelial system progress.

In the third or fourth week, in most cases, the syndrome of general infectious intoxication stops, arthralgia and myalgia become much less pronounced, mononuclear cells disappear from the peripheral blood. However, patients remain lymphadenopathy and hepatolienal syndrome.

Allocate lymphadenopathic, visceral, cerebral and ocular forms of toxoplasmosis.

In immunocompetent individuals, the acute stage in the overwhelming majority of cases (95-99%) passes into the latent form of the chronic stage, which is characterized by the formation of non-sterile immunity in patients with lifelong persistence of toxoplasma in the body (in the form of bradyzoites). In patients, organ pathology is not detected; specific IgG in "floating" concentrations are determined in the blood serum.

The manifest form of the chronic stage of toxoplasmosis develops under the influence of various immunosuppressive factors and is characterized by the presence of periods of exacerbations (clinically expressed and erased) and remissions.

For the period of exacerbation, signs of lymphadenopathy (occur in all patients), lesions of the central nervous system and the cardiovascular system, manifestations of general intoxication, hepatolienal syndrome, and dysfunction of the autonomic nervous system are characteristic.

Calcifications in the substance of the brain (especially in the vascular plexuses) are detected in a third of patients, foci of chorioretinitis - in 25% of

cases. Indicators of a general clinical blood test are characterized by a tendency to leukopenia, relative lymphocytosis. In such patients with inadequate therapy, periods of exacerbations (lasting from 1 to 4 months) alternate with remissions (from 8 months to 1.5 years).

When a woman is infected during pregnancy, a child is born with congenital toxoplasmosis (as a result of transplacental transmission of pathogens). Congenital toxoplasmosis can occur in acute and chronic (latent or overt) forms. The frequency of transplacental transmission of Toxoplasma is 1 case per 1000-3500 births.

About 30% of children infected in utero have clinically pronounced toxoplasmosis at birth. In other cases , these are asymptomatic and erased forms that give late clinical manifestations from any moment after birth, as a rule, after months and years. Clinical manifestations depend on the time of intrauterine infection of the fetus.

The stages of the disease change one another. Initially, signs of a generalization of the process are observed, then meningoencephalitis develops, after which postencephalic disorders remain.

The manifest form of the acute stage of congenital toxoplasmosis is manifested by fever, severe intoxication, symptoms of encephalitis, enlargement of the liver, spleen, pneumonia, jaundice, maculopapular rash and eye damage (central chorioretinitis).

If the acute stage of congenital toxoplasmosis occurs in utero and ends with the birth of a child, then the newborn has hydrocephalus, microcephaly, oligophrenia, paresis, paralysis, epilepsy, damage to the organ of vision (microophthalmos, chorioretinitis, blindness, etc.). After the infectious process subsides, persistent irreversible changes may persist: microcephaly, defects in the organ of vision, lag in psychomotor development.

People with AIDS tend to develop the septic form of the disease (regardless of stage). It is characterized by a severe course and the development of life-threatening complications. The CNS is most commonly affected. Diffuse

encephalitis or meningoencephalitis develops. In the cerebrospinal fluid, mononuclear pleocytosis with an increase in protein content is observed. Other organs may also be involved in the process - lungs, heart, liver, pancreas.

Septic forms of toxoplasmosis can develop in diseases requiring long-term and / or massive immunosuppressive therapy (oncological, collagenosis, organ transplantation). These patients also develop generalized forms of toxoplasmosis, which, in the absence of etiotropic treatment, quickly lead to death.

Diagnostics. Currently, the leading role in the diagnosis of toxoplasmosis belongs to laboratory research methods.

Based on the results of ***serological studies*** (EIA), it is possible to determine the fact that a given patient is infected with Toxoplasma (the presence of specific IgG). The detection of IgM indicates an acute stage of both acquired and congenital toxoplasmosis. Determining the concentrations of specific antibodies of other classes (IgA, IgE, IgD), as well as the avidity of antibodies, does not have significant diagnostic advantages. The absence of specific IgM and IgG in the patient makes it possible to exclude toxoplasmosis.

The latent form of chronic toxoplasmosis is diagnosed when only specific IgG is present in the patient's blood.

If congenital toxoplasmosis is suspected, cord blood is examined to determine specific IgM and IgG. The absence of antibodies excludes the diagnosis of congenital toxoplasmosis, the presence of IgM confirms it, the detection of only IgG requires a second study after 4-6 weeks (the concentration of maternal antibodies should decrease by at least 50%). The absence of a decrease in IgG allows you to confirm the diagnosis.

differential diagnosis. It is carried out in the acute stage of toxoplasmosis with infectious mononucleosis, cygomegalovirus infection, tuberculosis, tularemia, acute leukemia, lymphogranulomatosis.

In cases where the leading symptoms in the clinic are the symptoms of damage to the nervous system, it should be differentiated from meningoencephalitis of another etiology, organic lesions of the central nervous

system (tumors, hemorrhages, etc.). Fetal lesions in toxoplasmosis are similar to lesions in cytomegalovirus infection, rubella, herpes infection, syphilis, listeriosis.

In the differential diagnosis of exacerbations of the chronic stage of toxoplasmosis, it is necessary to exclude the initial manifestations of HIV infection , systemic connective tissue diseases, blood diseases, tuberculosis, focal infection (chronic tonsillitis, cholecystitis, cholangitis, appendicitis), chlamydia, mycoplasmosis.

Treatment. Patients with documented acute toxoplasmosis, regardless of the presence and severity of clinical manifestations, are subject to unconditional etiotropic treatment.

In adults, the regimen of choice is a combination of ***doxycycline*** given orally at 0.1 every 12 hours and ***metronidazole*** (0.25 every 8 hours). The course of treatment is 10 days. Children can be given oral ***spiramycin*** every 8 hours for the same period (adjusted for age). At the same time, antihistamines and desensitizing agents are prescribed in the usual dosages.

Treatment of acute toxoplasmosis in pregnant women is carried out in order to prevent congenital toxoplasmosis. Recommended ***spiramycin*** 1.5-3 million units orally 3 r / day. within 2-4 weeks. (after 12 weeks of gestation). Alternatively: use of pyrimethamine in combination with short-acting sulfa drugs or clindamycin.

In cases of proven congenital toxoplasmosis, treatment is with a combination of ***sulfadiazine*** (50 mg/kg orally every 12 hours), ***pyrimethamine*** (2 mg/kg/day orally for the first two days, then 1 mg/kg/day for 2-6 months, then 1 mg/kg/day 3 times a week) and ***folinic acid*** (orally 10 mg 3 times a week). The total duration of the course is at least 12 months. The volume of pathogenetic and symptomatic therapy is determined by the leading syndromes (cerebral edema, Infectious-toxic shock, respiratory, heart failure, etc.).

In the period of exacerbation of chronic toxoplasmosis, individual-oriented therapy acquires leading importance; long-term antiprotozoal therapy is pathogenetically unfounded.

Prevention. Limiting contact with infected cats, observing the rules of personal hygiene. The prohibition of the use (tasting) of raw minced meat, as well as meat dishes without sufficient heat treatment. Prevention of infection of women during pregnancy (avoid contact with cats and testing raw minced meat, wash hands after preparing raw meat dishes, etc.). Preventive measures in the outbreak are not carried out. Specific prophylaxis in humans has not been developed.

MALARIA

Malaria (code according to ICD10 - B50-54) is a group of anthroponotic protozoan vector-borne human diseases, the pathogens of which are transmitted by mosquitoes of the genus *Anopheles* . It is characterized by a predominant lesion of the reticulohistiocytic system and erythrocytes, manifested by recurrent febrile paroxysms, anemia and hepatosplenomegaly.

Etiology. There are four types of Plasmodium that cause malaria in humans:

- ***Plasmodium viva x*** - the causative agent of three-day malaria;
- ***Plasmodium ovale*** - the causative agent of three-day malaria (or ovale-malaria);
- ***Plasmodium falciparum*** - the causative agent of tropical malaria;
- ***Plasmodium malariae*** is the causative agent of four-day malaria.

Malaria has been known since ancient times. It is mainly distributed in countries with a tropical climate, but can also be recorded in temperate latitudes.

Life cycle. The development of plasmodia (fig. 7) occurs with a change of hosts and with alternation of forms of parasites that reproduce sexually in the body of the final host - a mosquito of the genus *Anopheles* and asexually (schizogony) in the body of an intermediate host - a human. Asexual reproduction includes tissue and erythrocyte schizogony .

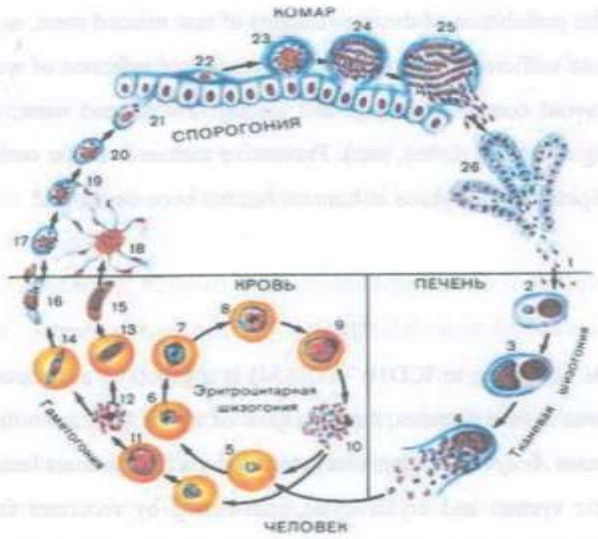

Figure 7. Development cycle of *Plasmodium falciparum* (according to D.E. Genis, 1985). 1 – sporozoites from mosquito salivary glands; 2-4 - tissue schizogony in hepatocytes; 5-10 - stages of erythrocyte schizogony; 11-16 - stages of formation of gamonts; 17 - macrogamete; 18 - exflagellation of microgametes; 19-21 - ookineta; 22-25 - stages of oocyst development; 26 - accumulation of sporozoites in the salivary glands.

The causative agents of malaria enter the body of the intermediate host (human) at the sporozoite stage with saliva when bitten by an infected female malarial mosquito. The sporozoite is a spindle-shaped cell with one nucleus. After 15-45 minutes with the blood flow, they enter the liver and are actively introduced into hepatocytes. From this moment, the **tissue stage** of development of plasmodia begins. In hepatocytes, sporozoites turn into tissue schizonts, which divide by schizogony. In this case, multiple division of the nucleus occurs, and then a section of the cytoplasm is isolated around each nucleus. As a result of schizogony, a large number of tissue merozoites are formed. There are 2 types of sporozoites in the P.

vivax and *P. ovale* populations : tachysporozoites and bradysporozoites. Rapidly developing tachysporozoites immediately start dividing in hepatocytes and then, getting into the blood, cause disease. Slowly developing bradysporozoites (hypnozoites) begin to multiply after 6-10 months and later, causing the development of late relapses of malaria.

After the destruction of the affected hepatocytes, merozoites enter the bloodstream. Some of them actively penetrate into the erythrocytes, where they grow and reproduce (erythrocyte schizogony). The remaining parasites die as a result of interaction with nonspecific resistance factors of the organism.

The erythrocyte schizogony of malarial plasmodia includes several successive stages of development:

The young trophozoite is the initial stage, which differs from the merozoite in its larger size and the presence of a central vacuole, which gives the parasite the shape of a ring or ring.

The developing trophozoite is the growing stage of the parasite. The nucleus and cytoplasm gradually increase in size, the central vacuole contracts, and grains of malarial pigment appear, which is a product of hemoglobin metabolism.

Mature trophozoite is the stage of preparation for nuclear division. The nucleus is large, the cytoplasm occupies most of the erythrocyte, the central vacuole is weakly expressed or absent, the pigment is clearly visible.

The developing schizont is characterized by an increasing number of nuclei, pigment grains are gradually concentrated into separate clusters, more often one.

A mature schizont is an accumulation of individual nuclei, around which areas of fragmented cytoplasm are isolated. This process is called merulation - the formation of daughter parasitic cells (merozoites), which are located inside the erythrocyte in a certain way, characteristic of each type of plasmodium. Between the merozoites there remains a bunch of pigment, the amount and location of which is also specific for a particular type of pathogen.

After the destruction of the erythrocyte membrane, merozoites are released into the blood plasma. A significant part of them die as a result of interaction with

the host's immune factors, while the rest are actively re-introduced into erythrocytes, and the cycle of erythrocyte schizogony is repeated.

The duration of this cycle is determined by the type of Plasmodium and is 48 hours for pathogens of three-day and tropical malaria, and 72 hours for pathogens of four-day malaria. The completion of each subsequent cycle of erythrocyte development leads to a progressive increase in the number of parasites circulating in the blood.

Some merozoites change the direction of their development and turn into immature germ cells - gametocytes (gamonts), which, in the process of further differentiation, form microgametocytes (male germ cells) and macrogametocytes (female germ cells). Female gametocytes reach the functional maturity of development in erythrocytes, male - when they enter the stomach of a mosquito.

With three-day, ovale and four-day malaria, gametocytes appear in the blood of patients already from the first cycles of erythrocyte schizogony, i.e. from the first day of illness. With tropical malaria, gametocytes appear in the peripheral blood only by the 7th-10th day of illness and can persist for a long time in the blood of those who have been ill (up to 4-8 weeks or even more).

Gametocytes enter the body of the female malarial mosquito when it feeds on the blood of an infected person. In the stomach of a mosquito, male microgametocytes are divided into several parts (exflagellation), forming 6-8 mobile microgametes (Fig. 8).

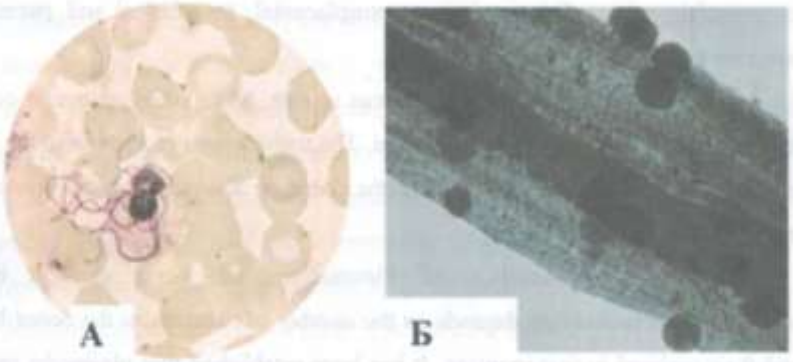

Figure 8. Stages of development of malarial plasmdia in a mosquito. A - exflagellation of male gamonts in the stomach of a mosquito. Coloring according to Romanovsky - Giemsa. B - oocysts on the walls of the stomach of a female malarial mosquito.

Female germ cells are fertilized by male ones, resulting in the formation of mobile zygotes - ookinetes. They penetrate into the submucosal layer of the mosquito's stomach, where they encapsulate and form oocysts, inside which many sporozoites are formed during sporogony .

After maturation, the oocysts are destroyed, and the sporozoites released from them accumulate in the salivary glands of the female mosquito. After that, it becomes dangerous for humans. When bitten with saliva, sporozoites enter the wound, and the cycle begins anew.

Epidemiology. Currently, the disease is widespread in Africa, the Middle East, Southeast Asia, and Latin America. Of the 180 countries, 90 are endemic for malaria. According to WHO, the number of deaths from malaria in the world annually is about 400 million, of which more than 70% occur in children under the age of 5 years. About 1.2 billion people around the world live in high-risk areas for malaria. WHO implements several programs to combat malaria. Since 1998, the WHO has been running the Roll Back Malaria program, since 2011 - the Global

Malaria Program. Malaria is a transmissible infection. In addition to the main, transmissible route of transmission, transplacental, transfusion and parenteral infection are also possible.

Transplacental infection of the fetus is rare. More often, infection occurs during childbirth with placental abruption. The transmission of malaria to the fetus is realized only under the condition of the complete absence of immunity to this infection in the mother.

Transfusion transmission of Plasmodium can occur through blood transfusion. Its probability depends on the number of parasites in the donor blood and their survival in preservatives. It has been established that plasmodia remain viable in the blood when stored in a refrigerator (at 4°C) for up to 2 weeks.

Parenteral contamination can occur when asepsis rules are violated and non-disinfected medical instruments are used. This route of transmission of malarial plasmodia is especially relevant among people who use drugs.

The source of infection is a sick person or parasite carrier, in whose blood there are mature sexual forms of plasmodia (gametocytes). Gametocytes of *P. vivax* , *P. ovale* , and *P. malariae* appear in the blood during the first days of illness, and their number increases after several cycles of erythrocyte schizogony. Patients with tropical malaria in the first days of the disease are not a source of infection, since gametocytes begin to be detected in their peripheral blood only from the 9-10th day of the disease. At 2-3 weeks of illness, their number increases significantly. Without specific treatment, germ cells can remain in the patient's body for a long time (up to 1.5 months).

In highly endemic areas, children of the local population aged 6 months to 5 years are the most active sources of infection. Their lack of specific immunity contributes to the emergence of a high level of parasitemia and intensive infection of vectors. At the same time, the disease in children occurs mainly in an atypical form, which is the reason for its late diagnosis.

Among the local population of hyperendemic regions, a significant number of asymptomatic parasite carriers are registered. Therefore, these individuals can serve as sources of infection for a long time.

In non-endemic areas, persons who have arrived from endemic areas and become infected with malaria can serve as sources of infection.

In countries with a temperate climate, the incidence is characterized by a pronounced seasonality with an increase in the summer-autumn months. In countries with a tropical climate, malaria infection occurs all year round.

Post-infectious immunity in malaria is unstable, short-term, species-specific. Most types of malaria are benign, but tropical malaria is fatal in 0.5-7% of cases.

Pathogenesis. The main pathogenetic mechanism in malaria is associated with the massive decay of affected erythrocytes and with the toxic-allergic effect of plasmodia metabolic products that enter the blood plasma. As a result, the patient develops a characteristic malarial attack.

In the first days of the disease (from 1 to 3 days for three-day and oval malaria and up to 7-10 days for tropical malaria), patients experience fever of the wrong type (initial fever), which is associated with the presence of several subpopulations of parasites at different stages. of its development. Then a dominant parasite subpopulation forms and the fever becomes relapsing (every other day in tropical, vivax, and oval malaria; two days later in four-day malaria).

Erythrocytes affected by *P. falciparum* adhere to the endothelial cells of the capillaries of the internal organs, which underlies the development of the malignant course of tropical malaria. The accumulation of such erythrocytes in the capillaries of the central nervous system leads to the development of malarial coma as a result of thrombosis, hemorrhages and cerebral edema. An important component in the development of this pathological process is a metabolic disorder, which is manifested by hypoglycemia. The presence of affected erythrocytes in the capillaries of the lungs leads to the development of symptoms of bronchitis and bronchopneumonia, in the capillaries of the intestine - to diarrhea and other signs of enteritis or etrerocolitis. Kidney damage is more common in four-day malaria

and is associated with deposits of soluble malarial immune complexes on the basement membrane of Malpighian bodies, resulting in membranous nephropathy with nephrotic syndrome.

The characteristic signs of malaria are hepato- and splenomegaly, which develop as a result of acute blood filling and a significant increase in the reaction of the reticuloendothelial system (RES) to the decay products of erythrocytes and plasmodium toxins. The enlargement of the spleen with three-day malaria is so fast that microtears form on its capsule, which are subsequently replaced by connective tissue, which makes the capsule of this organ fragile.

The degree of development of anemia due to the destruction of erythrocytes by plasmodia depends on the level of parasitemia and the duration of the disease, and often develops in tropical malaria, the plasmodia of which is able to infect both young and mature erythrocytes, in contrast to P. vivax and P. ovale, which parasitize in only in young erythrocytes.

Clinic. During malaria, the following periods are distinguished:

1) incubation period; 2) the period of primary manifestations (primary attack and early [erythrocyte] relapses); 3) latent period; 4) the period of late manifestations (late relapses); 5) the period of convalescence.

Incubation period with tropical malaria - 7-12 days, with three-day and oval malaria it can be short (10-20 days) or long-term (7-14 months or more), with four-day malaria - from 3 to 6 weeks.

The onset of the disease is usually acute, a short prodrome is possible in the form of malaise, chilling, dry mouth. Malaria is characterized by a paroxysmal course, while in the first days of the disease the fever may be of the wrong type - "*initial fever*". A malarial attack consists of three phases: 1) chills (1-3 hours), 2) heat (6-8 hours), 3) sweat (2-5 hours); the total duration of the attack is from 1-2 to 12 hours. Attacks are repeated every other day (three-day, oval- and tropical malaria) or after 2 days (four-day malaria). Between attacks, the patient's state of health is satisfactory. After two or four attacks, the liver and spleen increase.

Anemia develops only at 2-3 weeks with three-day, oval- and four-day malaria, and with tropical malaria - in the first week.

The period of primary manifestations of malaria (up to 10-14 attacks), even without specific treatment, is replaced **by a latent period** . By the end of the febrile period, the attacks become shorter, the maximum temperature is lower. The level of parasitemia also decreases. A few days after the end of the fever attacks, malaria parasites are not detected in the blood. The state of health of patients during this period is satisfactory.

More than half of the patients again have acute manifestations of malaria - **relapses.**

By the time of onset, relapses are early, or close, which appear within 2-2.5 months from the onset of the disease, and late, or distant, occurring after 7-11 months. and later from the onset of the disease.

Early relapses are observed in all types of malaria and occur due to single parasites that have survived in the blood after the disease (with inadequate etiotropic therapy).

Late relapses may occur after four days of malaria, as well as three days and *ovale* , but these relapses are of different origin. With four-day malaria, relapses occur due to the activation of the reproduction of parasites that can develop in erythrocytes for a long time without increasing their population, and with three-day and ovale malaria, late relapses are due to the release of bradysporozoites from liver cells.

Three-day and ovale-malaria . There are no significant differences in the clinical manifestations of these forms. A prodromal period is characteristic, which is manifested by chilling, headache, pain in the lower back and extremities, low-grade fever. Sometimes there is an initial fever of the wrong type lasting from 1 to 3 days, after which the correct alternation of malarial paroxysms is established - every other day. As a rule, malarial attacks develop in the daytime, in the afternoon (with oval-malaria - more often in the evening). The spleen is usually enlarged after the first paroxysms, and after a week becomes painful on palpation. The liver

also moderately increases without a significant change in its functional parameters. Early (erythrocyte) and late (exoerythrocytic) relapses develop in individuals who have not received treatment or are treated only with hemoschizotropic drugs. Relapses begin without prodrome and initial fever with clear paroxysms and periods of apyrexia. A rare complication of three-day malaria is a ruptured spleen.

Four-day malaria. The onset of the disease is acute, from the first attack a strict frequency of attacks is established - two days later on the third. There is no initial fever, and paroxysms usually develop at noon. The duration of the paroxysm itself is on average 13 hours. Anemia, spleno- and hepatomegaly develop slowly and are detected no earlier than 2 weeks after the onset of the disease. Clinical manifestations of four-day malaria stop on their own after 8-14 attacks. Usually, after the primary attack, there are up to 3 relapses that occur after remissions (14-53 days). Four-day malaria in adults is usually benign. A feature of four-day malaria is that even against the background of a developed clinical picture, it is not always possible to find parasites in the blood. However, erythrocyte schizogony can persist at the submicroscopic level after the illness for many years.

In endemic areas, this type of malaria is considered the main cause of nephrotic syndrome in children. At the same time, even adequate specific therapy for malaria cannot prevent its development. Patients develop edema, massive proteinuria, hypoproteinemia. The course of nephrotic syndrome is chronic progressive, treatment is ineffective, the prognosis is unfavorable.

Tropical malaria occurs in the most severe forms, and in 98% is the cause of all malaria deaths. The prodromal period is usually noted only in non-immune individuals, and manifests itself in the form of severe headache, myalgia, chilling, nausea, vomiting, and diarrhea. The disease begins acutely, for several days the fever may be permanent (initial fever), and later it takes on a stable intermittent character with typical paroxysms. A feature of paroxysms in tropical malaria is the short duration and severity of chills. Polymorphism of temperature curves is often observed: from typical paroxysms every other day to daily and even attacks that

occur twice a day, constant fever is possible. With prolonged daily attacks during apyrexia, subfebrile temperature often persists.

Enlargement of the liver and spleen is usually noted after a week. During these periods, signs of nonspecific hepatitis may appear. Already after the first attacks, anemia develops, which is more pronounced in tropical malaria than in other forms. At the height of the disease, there is a thickening of the blood, so it is necessary to examine the hematocrit index to diagnose latent anemia. A quarter of patients with tropical malaria have changes in the cardiovascular system: deafness of heart tones, tachycardia, hypotension. When you try to get out of bed, collapse may occur. Often the disease is accompanied by severe headaches, bouts of nausea and vomiting.

Diarrhea and abdominal pain, cough, and signs of bronchitis or bronchopneumonia may occur.

The duration of the acute period of untreated tropical malaria in non-immune individuals ranges from several days to 3-4 weeks. Against the background of the formation of specific immunity, the state of health of patients slowly improves, and the disease passes into a latent period. Then, in most of them, within 10-14 days to 2 months, early relapses occur, which occur in a milder form and with a small level of parasitemia. There are no late recurrences in tropical malaria. The total duration of infection after a single infection in the absence of treatment is from 1 year to 1.5 years.

Malignant forms of tropical malaria. Prognostically unfavorable signs of the development of malignant malaria are daily fever in the absence of apyrexia between attacks, severe headache, nausea, vomiting, convulsions and increasing leukocytosis, as well as high rates of parasitemia (more than 100,000 parasites in 1 μl of blood). The most frequent complications: malarial coma (cerebral malaria), acute renal failure. Malarial algid, hemoglobinuric fever, rupture of the spleen, pulmonary edema are less common. Complications in tropical malaria can develop both in the first hours from the onset of fever, and on days 4-6.

Complications. Malarial coma occurs mainly in primary cases. In the development of a coma, a period of somnolence (stupor) is distinguished, then stupor (consciousness is partially preserved and the patient reacts to persistent questions and strong stimuli), and, finally, a period of deep coma with a complete blackout of consciousness. Meningeal symptom complex, pathological tendon reflexes, convulsions are noted. Intracranial pressure is increased, changes in cerebrospinal fluid are insignificant. With cerebral malaria, the development of malarial psychoses is possible. They can occur in the early phase of a coma or in the post-fever period, as a result of degenerative changes in the brain tissues. In the acute period, psychoses occur in the form of delirium, amentia, epileptic seizures, and manic states.

Acute renal failure in malaria can occur against the background of an infectious-toxic shock as a result of a violation of the systemic circulation, as well as due to obstruction of the renal tubules by the decay products of erythrocytes. Pronounced changes in the kidneys, sometimes accompanied by necrosis, lead to the development of oliguria and anuria. The specific gravity of urine is low, there is a pronounced proteinuria, cylindruria, pyuria, microhematuria. The content of nitrogenous wastes, creatinine increases in the blood.

Algid malaria is an infectious-toxic shock and occurs with symptoms of cardiovascular insufficiency. The pulse is thready, blood pressure is sharply reduced. The temperature drops to subnormal figures. Consciousness is preserved. The skin is cyanotic, facial features are pointed. There is involuntary diarrhea. There is oliguria and anuria.

Hemoglobinuric fever - this complication is associated with low activity of the enzyme glucose-6-phosphate dehydrogenase (G6PDH), characterized by acute intravascular hemolysis with hemoglobinuria, fever, hemolytic jaundice, acute renal failure. It develops as a result of both massive invasion and the use of antimalarial drugs (quinine or primaquine, which reduce the activity of G6PD). The main symptom of hemoglobinuria is the excretion of urine the color of dark beer. Urine, when settling, is divided into two layers: the upper layer is transparent

in the color of red wine, the lower one is dark brown or dirty-cloudy ("coffee grounds"). In the general blood test: anemia, leukocytosis, increased ESR. With the development of acute renal failure, the death of the patient may occur. Lethal outcomes in the development of this complication are observed in 30% of cases. With a favorable outcome, hemolysis stops within 2-5 days.

Pulmonary edema is often fatal; develops due to an increase in the permeability of the pulmonary capillaries due to the presence in them of a large number of erythrocytes affected by plasmodia. Edema is promoted by irrational infusion therapy, when an excessive amount of solutions is administered to combat intoxication.

An important component in the pathogenesis of malignant malaria is a metabolic disorder, which is manifested by **hypoglycemia** . It develops due to a decrease in gluconeogenesis in the liver, an increase in the consumption of glucose by parasites, and stimulation of insulin secretion. Hypoglycemia may also result from hyperinsulinemia due to side effects of quinine. Hypoglycemia is usually accompanied by acidosis and accumulation of lactic acid in tissues. Hypoglycemia is especially dangerous in pregnant women.

Differential diagnosis. It is carried out with brucellosis, typhoid fever, influenza, visceral leishmaniasis, leptospirosis, tick-borne spirochetosis, sepsis, dengue fever, hemolytic disease and anemia. Malarial coma is differentiated from hepatic, diabetic, uremic, cerebral coma.

Diagnostics. The diagnosis of malaria is established on the basis of clinical and epidemiological data and requires mandatory laboratory confirmation.

Clinical triad in the diagnosis of malaria:

1) regularly alternating typical febrile attacks; (a typical attack includes a sequence of phases of chills - fever - sweat);
2) enlargement of the spleen and liver;
3) detection of malarial plasmodia in the blood.

To confirm the diagnosis of malaria, *laboratory studies* of thick drop preparations and blood smears are necessary. The following individuals should be laboratory tested for malaria:

- feverish 5 days or more;
- who arrived within the last 3 years from malaria-prone countries with any disease accompanied by fever;
- those who have had malaria in the last 3 years, with an increase in their body temperature;
- with continuing periodic rises in temperature, despite the treatment carried out in accordance with the established diagnosis;
- with an increase in temperature within three months after a blood transfusion;
- with an increase in the liver and spleen, as well as anemia of unknown etiology.

If the first analysis is negative, then blood tests (smear and thick drop) are repeated 2-3 times with an interval of several days. High-risk groups include people who have arrived from malaria-affected countries (workers and specialists in mobile professions, seasonal workers, construction workers, military personnel, tourists).

Laboratory diagnosis of malaria

The main method of laboratory diagnostics is the detection of parasites in the blood. The study can be carried out both during an attack and in the interictal period. Blood is examined by the method of a thin smear and a thick drop, stained according to Romanovsky-Giemsa. Both methods are used together. The study requires 5 preparations of a smear and a thick drop. The taken drugs are delivered to the clinical laboratory and examined by " Cito !". The smear preserves the morphology of the parasites and the species of Plasmodium can be determined. In a stained preparation "thick blood drop", the probability of detecting plasmodium is 40-60 times higher than in a smear. If malaria is suspected, blood tests should be done frequently (every 6-8 hours until the diagnosis is confirmed).

The study of blood products also allows you to monitor the effectiveness of treatment (plasmodia disappear from the blood after 3-4 days from the start of treatment). When registering the results of the study, the type of plasmodium, the stage of development and the level of parasitemia are indicated.

The level of parasitemia is most often assessed by a "semi-quantitative" method in the preparation "thick drop" according to the following scale:

+ - corresponds to 1-10 parasites in 100 fields of view (5-50 parasites in 1 μl of blood);

++ - corresponds to 10-100 parasites in 100 fields of view (50-500 parasites in 1 μl of blood);

+++ - corresponds to 1-10 parasites in each field of view (500-5000 parasites in 1 μl of blood);

++++ - corresponds to 10-100 parasites in each field of view (more than 5000 parasites in 1 μl of blood);

+++++ - corresponds to more than 100 parasites in each field of view.

Microscopy of blood products

Blood for the preparation of preparations is taken from the 4th finger of the left hand after being punctured with a sterile scarifier. The skin puncture site is treated with 70% alcohol. The first drop of blood that comes out is wiped off with a sterile cotton swab.

To *prepare a smear* , a small drop of blood is placed on the edge of a sterile glass slide. With the right hand, a second slide with polished edges is brought to it, which is placed on a drop of blood at an angle of 40-45 °. In this case, the drop should be distributed in an even strip between the edge of the glass slide and the ground glass. Then, while maintaining the same angle of inclination, a strip of blood is quickly spread over the glass slide with a ground glass. The smear should be uniform, thin, with a fringe at the end.

To *prepare a thick drop* , a large drop of blood is applied to the surface of a freshly prepared, not very thin and not yet dried smear, over which the drop itself

spreads. A drop of blood applied to the glass can also be smeared in a circular motion with the corner of another glass slide up to approximately 2 cm in diameter. The layer of blood in a thick drop should not be very thick, otherwise, after drying, it cracks and may fall off. A thick drop is considered normal, through which, after drying, a large printed text is weakly translucent, and under microscopy, there are an average of 10–15 leukocyte nuclei in one field of view.

Smears and thick drops are dried in air. Dry smears are fixed with methyl alcohol (2-3 minutes) or for 10-15 minutes with a mixture of Nikiforov (ethyl alcohol and ether 1: 1). Then smears and non-fixed thick drops are stained with azure II -eosin according to the Romanovsky method. Before use, the paint is diluted with neutral distilled water (pH 7.2) in the ratio of 1-2 drops of paint per 1 ml of water. Coloring lasts 40-45 minutes. After exposure, the preparations are carefully washed with running water, dried in air, and examined under a microscope. A correctly colored thick drop has a purple color. In this case, erythrocytes are stained pink, parasite nuclei - cherry red, cytoplasm - blue.

To identify Plasmodium, blood products are examined at a total magnification of the microscope × 900-1000 using oil immersion.

In blood smears intraerythrocyte stages of development of parasites are found: trophozoites, schizonts and gametocytes. *Trophozoites* (from the Greek trophe - nutrition) - feeding on hemoglobin, growing stages with one nucleus. Trophozoites are divided into 5 age classes: 1) rings - the earliest stages; 2) young; 3) developing; 4) mature. *Schizonts* (from the Greek schizo - splitting) are divided into developing and mature (the latter are also called *morula*).

P. vivax in a smear can be detected at all stages of development (Fig. 9).

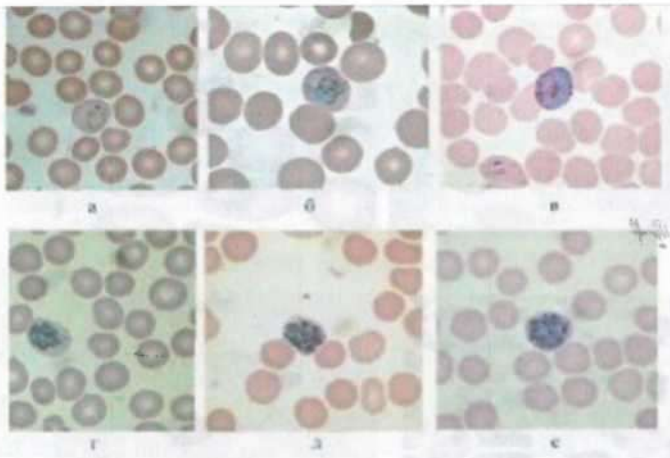

Figure 9. *Plasmodium vivax* in a thin blood smear. a – young (annular) trophozoite; b – mature trophozoite; c – young schizont; d, e – developing schizonts; f - morula (mature schizont). Coloring according to Romanovsky - Giemsa.

Plasmodium-infected erythrocytes are enlarged, discolored, and have fine, dense, red-violet granules (Schuffner granularity). Plasmodium *P. vivax* at the stage of a young trophozoite (ring) has a size of about 1/3 of the diameter of an erythrocyte. Developing trophozoites are often amoeboid in shape. Schizonts contain from 2 to 14-16 nuclei . Mature schizonts (morulas) consist of a bunch of merozoites (on average 14-16), resembling a "mulberry". Female gametocytes are similar to large trophozoites, while male gametocytes are identified by a large loose nucleus.

P. _ malariae in a smear it can also be detected at all stages of development (Fig. 10- A, B).

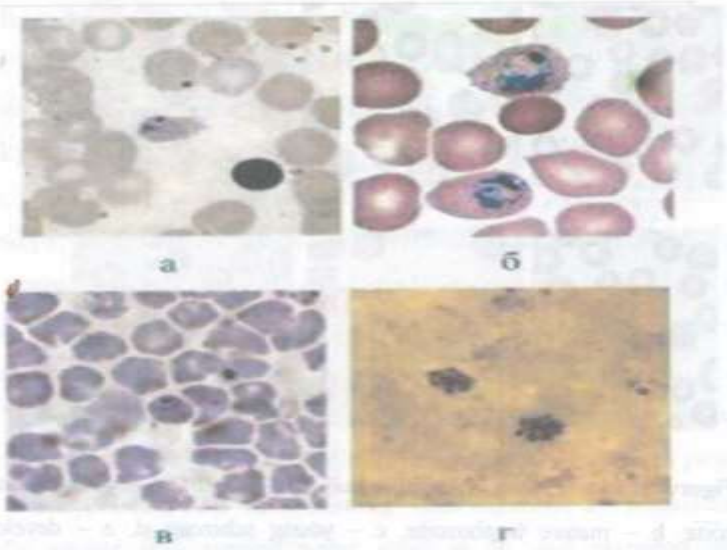

Figure 10. Causative agents of four-day and oval-malaria in a thin blood smear. a , b – young trophozoites *P. oval* ; (c *)* young trophozoite *P. malariae* ; d - mature schizont (rosette *)* *P. malariae* . Coloring according to Romanovsky - Giemsa.

Erythrocytes affected by *P.malariae* do not change in size, the parasites are smaller compared to *P.vivax* . Ring-shaped trophozoites are similar to P. *vivax* . Trophozoites and schizonts have a round compact shape. There are ribbon-like trophozoites. Morulae contain 8-10 merozoites, outwardly similar to a "daisy". Gametocytes are similar to P. vivax , but smaller.

P. ovale in smears occurs at all stages of development (Fig. 5 - C, D). Erythrocytes affected by *P. ovale* , as a rule, are enlarged, discolored, have a large rare red granularity (James granularity), some of the erythrocytes take an oval shape with a scalloped edge. All other developmental stages are similar to *P. vivax*

P. _ falciparum in peripheral blood smears, it occurs only at the stage of annular trophozoites and gametocytes (Fig. 11).

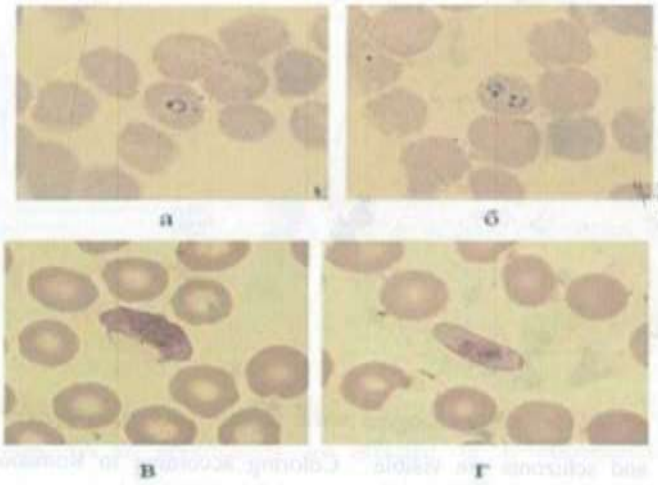

Figure 11. *Plasmodium falciparum* on a thin blood smear. A, B - young trophozoites; C.G - gametocytes. Coloring according to Romanovsky - Giemsa.

Developing and mature trophozoites, as well as schizonts and morulas, develop in the capillaries of the internal organs and are found in the peripheral blood only in the malignant course of tropical malaria. The erythrocytes affected by parasites are not enlarged in size. Ring-shaped trophozoites are very small (1/5 of the diameter of an erythrocyte), there may be several of them in one erythrocyte. Gametocytes are large crescents, shaped like bananas.

In the "thick drop" preparation, *P. vivax* is found at all stages. Distinctive feature: erythrocytes are often preserved in the form of delicate translucent discs (shadows of erythrocytes) (Fig. 12).

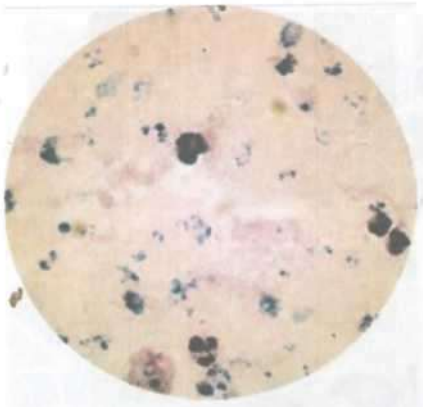

Figure 12. *Plasmodium* pathogens *vivax* in the "thick drop" preparation. Trophozoites and schizonts are visible . Coloring according to Romanovsky-Giemsa.

P. malariae in thick droplets are less deformed compared to P. vivax and therefore retains its morphological features better.

P. ovale in thick drops are similar to *P. vivax* .

P. _ falciparum in thick drops occurs in two stages: ring-shaped trophozoites and gametocytes; the latter are well preserved. The rings are subjected to severe deformation (Fig. 13).

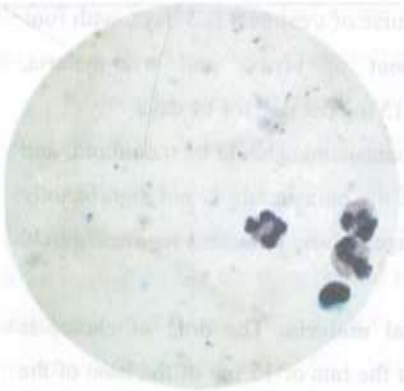

Figure 13. Pathogens *P. falciparum* in the "thick drop" preparation. Only juvenile annular trophozoites are visible . Coloring according to Romanovsky-Giemsa.

If the number of parasites in one field of view of a thick drop is 10 or more, if more than 5% of erythrocytes are affected, or if *intermediate* stages of development are detected *P. falciparum* , this indicates an unfavorable course of malaria with the threat of complications.

There are **express methods for diagnosing tropical malaria** (ParaSight - F , ICT , etc.), which are based on the qualitative detection of malarial antigen (protein II rich in histidine) using an immunochromatographic reaction. Express methods have high sensitivity and specificity (98.8%) and become positive from the first hours of the disease. They are easy to perform, do not require special medical training, and their duration is about 10 minutes. Express methods are of particular value in the field, when there is no possibility for microscopic examination of blood. PCR methods have been developed to detect the DNA of malarial plasmodia in the blood.

Serological diagnosis of malaria is not used in clinical practice.

Treatment of benign forms of malaria (three-day, *ovale* , four-day) is not difficult.

Currently, ***chloroquine is mainly used for their treatment*** . Scheme for adults: on the first day 1.0 ***chloroquine*** (in 1 tablet - 0.25), after 6-8 hours - another 0.5, on the 2nd and 3rd day - 0.5 each (per course - 2,5 gr). If fever persists, treatment can be extended for another 2 days (0.5 per day).

With three-day and oval malaria, the course of treatment is 3 days, with four days - 5 days. For the radical treatment of vivax- and oval-malaria, histoschizotropic drugs are used: ***primaquine*** 15 mg per day for 14 days.

When treating the patient, the level of parasitemia should be monitored, and if after 48 hours from the start of treatment the parasitemia is not significantly reduced, the antimalarial drug should be changed or the treatment regimen should be changed.

Treatment of uncomplicated tropical malaria. The drug of choice *is* ***mefloquine*** , which is prescribed for adults at the rate of 15 mg of the base of the drug per 1 кgrbody weight, orally, after a meal, with a large amount of liquid (at least 200 ml). The entire dose is divided into 2-3 doses, and is given every 2 hours. If within 30 minutes after taking the drug the patient vomits, you should re-take the full dose, and if 30-60 minutes after taking it, then half the dose.

WHO (Malaria treatment Guidelines , 2009) recommends the following treatment regimens for uncomplicated tropical malaria:

- ***Artemether + lumefantrine*** (coartem, riamet). The combination contains 20 mg ***of artemether*** and 120 mg of ***lumefantrine*** in each tablet. Recommended treatment regimen: 1 dose of the drug 2 times a day for 3 days. Weight doses: 5 14 кgr- 1 tab., 12 24 кgr- 2 tab., 25 34 кgr- 3 tab., 35 кgrand more - 4 tablets.
- ***Artesunate + amodiaquine*** . The recommended course dose is 4 mg of ***artesunate*** per kg of body weight and 10 mg ***of amodiaquine*** per kg of body weight. It is divided into 3 parts, each of which is administered once a day for 3 days.
- ***Artesunate + mefloquine*** . The daily dose is 4 mg of ***artesunate*** per kg of body weight and 25 mg of ***mefloquine base*** per kg of body weight and is taken in one dose. The course of treatment is 3 days.
- ***Artesunate + sulfadoxine-pyrimethamine*** . The course of treatment is 3 days. On the first day, ***artesunate*** (4 mg/kg) and ***sulfadoxine + pyrimitamine***

(1.25/25 mg/kg) are prescribed in one dose. In the next 2 days, treatment is carried out only with ***artesunate*** at the rate of 4 mg/kg in one dose.

In the **treatment of severe and malignant forms of tropical malaria** , as well as in the absence of the effect of taking the drug orally, parenteral administration of ***quinine is prescribed*** . The drug is diluted in 200-250 ml of 5% glucose or saline and injected slowly intravenously over 2-4 hours. The first dose is 20 mg/kg, further the drug is used at a dose of 10 mg/kg. The interval between injections is 8 hours. The highest daily dose for adults is 3,0 gr, and the recommended daily dose is 2,0 gr. Intravenous infusions of quinine are carried out until the patient recovers from a serious condition, after which they switch to oral administration of ***quinine disulfate*** 10 mg / kg three times a day in combination with tetracycline (250 mg four times a day) or doxycycline (100 mg twice a day). day). The general course of treatment is 7-10 days.

Simultaneously carry out pathogenetic therapy. Due to the possibility of developing acute renal failure, as well as pulmonary edema, the amount of fluid administered should correspond to the amount of urine excreted. Pathogenetic therapy is aimed at reducing the permeability of the vascular walls, metabolic acidosis, hyperazotemia, and eliminating cerebral edema. For this purpose, solutions of 5% glucose, Ringer, Gemodez, dextran, antihistamines, glucocorticosteroids (prednisolone 1-2 mg / kg of body weight), mannitol, cardiovascular drugs, vitamins are prescribed.

In the treatment of hemoglobinuric fever, the drugs that caused hemolysis are canceled, and glucocorticosteroids are prescribed. With anuria, peritoneal dialysis or hemodialysis is performed.

Treatment of renal failure, acute hemolysis with anemia and shock, DIC, pulmonary edema and other complications of malignant malaria is carried out against the background of antimalarial treatment according to the general principles of syndromic therapy. Patients with severe malaria should be hospitalized in specialized intensive care units and resuscitation.

Prevention. The main link in the system of antimalarial measures is the timely detection and treatment of sources of infection.

The fight against malaria vectors - mosquitoes - is carried out using physical, chemical and biological methods. Processing is carried out in residential premises, in territories, in reservoirs. To protect against mosquito attacks, measures are taken to prevent the penetration of insects into residential premises, and personal protective equipment, repellents are used.

Individual chemoprophylaxis is carried out for non-immune persons temporarily staying in regions endemic for tropical malaria. Used in chloroquine-resistant strains of *P. falciparum* ***mefloquine*** 250 mg once a week (one week before departure and 4 weeks after departure, the course is not more than 4 months), ***savarin*** (proguanil 200 mg and chloroquine 50 mg) - 1 tablet per day (one week before departure and 4 weeks after departure), ***doxycycline*** (recommended for those traveling to countries in Southeast Asia where resistance to mefloquine has been established) only for adults and children over 14 years old, 100 mg daily, but not more than 30 days.

Mass seasonal chemoprophylaxis is carried out to the population of active foci of three-day malaria with ***chloroquine*** - 0.25 mg / kg once a week. Persons who arrived from the foci of three-day malaria and received seasonal prophylaxis are given off-season prophylactic treatment with ***primaquine*** at a dose of 0.25 mg/kg daily at one time for 14 days.

Giardiasis

Giardiasis (Giardiasis) (ICD10 - A07.1) is a parasitic invasion caused by Giardia, asymptomatic or in the form of manifest forms with symptoms of enteritis, cholepathy and asthenia.

Etiology. The causative agent is a protozoan that parasitizes the small intestine - the flagellate *Lamblia intestinalis* . It was first discovered by professor of Kharkov University D.F. Lyambl in 1859. In 1888, the French scientist

Blanchard proposed to name these protozoa in honor of the discoverer *Lamblia intestinalis* . However, in foreign literature, another name for the pathogen is often used - *Giardia intestinalis* (*G. lamblia* , *G. duodenalis*), and the disease itself is called *giardiasis* .

Giardia exists in two forms: vegetative and cysts. Vegetative form *of Lamblia intestinalis* is a large cell 12-15 microns long and 8-10 microns wide, which has a pear-shaped shape with a pointed (narrowed) tail end. On the stained preparation, two nuclei are visible inside the cell, as well as the parabasal body. Anteriorly, in the expanded part, the body of the trophozoite is slightly flattened and somewhat depressed, which forms a suction disk. Thus, in the lateral projection, lamblia looks like a bucket. Due to 4 pairs of flagella, the trophozoite actively moves (Fig. 14).

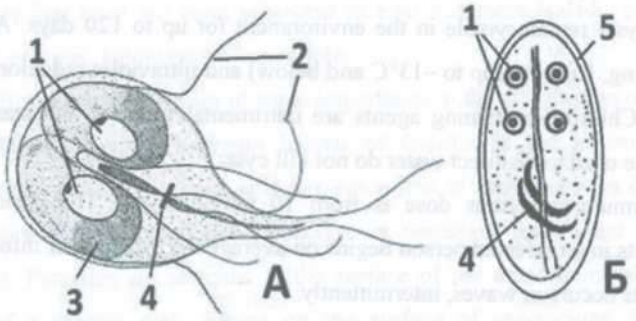

Figure 14. Giardia. A - vegetative stage (according to Gottschalk); B - cyst. 1 - cores; 2 - flagella; 3 - suction disk; 4 - parabasal bodies; 5 - cyst shell.

Giardia cysts are oval in shape. The anterior end of the cyst is narrower than the posterior one. Nuclei are located near it: two in immature cysts and four in mature ones. Thin filaments of axonemes, flagella, and parabasal bodies are also visible in the cytoplasm. The cyst membrane often lags behind the cytoplasm and a crescent-shaped gap forms between them. Cysts are 12-14 μm long and 6-10 μm wide.

Life cycle. Giardia live in the upper parts of the human small intestine. Their reproduction occurs by longitudinal binary fission. As a result of peristalsis, part of Giardia is carried to the lower intestines, where they are encysted. Cysts excreted from the host organism together with faeces can exist in the external environment for a long time. After entering the digestive tract of the host with water or food, two motile trophozoites emerge from the cyst, which leave the cyst membrane and attach to the surface of the epithelium of the small intestine.

Epidemiology. Currently, molecular biological methods have proven the existence of several Giardia genospecies, of which two are adapted to humans, as well as to primates, dogs, cats, cattle, horses and rats. However, the main source of giardiasis is a sick person who can excrete a huge amount of cysts (about 1.8 million in 1g of feces).

Giardia cysts remain viable in the environment for up to 120 days. At the same time, boiling, freezing (up to –13°C and below) and ultraviolet radiation lead to their death. Chlorine-containing agents are detrimental to cysts, but standard doses of chlorine used to disinfect water do not kill cysts.

The minimum infectious dose is from 10 to 100 cysts. The period of excretion of cysts in an infested person begins on average 9-12 days after infection. Isolation of cysts occurs in waves, intermittently.

Most often, children aged 3 to 15 years are infected with giardiasis. With giardiasis, group morbidity can be recorded.

Occupational risk groups include employees of children's institutions, sewage and irrigation services, mentally retarded and mentally ill people due to the low level of personal hygiene skills.

Factors predisposing to infection with giardiasis are: protein starvation, predominantly carbohydrate diet, consumption of large amounts of sucrose, low acidity of gastric juice, impaired immune status.

The mechanism of transmission of giardiasis is fecal-oral. Ways of distribution - water, contact and food.

The main route of transmission of Giardia cysts is water. Giardia cysts remain viable in water at temperatures from 4 to 20 ^{0}C during three months. Water outbreaks of the disease are described when drinking water from natural sources, wells, etc.

The contact-household way is typical for preschool institutions. As transmission factors, the main role is played by dirty hands of children, toys, the surface of furniture and toilet items; to a lesser extent - service personnel. In children with bad habits of holding a finger in their mouth, biting their nails, pencils, pens, etc., lamblia are detected much more often. In families where there is a child with giardiasis, all family members are often infected.

The food path is less significant. The few known food outbreaks of giardiasis have been associated with the contamination of Giardia cysts with food products that have not been subjected to heat treatment (salads, puddings, etc.). With giardiasis, autoinvasion is possible.

Household insects are of some importance in the distribution of Giardia.

Pathogenesis. The main habitat of Giardia in the human body is the duodenum and the proximal jejunum, since it is in these sections of the intestine that intensive parietal digestion occurs - a necessary component for the life of Giardia. Parasites are attached to the surface of the mucous membrane with the help of a suction disk. Fixing on the surface of enterocytes, they close the absorption surface of the small intestine and intercept the nutrients entering it.

Previously, it was erroneously believed that Giardia can live in the biliary tract, in the gallbladder, and even penetrate into the submucosal layer of the intestine.

Giardia cause mechanical damage to the epithelium on a large surface of the intestine with the development of subatrophy of enterocytes and shortening of microvilli. Multiple attachments to and detachments from the mucosa destroy the glycocalyx, which adversely affects the efficiency of parietal digestion. Irritation of the nerve endings of the intestinal wall can lead to the development of biliary dyskinesia, in which viscero-visceral reflexes lie.

Giardia metabolic products, being absorbed from the intestines, can cause sensitization of the human body.

The clinic of giardiasis is diverse and nonspecific. Severe fatal cases are not known.

According to the WHO classification, there are:

1. ***giardia carrier (latent form)*** ;
2. ***giardiasis (manifest form).***

In most of the infested (74-80%), giardiasis occurs without any clinical manifestations in the form of a latent form.

With the manifest form, the duration of the incubation period is from 7 to 28 days (on average, 2 weeks). Patients are concerned about nausea, belching when eating, heartburn, loss of appetite, cramping abdominal pain, increased gas formation and rumbling in the intestines. Sometimes subfebrile fever is noted. In most patients, the tongue is lined, there is pain on palpation in the epigastric region and the projection point of the gallbladder. The phenomena of enteritis and steatorrhea are expressed. Stools are frequent (2-4-6 times a day), watery, frothy, fetid, without admixture of blood and mucus.

Against the background of immunodeficiency, giardiasis acquires a chronic course and can last for many months or years with periodic exacerbations, during which patients report malaise, headaches, decreased ability to work, dyspeptic disorders, symptoms of gastroduodenitis.

Complications with giardiasis are not recorded. Sometimes intestinal dysbiosis develops.

Diagnostics. The polymorphism of clinical manifestations and the frequent combination of giardiasis with other intestinal infections significantly complicate clinical diagnosis.

The diagnosis of giardiasis is established on the basis of the results of parasitological examination of feces or duodenal contents by microscopy. The most informative method of laboratory diagnostics is the detection of trophozoites in the duodenal contents (most often in portion A).

To detect cysts, smears of feces prepared from mushy or shaped feces are examined, which are stained with Lugol's solution. (Fig. 15).

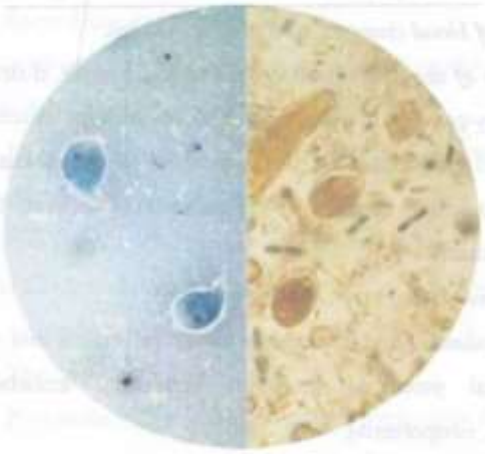

Figure 15. On the left, *Lamblia* trophozoites *intestinalis* (Romanovsky stain - Giemsa); on the right - cysts (stained with Lugol's solution).

The use of enrichment methods (ether-acetic and ether-formalin sedimentation methods) increase the sensitivity of microscopy. Coproscopy often reveals steatorrhea.

In chronic forms of giardiasis, cysts are isolated periodically, therefore, to confirm the diagnosis, it is recommended to conduct a 3-fold study with an interval of 3-5 days.

With microscopic diagnosis, it is necessary to differentiate Giardia and their cysts from other protozoa, as well as objects of a non-parasitic nature (fungal spores, plant cells, pollen grains, etc.).

Serological methods are indirect, additional methods for diagnosing giardiasis. They are characterized by low specificity and sensitivity, and can give both false positive and false negative results. The method based on solid-phase immunophoresis for the determination of Giardia coproantigen should also be referred to low-specificity.

The PCR method for the detection of Giardia DNA in feces is not widely used in laboratory practice due to the high cost of research and conflicting results.

In the general analysis of blood changes, as a rule, are absent.

Ultrasound examination of the abdominal organs in some cases, it detects signs of disorders of the biliary-pancreatic system in the form of hypotonicity or hypertonicity of the sphincter of the gallbladder, the phenomena of cholestasis and sludge syndrome, as well as reactive changes in the pancreas. However, these changes are not specific to giardiasis.

differential diagnosis. Giardia invasion is differentiated:

- with enteritis, duodenitis and gastroenteritis of infectious and non-infectious etiology, intestinal protozoal invasions (intestinal amoebiasis, balantidiasis, cryptosporidiosis, isosporiasis);
- with cholecystitis, cholecystocholangitis of another etiology;
- with malabsorption syndrome;
- with irritable bowel syndrome.

Treatment. For etiotropic therapy, drugs of *the nitromidazole (metronidazole and its derivatives: ornidazole, secnidazole, etc.), nitrofuran (furazolidone, nifuratel) and benzimidazole groups (* albendazole *) are used.*

Nonspecific therapy includes diet, *choleretic drugs, enterosorbents, enzyme preparations, vitamins, pro- and prebiotics.*

You should limit your intake of carbohydrates, especially sweets. To eliminate dysbacteriosis and secondary fermentopathy of the intestine and pancreas, prebiotics are used in combination with probiotics or synbiotics, as well as enzymatic preparations (creon, festal, enzistal, mezim-forte, etc.).

The drug of choice is ***metronidazole*** 0.25 three times a day, the course is 5-7 days.

You can also use ***tinidazole*** - for children 50-75 mg / kg of body weight per day (adults 2.0 g) in 1 dose after meals, course - 1 day;

Ornidazole - for children 40 mg / kg of weight (adults 1.5 g) per day in 1 dose after meals, course - 1-2 days.

Secnidazole - 30 mg / kg of body weight per day in 1 dose after meals, course - 1 day. Adults 2.0 g once.

Second line drugs are:

Nifuratel - for children 15 mg / kg of weight (adults 400 mg) per day in 2 divided doses after meals, course - 7 days;

Furazolidone - for children 8-10 mg / kg of weight (adults 400 mg) per day in 4 doses, the course is 5-7 days.

also ***highly effective, for*** adults and children over 12 years old - 0,4 grper day, for children 2-12 years old - 15 mg / kg of body weight per day, one dose after meals, course - 5 days.

The prognosis is favorable.

Prevention The main measures of non-specific prevention, as well as the prevention of all intestinal infectious diseases, are measures to prevent fecal contamination of water, food and other environmental objects. It is necessary to strictly observe the rules of personal hygiene, to destroy mechanical carriers - flies and cockroaches. Of particular importance is the maintenance of a strict sanitary and hygienic regime at food and water supply facilities, as well as in children's institutions. All those who come to work at these enterprises and in these institutions are examined for intestinal protozoa. Children undergo the same examination when registering for kindergartens and boarding schools. Identified carriers are sanitized. It is advisable to combine preventive measures in children's groups with measures to prevent enterobiasis. All patients with acute intestinal infections are also examined for infection with intestinal protozoa. Infested with Giardia are treated. If the quality of drinking water is not controlled for the content of protozoa, it is recommended to disinfect it by boiling.

leishmaniasis

Leishmaniasis (code according to ICD10 - B55) are obligate transmissible diseases of a predominantly zoonotic nature, the causative agents of which are protozoa of the genus *Leishmania* .

Etiology and classification . Leishmaniasis is endemic to many areas of the tropical and subtropical zones of the globe. There are three types of leishmaniasis in the Republic of Uzbekistan:

1. **Visceral Mediterranean-Central Asian** (children's) - zoonosis caused by *L. infantum* ;
2. **Cutaneous anthroponous** (or "urban"), caused by *L. tropica* ;
3. **Cutaneous zoonotic** (or "rural") caused by *L. major* ;

The causative agents of cutaneous leishmaniasis were identified by different researchers - Kunningham (Cunningham , 1884), Firth (Firth , 1891). B 1898 gr. P.F.Borovsky determined that these organisms belong to the simplest. B 1900 gr. Wright observed similar parasites in the spleen of a patient with visceral leishmaniasis and in 1903 gr. first published an accurate description of these parasites and drawings.

Leishmania exist in two forms: amastigotes and promastigotes.

Amastigotes are found in the body of warm-blooded hosts in the cells of the reticuloendothelial system, capable of phagocytosis. They have the appearance of small oval or rounded bodies ranging in size from 2 to 5 microns (Fig. 16).

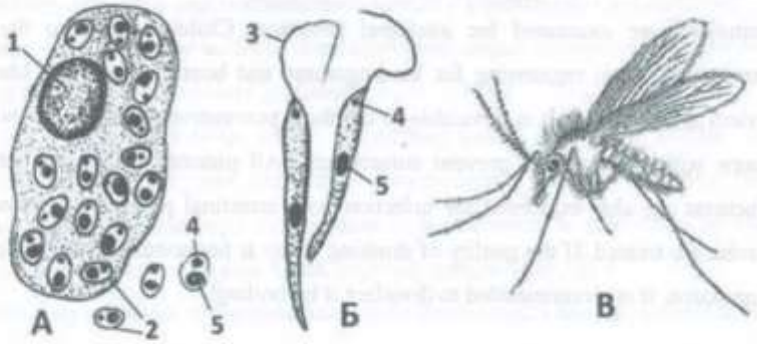

Figure 16. Leishmania. A - non-flagellated (amastigote) forms in a mammalian leukocyte; B - leptomonas (flagellated) forms from mosquito intestines . 1 - the nucleus of a leukocyte; 2 - amastigotes; 3 - flagellum; 4 - kinetoplast; 5 - core; **B - mosquito** - a carrier of leishmania (from Zenkevich).

Protoplasm is stained according to Romanovsky-Giemsa in a grayish-blue color. In the central part or on the side there is an oval nucleus, which is painted red or red-violet. A kinetoplast is located near the nucleus (a round grain or a short stick, lying eccentrically and staining more intensely than the nucleus, in a dark purple color). The presence of a nucleus and a kinetoplast is the main feature that distinguishes Leishmania from other formations (platelets, yeast cells, etc.).

promastigotes are found in the intestines of the mosquito vector and have an elongated fusiform shape.

Their length is 10-20 microns, width - 3-5 microns. The kinetosome and kinetoplast are located in the anterior part of the body, through the anterior end of which a short flagellum emerges. The nucleus, protoplasm and kinetoplast are stained according to Romanovsky-Giemsa in the same tones as amastigotes. In cultures, promastigotes often gather in bunches in the form of rosettes, with flagella facing the center (agglomeration phenomenon).

The life cycle of Leishmania proceeds with a change of hosts: a vertebrate animal, including humans, and a carrier, the mosquito (Diptera: Psychodidae: Phlebotominae). Mosquitoes are small two-winged insects ranging in size from 1.2 to 3.7 mm.

The mosquito becomes infected with amastigotes by sucking blood on an infected vertebrate. In the intestines of the mosquito, Leishmania pass into the promastigous stage, multiply by longitudinal division, develop within a week and turn into invasive forms that concentrate in the anterior sections of the intestine and in the proboscis of the carrier.

With repeated bloodsucking, promastigotes from the mosquito enter the tissues of the vertebrate host, are phagocytosed by cells of the reticuloendothelial system, turn into amastigotes and multiply by simple fission in two. Cells overflowing with parasites are destroyed, amastigotes are captured by other cells, in which the reproduction process is repeated.

MEDITERRANEAN-CENTRAL ASIAN CHILDHOOD VISCERAL LEISHMANIASIS

Etiology. The causative agent is *Leishmania infantum* .

Epidemiology. The Mediterranean-Central Asian form of visceral leishmaniasis is a zoonosis. Dogs are the main natural reservoir of the pathogen. - Additional reservoirs in different territories can be jackals, foxes, wolves.

In North Africa and Southwest Asia, children aged 1–4 years are most commonly affected; in East Africa - 5-9 years. In China, the countries of Central Asia and Southern Europe, patients are also registered in older age groups. The season of infection is summer, and the season of incidence is autumn or spring of the following year.

Mosquitoes of various species serve as carriers, each of which is characterized by its specific population size, seasonal dynamics of activity and the number of generations per season, the degree of anthropophilicity, endo- or exophilicity; certain requirements for temperature conditions, air humidity and breeding sites.

Pathogenesis. At the site of the mosquito injection, a histiocytoma is formed on the skin, consisting of macrophages, reticular, epithelioid, giant and other cells (primary affect). Then the process is generalized. Leishmania, multiplying in the cells of the system of mononuclear phagocytes (SMP), penetrate into the regional lymph nodes, then disseminate to the spleen, bone marrow, liver, lymph nodes of the intestine and other internal organs. Due to the proliferation of SMP cells, the development of dystrophic and necrotic processes, the functions of parenchymal organs are disrupted, and their sizes increase. The pulp of the spleen is most affected, as a result of which hematopoiesis is suppressed, anemia progresses, which is aggravated by damage to the bone marrow. The life expectancy of leukocytes decreases, which leads to the development of granulocytopenia. Hyperplasia of liver Kupffer cells and interglobular fibrosis lead to compression and atrophy of hepatocytes. The production of prothrombin decreases, which, in

combination with thrombocytopenia, can lead to severe bleeding in the mucous membranes. Hepatic amyloidosis may develop.

Clinic. The most significant feature of the Mediterranean-Central Asian leishmaniasis is the involvement in the pathological process of both peripheral and visceral lymph nodes with the development of mesadenitis and bronchoadenitis, accompanied by abdominal pain and coughing fits. Often there is the development of bacterial pneumonia. The skin is pale, has an earthy tint without hyperpigmentation.

The incubation period for the manifest course of infection varies from one month to a year. The disease can occur in acute, subacute and chronic forms.

The acute form is rare and occurs mainly in young children. It is characterized by a rapid course and, without treatment or with delayed therapy, ends in death.

Slightly more common ***subacute form*** , which is difficult, often with the development of complications. Due to hyperplasia of the lymphoid-macrophage system, anemia, leukopenia, granuloitopenia, hypergammaglobulinemia develop, the spleen and liver increase sharply. On palpation, the lower edges of the liver and spleen are dense, painless (Fig. 17).

Figure 17. Splenomegaly in visceral leishmaniasis. From the Parasitological Museum of the Military Medical Academy. CM. Kirov.

The fever takes on an irregular (atypical) character. A sharp exhaustion of the patient occurs, muscle tone decreases, the bone marrow is affected, and a secondary immunodeficiency state develops. The bacterial flora is activated, enterocolitis, furunculosis, multiple abscesses, ulcerative lesions of the oral and intestinal cavities occur. Without specific therapy, the death of the patient may occur in 5-6 months.

chronic form is the most common. It occurs mainly in older children, less often in adults. This form is characterized by a milder course, prolonged remissions and, with timely treatment, ends in recovery. With specific treatment, even a greatly enlarged liver and spleen quickly decrease to their normal size. A significant number of cases of invasion proceeds in an asymptomatic or erased form and ends with spontaneous recovery.

In immunocompromised individuals, especially those infected with HIV, the disease develops rapidly and is very difficult, difficult to treat and often leads to rapid death of the patient. The Mediterranean-Central Asian form of visceral leishmaniasis, like its other forms, belongs to the group of AIDS-associated invasions.

Diagnosis in endemic foci is possible on the basis of clinical symptoms.

In the hemogram - a decrease in the number of erythrocytes (up to 1-2 × 10^{12} / l or less), hemoglobin (up to 40-50 g / l or less) and color index (0.6-0.8). Characterized by poikilocytosis, anisocytosis, anisochromia, leukopenia (up to 2-2.5×10^{9} /l) and neutropenia (sometimes up to 10%) with relative lymphocytosis. Perhaps the development of agranulocytosis, moderate thrombocytopenia is characteristic. ESR rises to 90 mm/h. The indicators of blood clotting and the osmotic stability of erythrocytes decrease. The level of globulins increases.

The diagnosis is confirmed by the detection of leishmania in smears from bone marrow punctate, stained according to Romanovsky-Giemsa . It should be noted that the number of leishmania in the preparations is small, and for a qualitative diagnosis, at least 3 slides with bone marrow smears should be viewed.

NNN medium to detect Leishmania promastigotes (Fig. 18).

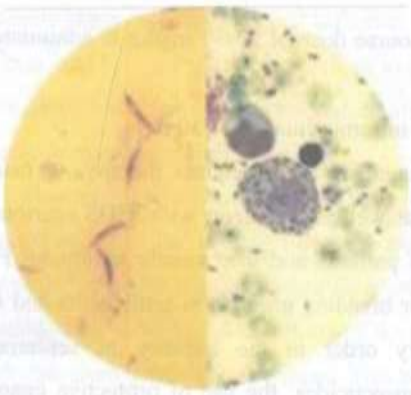

Figure 18. Promastigonic forms of Leishmania (left), amastigotic forms in a macrophage (right). Coloring according to Romanovsky - Giemsa.

Serological research methods are also used (direct agglutination test, IDIFT and EIA). It must be borne in mind that in almost half of patients with HIV infection and visceral leishmaniasis, the results of serological tests are negative. In recent years, the rK -39 immunochromatographic test , which is characterized by high specificity and sensitivity, has become widely used for diagnostics. PCR can also be used to detect Leishmania DNA in the tissues of affected organs. Sometimes a biological test is used - infection of golden hamsters.

Treatment. Assign preparations of 5-valent antimony: ***sodium stibogluconate*** or ***meglumine antimonate*** . The calculation of the daily dose is 20 mg/kg (based on antimony). Injections are given daily, intramuscularly or intravenously. The course of treatment is 30 days. In case of recurrence of the disease, treatment is repeated after a 14-day interval.

In case of severe course of the disease and ineffectiveness of treatment with pentavalent antimony preparations, second-line drugs are prescribed, which include amphotericin B and paromomycin.

Amphotericin-B is administered at 0.5 mg/kg intravenously daily or every other day until a course dose of 20 mg/kg is reached.

Liposomal amphotericin-B , a course dose of 20-30 mg/kg is administered for 10-20 days.

Paromomycin - 15 mg / kg daily intramuscularly for 30 days.

The prognosis, with timely diagnosis and adequate therapy, is usually favorable. In the absence of treatment, as well as in patients with AIDS - serious.

Prevention. Active detection of patients and their timely treatment. Fight against mosquitoes: destruction of their breeding grounds in settlements and their environs; maintaining proper sanitary order in the territory of settlements; treatment of premises with effective insecticides; the use of protective canopies and nets treated with insecticides. Carrying out activities to eliminate stray dogs and control the incidence of domestic dogs using serological tests.

ZOONOUS (RURAL) SKIN LEISHMANIASIS

Etiology . The causative agent of the disease is *Leishmania major* .

Epidemiology. *The* main natural reservoir *L. major* are served by rodents. In the desert areas of Central Asia, where a significant part of the endemic regions of the Old World for cutaneous leishmaniasis is located, rodents that lead a colonial way of life - large gerbils (*Rhombomys opimus*). They build deep complex burrows with many passages and chambers. In the Middle East, North and West Africa, sand rats (*Psamomys obesus*). Transfer *L . major* among rodents are carried by many species of mosquitoes. For example, *Phlebotomus papatassi* , *Ph . mongolensis* , *Ph . andrejevi* - in Central Asia, Kazakhstan, Mongolia; *Ph . caucasicus* - in Transcaucasia, Iran, Afghanistan. A person does not play an epidemiological role as a source of invasion. In intensive natural foci of zoonotic cutaneous leishmaniasis, the majority of the local population is ill with leishmaniasis in childhood. After the illness, persistent lifelong non-sterile immunity to all forms of cutaneous leishmaniasis develops .

Pathogenesis. The disease begins with the appearance at the injection site of a mosquito (most often on the skin of the extremities, especially the lower ones) of

a tubercle surrounded by a rim of inflamed skin. The size of the tubercle at first is 2- 4 mm, and by the 2nd day it increases to 8-10 and even up to 15 mmin diameter. At the same time, the surrounding inflammatory edema of the skin also increases, sometimes reaching significant sizes.

The emerging inflammatory papule or furuncle-like node in the process of development after 1-2 weeks undergoes central necrosis, as a result of which an ulcer is formed with steeply steep edges with a diameter of 2- 4 mm. The ulcer is surrounded by a powerful infiltrate of doughy consistency, due to the decay of which the ulcer gradually increases. The bottom of the ulcer is uneven, usually covered with a yellowish-gray or yellowish-green coating, the discharge of ulcers is serous-purulent, abundant. The collapse of the central part of the tubercle goes very quickly, the ulcer expands along the periphery and sometimes reaches the size of a child's palm and more, especially on the back and stomach. Around the ulcer in the infiltrate zone, new, smaller leishmaniomas ("tubercles of seeding") sometimes appear, which, after going through the same development cycle, turn into ulcers that merge with the main ulcer, forming larger, almost continuous acute inflammatory ulcerative foci of scalloped outlines. They are surrounded by a roller of infiltrate, which along the periphery gradually merges with healthy skin.

Clinic. The incubation period lasts from a week to 1-1.5 months (most often 10-20 days). With multiple leishmaniomas, which occur as a result of multiple mosquito injections (or repeated bloodsucking of the same infected mosquito), several tens or even hundreds of ulcers may occur. Usually they are small, round or irregular in shape, with undermined, even or scalloped edges (Fig. 19).

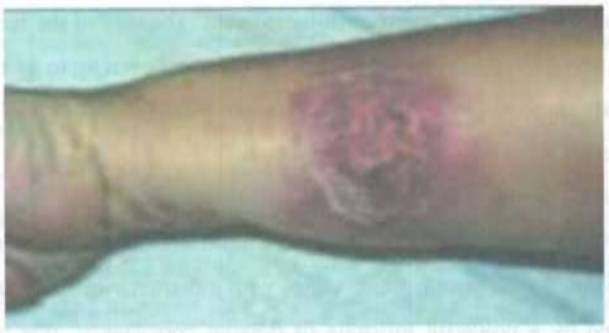

Figure 19. Ulcer in cutaneous zoonotic leishmaniasis.

The ulcers themselves are not painful. However, severe pain occurs during dressings or when the ulcers are accidentally injured (blows, pressure). The appearance of severe soreness of the ulcer often indicates the addition of a secondary bacterial pyogenic flora.

Zoonotic cutaneous leishmaniasis is characterized by the development of painful regional lymphadenitis with severe lymphangitis (Fig. 20).

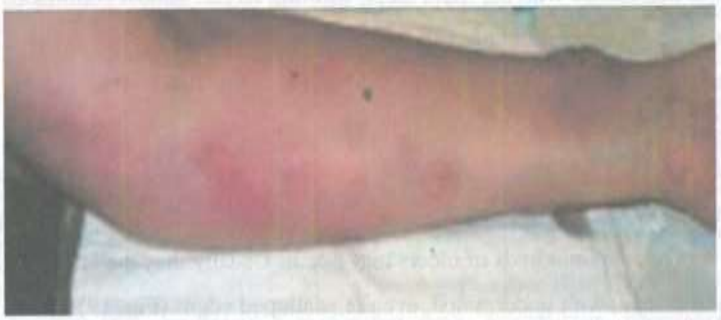

Figure 20. Regional lymphangitis in cutaneous zoonotic leishmaniasis.

On the 2-3rd month after the formation of ulcers, their bottom is gradually cleared of necrotic-purulent plaque and islands of fresh and juicy granulations appear, which gradually fill the bottom of the ulcer. The infiltrate is reduced. The formed scars are hyperemic. In the future, the scars become atrophic and depigmented. They stay for life.

The whole process from the moment a papule or tubercle appears to complete scarring lasts from 2 to 5-6 months, that is, much shorter than with anthroponotic cutaneous leishmaniasis.

Complications . The most common complication is the addition of a secondary infection, in which pain increases, inflammatory changes increase and the process of epithelization becomes more difficult. With lymphadenitis on the lower extremities, swelling of the legs and feet is possible due to lymphostasis.

Diagnostics. Of great importance are anamnestic data on the stay of the patient in the endemic territory. Material for microscopic examination should be taken from the edge of the lesion or infiltrate. The resulting small tissue elements and serous fluid are used to prepare a smear. A fixed smear is examined in the usual way after staining according to Romanovsky-Giemsa. In the presence of ulcers or large granulomas, the material is taken from the marginal infiltrate. The resulting material can be cultivated.

To ensure the correct diagnosis, a comprehensive examination of the patient is required, including histological, bacteriological and immunological research methods.

Differential diagnosis is carried out with furunculosis, pyoderma, trophic ulcers.

Treatment. If the disease does not really bother the patient, it is better not to carry out etiotropic therapy, but to allow the course of the disease to develop naturally. Parenteral administration of drugs used to treat visceral leishmaniasis (according to the same schemes) is indicated only with a strong inflammatory reaction and the development of regional lymphadenitis, and also if leishmaniomas are located in places where the formation of scar tissue can cause disability (for example, in the area joints) or cause a disfiguring cosmetic defect.

The prognosis is favorable.

Prevention. The main measure against zoonotic cutaneous leishmaniasis in the Central Asian republics has been and remains the destruction of great gerbils. The most effective measures are the destruction of rodent burrows during the

planned development of the territory for agricultural production. Less successful are local measures to inoculate rodent burrows in limited areas (for example, around settlements).

Mosquito control, carried out by insecticide treatments of houses, outbuildings and rodent burrows, is ineffective. The use of personal protective equipment - curtains, repellents - has not received practical application.

Previously, a highly effective preventive measure was leishmanization - artificial infection ("vaccination") with a low-virulence strain *L. _ major* . Currently, Leishmanization is not applied.

ANTHROPONOUS (URBAN) SKIN LEISHMANIASIS

Etiology. Pathogen - *Leishmania tropica* .

Epidemiology. The disease is common in Europe, Asia, Africa, Central Asia and Transcaucasia.

The source of the invasion is a sick person. Dogs serve as an additional reservoir. The main carrier is the mosquito *Ph . sergenti* . Diseases occur throughout the year, which is explained by the variation in the incubation period from several months to 2 years. The anthroponotic type of cutaneous leishmaniasis occurs mainly in cities and urban-type settlements, but is sometimes also observed in rural areas. The disease is sporadic. Epidemic outbreaks are rare. Among the local population, mainly children are ill, among visitors - people of all ages.

The pathogenesis is similar to that of zoonotic cutaneous leishmaniasis.

Clinic. The incubation period ranges from 2-4 months to 1-2 years. It is possible to extend the incubation up to 4-5 years. After its expiration, at the site of the bite of infected mosquitoes (more often the face, upper limbs), inconspicuous single, less often multiple tubercles with a diameter of 2- 3 mmwith a smooth, as if shiny surface appear. They slowly increase and after 3-4 months reach 5 10 mmin diameter. Their color becomes reddish-brown with a bluish tinge. After several months, the bumps can gradually dissolve and disappear almost without a trace.

However, such an abortive course is rarely observed. More often , a barely noticeable retraction appears on the surface of the tubercle, and a scale is formed, which then turns into a yellowish-brown crust tightly attached to the tubercle.

After falling off or removing the crust, a bleeding erosion or a shallow, often crater-shaped ulcer with a smooth or fine-grained bottom covered with a purulent coating is found. The edges of the ulcer are uneven, corroded, sometimes undermined. For a long time, the ulcer is covered with a dense crust (Fig. 21). Ulcers are usually painless even when infused. Regional lymphangitis and lymphadenitis are not typical.

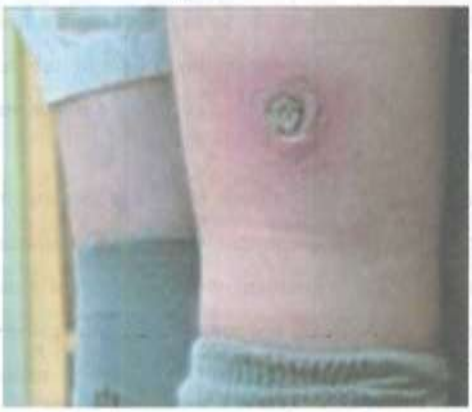

Figure 21. Ulcer in cutaneous anthroponotic leishmaniasis

After 2-4 months after the formation, the process of scarring of ulcers gradually begins, which ends on average a year from the moment the tubercle appears. In some cases, the disease is delayed for 2 years or more.

Sometimes, after the first leishmanioma, new tubercles appear, which often resolve without undergoing ulcerative decay, especially late ones. Since resistance to superinvasion develops only 6 months after the disease, the clinical course of subsequent leishmaniomas is almost the same as the development of primary ones.

After suffering anthroponotic cutaneous leishmaniasis, in about 10% of cases, a sluggish chronic tuberculoid cutaneous leishmaniasis (recurrent cutaneous leishmaniasis), clinically resembling lupus erythematosus , which can last for

decades . It is assumed that the cause of the development of tuberculoid cutaneous leishmaniasis is immunodeficiency.

Complications. The development of pyodermatitis as a result of the addition of a secondary infection.

Differential diagnosis is carried out with furunculosis, pyoderma, trophic ulcers.

Diagnostics. Same as for zoonotic cutaneous leishmaniasis.

Treatment. If the disease does not really bother the patient, it is better not to carry out etiotropic therapy, but to allow the course of the disease to develop naturally. As a specific treatment, the same drugs are used as for the treatment of visceral leishmaniasis. In the early stages, with the formation of single tubercles, it is possible to chip with specific drugs or use ointments containing ***chlorpromazine*** **(2%),** ***paromomycin*** **(15%)** or ***clotrimazole*** **(1%)** .

Tuberculoid cutaneous leishmaniasis is difficult to treat. Therapy of this complication is carried out similarly to the treatment of visceral leishmaniasis (according to the same schemes). Specific treatment with pentavalent antimony does not always lead to improvement. Long-term repeated courses of treatment are often required in combination with immunostimulants, vitamins and restorative drugs.

The prognosis is favorable.

Prevention. Patients are actively identified and treated. Areas of affected skin should be covered with bandages (especially at night) to prevent mosquito infestation.

A set of measures is being carried out aimed at the destruction of mosquitoes and the elimination of their breeding grounds (beautification of settlements, maintenance of proper sanitary conditions in them, timely cleaning of construction and household waste, treatment of premises with insecticides). To eliminate anthroponotic cutaneous leishmaniasis, mass treatment of premises with residual insecticides is necessary, leading to a decrease not only in the number of mosquitoes that carry malaria, but also mosquitoes.

SECTION II. HELMINTHOSES

GENERAL CHARACTERISTICS OF HELMINTHOSIS

Worms ***(Vermes)*** are the first bilaterally symmetrical three-layered invertebrates . They are represented by three types: Flat (*Plathelminthes*), Round (*Nemathelminthes*) and Annelids (*Annelides*).

Along with free-living forms, there are a large number of worm species that lead a parasitic lifestyle. Parasitic worms are called helminths and the diseases they cause are called helminthiases. The branch of parasitology that studies helminths is called helminthology.

Human helminthiases are caused by representatives of two types of worms: Flat and Round. The source of infection with helminths are humans, domestic and wild animals.

Depending on the characteristics of the biological cycle of development of pathogens, the factors of their transmission, three groups of helminthiases are distinguished: geohelminthiases, biohelminthiases and contagious helminthiases.

Geohelminths develop without changing hosts. In eggs laid by females, larvae develop to the invasive stage in the external environment, since this requires free oxygen from the air (roundworms, hookworms).

Biohelminths **are** characterized by development with a change of hosts. Their larvae develop in one or two intermediate hosts, and the sexually mature phase is formed in the final host. For most biohelminths, a person serves as the final host (bovine tapeworm, liver fluke, etc.) and only for some - intermediate (cystic echinococcus, alveococcus). There are biohelminths whose larvae and mature forms live in the same host and even in the same host individual, but in different tissues or organs (Trichinella).

The group of **contagious helminthiases** , transmitted directly from person to person, includes enterobiasis and hymenolepiasis.

The penetration of helminths into the human body occurs mainly by the oral route, in some cases - percutaneous and transmissible way.

Each type of helminth is characterized by a certain localization in the human body. Most of the worms that parasitize humans are intestinal worms. But there are also tissue helminths, which in the adult stage live in the subcutaneous tissue or in the lymphatic system. Some helminths at different stages of their development can be in different organs.

Immunity in most helminthiases is unstable. Antibodies after deworming or spontaneous recovery disappear within 6-12 months.

FLAT WORMS' TYPE

Flukes

Fascioliasis (ICD10 - B66.3) is a zoonotic trematodosis characterized by a predominant lesion of the biliary system.

Etiology. The causative agent of the disease serves as a liver fluke (fasciola) - *Fasciola hepatica* , sometimes giant fasciola (*F. gigantica*) .

A mature individual of the liver fluke has a leaf-shaped body about 2-3 cm long (giant - up to 6 cm). At the front end of the body there is an oral sucker, and I will fight after it - the abdominal one. Behind the mouth opening, then follows the muscular pharynx. Both of these formations are the sucking apparatus. The pharynx passes into a short esophagus, followed by a branched, blindly ending intestine. They feed on the epithelium of the bile ducts, erythrocytes and leukocytes (Fig. 22).

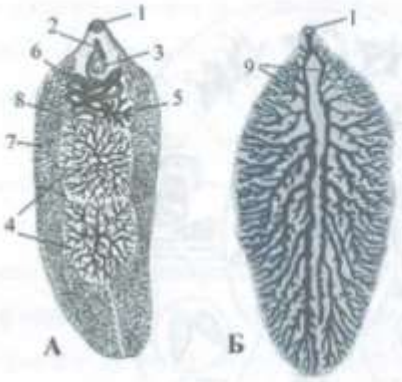

Figure 22. Liver fluke. A - reproductive system; B - digestive system. 1 - oral sucker; 2 - cirrus; 3 - abdominal sucker; 4 - testes; 5 - ovary; 6 - uterus; 7 - vitelline glands; 8 - ootype; 9 - branches of the intestine .

Fasciola is a hermaphrodite. The genital opening is located between the oral and ventral suckers. Behind the ventral sucker lies the uterus, behind which is an unpaired branched ovary, and on the sides of the body there are fine-grained yolk glands, their ducts flow into the ootype. The middle part of the body is occupied by two highly branched testes. From them go the vas deferens, then the ejaculatory canal, ending with a cirrus.

The eggs are large, 120-140 × 70-80 microns, oval, yellowish-brown in color, there is a lid on one of the poles

Life cycle. Fasciola is localized in the bile ducts of the liver, gallbladder, rarely in the pancreas of the final hosts - herbivorous mammals and humans (Fig. 23).

Figure 23. The development cycle of the liver fluke (according to K.I. Abuladze, 1990).

The eggs of the parasite, along with the bile, enter the small intestine of the final host, and then with feces are excreted into the external environment. For further development, they must fall into the water. Eggs mature within 3-4 weeks and a larva is formed inside - miracidium, covered with cilia. The larva emerges from the egg, swims, and penetrates into the body of a small pond snail, which is an intermediate host for the parasite. In the liver of the mollusk, miracidium turns into a sporocyst, in which redia are formed parthenogenetically from germ cells. The latter, reproducing parthenogenetically, give rise to a generation of cercariae, which leave the body of the mollusk, swim in the water and, attaching to underwater plants, turn into adolescariae - a larva covered with dense shells.

Adoleskaria is the invasive stage for the definitive hosts. After entering the intestines of a mammal, they are released from the shell, and penetrate into the places of final localization.

Epidemiology. Fascioliasis occurs in almost all countries of the world with developed animal husbandry, especially widely in Asia and Africa. The disease at people meets usually in the form of sporadic cases; sometimes outbreaks occur,

covering up to a hundred or more people. Sporadic cases of human fascioliasis are constantly recorded in the Republic of Uzbekistan.

Fascioliasis is one of the most dangerous and widespread helminthiases of farm animals. It is registered on all continents of the globe and causes great damage to livestock farms due to the mass death of animals during epizootics, significant loss of live weight, and a decrease in milk yield.

Fascioliasis is a zoonosis. Infection of the external environment occurs by excreting eggs with the feces of the final host. A person becomes infected rarely and accidentally by eating raw wild watercress, sorrel, wild onion and garlic, as well as by drinking raw water from open natural reservoirs. Adolescaria attached to plants survive up to a year in moist soil and water, quickly die when dried, but can survive in hay for several months.

Pathogenesis. Once in the human gastrointestinal tract with food, fasciol larvae are released from the membranes and migrate to the liver.

The migration of larvae from the intestine to the site of localization in the liver can occur in two ways: hematogenously through the vessels of the portal vein system or actively through the peritoneal cavity. In the latter case, the larvae penetrate the intestinal wall and exit into the abdominal cavity, then perforate the Glison capsule and enter the liver tissue. Moving along the liver tissue, they damage the parenchyma. Moves are formed, which later turn into fibrous strands. Ultimately, fascioli are localized in the bile ducts, less often in the gallbladder.

Sometimes fascioli are carried by blood flow to other organs, most often to the lungs, where they encapsulate and, before reaching puberty, die. In addition, young fascioli can bring microflora from the intestine to the liver, causing the development of secondary cholangitis. The main pathological changes are observed during the migration of larvae through the liver parenchyma, the duration of which is 4-6 weeks.

The development of acute cholestasis is explained by impaired patency of the common bile duct. Adult helminths cause proliferative cholangitis with

adenomatous changes in the epithelium , periductive fibrosis , and fibrosis of the gallbladder wall.

Clinic. The incubation period is 1-8 weeks. There are acute and chronic stages of the disease.

The acute stage of the disease is characterized by a sudden increase in body temperature up to 38 °C and above, which lasts for 1-3 weeks. There is weakness, increased fatigue. The liver and spleen increase in size (Fig. 24).

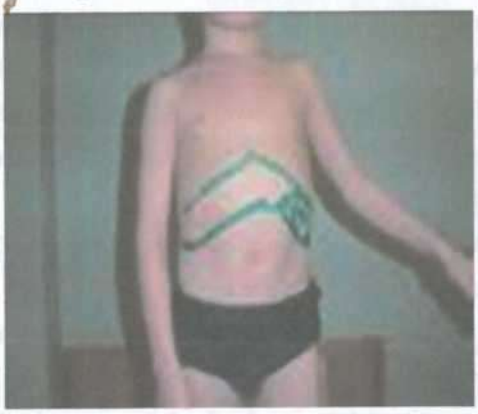

Figure 24. A 7-year-old child with fascioliasis with hepatosplenomegaia.

Sometimes there are vomiting and diarrhea, bronchitis with an asthmatic component. There are frequent cases of development of allergic reactions of immediate type (urticaria, etc.).

In the acute stage of the disease, leukocytosis with eosinophilia (from 10–20 to 60–80%) is observed in the peripheral blood, often with the development of a leukemoid reaction (20–60 × 10^9/l), an increase in ESR. These changes are most pronounced at 2-3 weeks of illness. A biochemical study in the blood reveals an increase in the level of transaminases, alkaline phosphatase and direct bilirubin.

The acute stage of the disease lasts up to two months, after which the clinical symptoms gradually disappear, and the disease passes into the chronic stage, in which the main symptoms are associated with organ damage to the liver and biliary tract. The liver is enlarged, its dense and painful lower edge is palpated on

palpation. Sometimes patients are disturbed by severe paroxysmal pain in the right hypochondrium. Periods of relative well-being are replaced by periods of exacerbation, during which jaundice appears with relatively low levels of ALT and AST and a significant increase in alkaline phosphatase. However, peripheral blood parameters during this period remain within the normal range, including the content of eosinophils. As the liver dysfunction progresses, hypo- and dysproteinemia develop, and transaminase activity increases. The size of the liver decreases, fibrosis develops.

Complications. Purulent angiocholangitis, liver abscesses, sclerosing cholangitis, obstructive jaundice. With prolonged invasion - the development of cirrhosis of the liver.

Diagnostics. Fasciolosis can be suspected by the combination of epidemiological history and clinical symptoms:

- the presence of cases of fascioliasis in the area (in animals and humans);
- eating unwashed greens growing on waterlogged banks of water bodies unprotected from fecal pollution or in wetlands;
- acute onset of the disease, fever in combination with allergic reactions (angioedema, urticaria), pain in the right hypochondrium or epigastric region, liver enlargement, leukocytosis, eosinophilia. Already in the acute stage of the disease, serological methods (IHAR - indirect hemagglutination reaction, EIA) are informative, but due to insufficient sensitivity and specificity, they cannot be used to establish a final diagnosis.

3-4 months after infection, the diagnosis can be confirmed by the detection of helminth eggs in the duodenal contents or in the feces (Fig. 25).

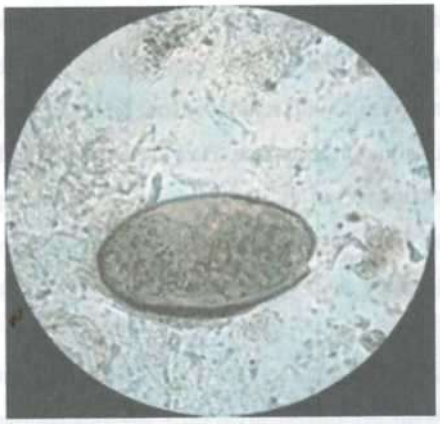

Figure 25. Fasciola egg hepatica.

The parasitological diagnosis of fascioliasis presents certain difficulties due to the fact that parasites do not lay eggs for a long time, and also due to the relatively small number of eggs shed. Therefore, in the study of feces, it is advisable to use sedimentation methods (ether-formalin, etc.).

Transient eggs may be found in faeces after ingestion of fasciola-infected bovine liver. In these cases, it is necessary to conduct repeated parasitological studies 3-5 days after the exclusion of the liver and offal from the patient's diet.

In some cases, fascioli can be detected by ultrasound of the liver, when helminths are found in the gallbladder and large bile ducts (Fig. 26).

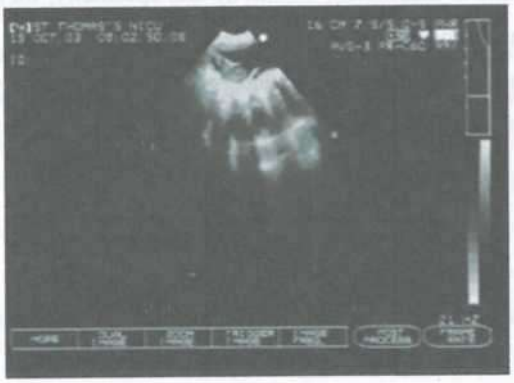

Figure 26. Fascioli at the bottom of the gallbladder. ultrasound.

Differential diagnosis. Fascioliasis should be differentiated from other liver trematodes, viral hepatitis, liver tumors, leukemia, and biliary tract diseases of other etiologies.

Treatment of patients with fascioliasis is carried out in a hospital. For the acute period of the disease, bed rest is prescribed, a diet with a restriction of coarse fiber and fats. Diet #5 is preferred. In the acute stage, anthelmintic drugs are prescribed after the relief of fever and allergic manifestations.

According to WHO recommendations, the drug of choice for the treatment of fascioliasis is ***triclabendazole*** , which is prescribed at a dose of 10 mg / kg (in severe cases 20 mg / kg) in two doses with an interval of 12 hours for one day. You can use ***bitionol*** at a dose of 30-50 mg / kg / day. in 2-3 doses every other day - 10-15 doses. Both funds are not registered in Uzbekistan.

You can use ***praziquantel*** 75 mg/gk/day in 2-3 doses for 1-2 days. It is not recommended to prescribe the drug to children under 2 years of age, women in the 1st trimester of pregnancy. Given the partial excretion of the drug from the body with breast milk, lactating women are not recommended to breastfeed on the day of treatment and the next day.

However, the effectiveness of *praziquantel in* relation to *F. hepatica is* low.

To quickly remove dead parasites from the bile ducts, it is advisable to prescribe choleretic agents, and to prevent possible allergic reactions, antihistamines.

Monitoring the effectiveness of treatment is carried out 3 and 6 months after the course of therapy by examining feces or duodenal contents.

The prognosis for early detection and treatment is favorable. With a long course of invasion, the development of purulent cholangitis and cirrhosis of the liver is possible.

Prevention. Veterinary measures are being taken to sanitize infected animals. To prevent fascioliasis , it is necessary to refrain from drinking unboiled water from stagnant or slowly flowing sources, as well as from eating poorly washed greens (wild watercress, etc.).

DICROCELIOSIS

Dicroceliosis (ICD10 - B66.2) is a zoonotic biohelminthiasis characterized by damage to the hepatobiliary system.

Etiology. The causative agent of dicroceliosis is the lanceolate fluke *(Dicrocoelium lanceatum)* (Fig. 27).

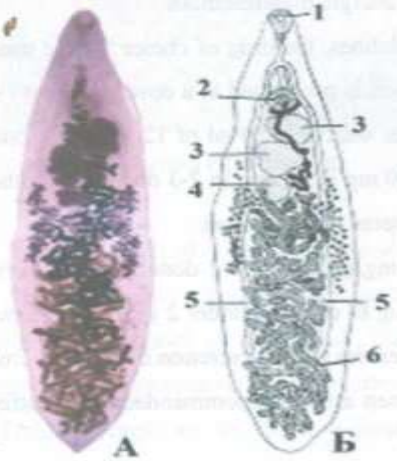

Figure 27. Dicrocoelium lanceatum . A is the appearance. Stained with alum carmine. B - scheme of the structure. 1 - oral sucker; 2 - abdominal sucker; 3 - testes; 4 - ovary; 5 - intestines; 6 - uterus (according to G.G. Smirnov, 1959).

The body of the lanceolate fluke is elongated, usually pointed at both ends, 10-14 mm long. The pharynx departs from the oral sucker, passing into the esophagus. Two straight branches of the intestine depart from it. The testicles are compact, located immediately behind the ventral sucker. Behind the testicles lies a rounded ovary. The entire back of the body is occupied by a highly developed uterus, clogged with eggs. In the lateral parts of the body are vitelline glands.

The eggs are oval, yellowish or brown in color, with a lid, 38 - 45 microns in size (Fig. 28).

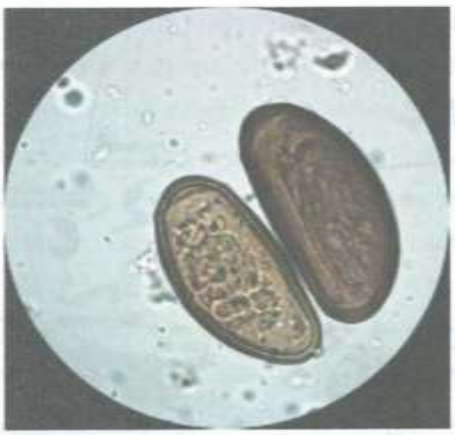

Figure 28. *Dicrocoelium* eggs *lanceatum* .

Life cycle. Sexually mature individuals (marites) parasitize in the liver ducts of herbivores, rarely in humans. Eggs with faeces enter the soil and are swallowed by terrestrial mollusks of the genus *Zebrina* or *Helicella* , in the liver of which parthenogenetic reproduction of the parasite occurs, resulting in the formation of a large number of cercariae. They, encysting, stick together into prefabricated cysts and are released from the mollusk to the outside. Further, these cysts enter the ants of the genus *Formica* . In the body of an ant, cercariae leave the intestine and encyst. One of them is localized near the brain ganglion of the insect, as a result of which the behavior of the ant changes. Infected ants normally perform their working functions during the day, but by evening, when the air temperature drops, they climb onto the tops of the stems and leaves of herbaceous plants and, clinging to them with their jaws, hang until morning, completely losing the ability to move. As a result, the likelihood of parasites getting into their final hosts, which swallow the infected ants along with the grass, greatly increases (Fig. 29).

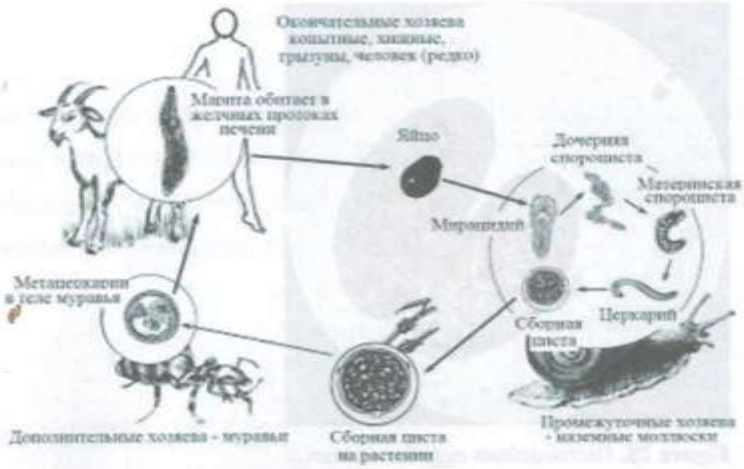

Figure 29. Development cycle of the lanceolate fluke

Epidemiology. Dicroceliosis is registered in Europe, Asia, Africa, North and South America . In Uzbekistan in domestic ungulates infection occurs almost everywhere .

The source of infection is the final hosts of the helminth (sheep, goats, cattle, wild ungulates, some rodents and lagomorphs). A person becomes infected by accidentally swallowing infested ants with vegetables, berries, wild-growing edible herbaceous plants. Children get sick more often. Sporadic cases of human invasion are recorded on all inhabited continents. The life span of parasites in the body of the final hosts is more than 6 years.

Pathogenesis the same as in fasciolosis, but the pathological processes and clinical symptoms are much less pronounced. With intensive invasion, due to a violation of the outflow of bile, catarrhal cholangitis, angiocholitis, biliary dyskinesia, and sometimes hepatitis develop.

Clinic. With a low intensity of invasion, the disease proceeds subclinically (most cases) or asymptomatically.

With a manifest course of the disease, the acute phase is pronounced. There is nausea, vomiting, salivation, heartburn, bitterness in the mouth,

headaches appear. The body temperature rises. Patients complain of pain in the right side of the abdomen or in the epigastrium. Subicteric sclera and skin are noted. The liver is enlarged, its surface is smooth, the edges are rounded; sometimes the spleen is enlarged. Eosinophilia in the acute period is 10% or more. Often an allergic syndrome (urticaria) is detected, loose stools appear. With massive invasion, hyperbilirubinemia, moderate hyperenzymemia (mainly due to alkaline phosphatase) are detected.

For the chronic stage of the development of the disease, which begins 3-4 weeks after infection, the subsidence of pathological processes is characteristic - the disappearance of allergic reactions, the normalization of peripheral blood parameters. At the same time, paroxysmal pains in the right hypochondrium, loss of appetite, and unstable stools may persist.

With the addition of a bacterial infection and the development of ascending cholangitis - fever, an increase in the size of the liver, leukocytosis, an increase in ESR.

Complications. In rare cases, biliary cirrhosis develops.

Diagnostics . _ The diagnosis is established on the basis of the clinical picture of the disease, epidemiological history and laboratory results .

In a laboratory examination, eggs are found in the duodenal contents or in the feces of the patient.

After eating the liver of small or cattle affected by these helminths, transit eggs can be found in the feces. Therefore, a few days before the study, the liver and its products should be excluded from the diet.

Differential diagnosis is carried out with other trematodes.

Treatment . The drug of choice is *praziquantel* . It is prescribed in a daily dose of 75 mg / kg immediately after meals, in 3 doses with an interval of 4-6 hours for one day. The maximum single dose is 2 g, the maximum daily dose is 6 gr(10 tablets).

In the late stage of the disease, with the development of cholangitis and biliary dyskinesia, duodenal soundings are prescribed with the introduction

magnesium sulfate or ***sorbitol*** , which are carried out 1-2 times a week for 1-2 months. Choleretic agents are shown, preferably from the group of cholekinetics. For pain, antispasmodics and analgesics are used. In case of accession of a secondary infection of the biliary tract, antibiotics are prescribed, taking into account the sensitivity of the microflora of the duodenal contents to them. With the development of anemia, especially in children, patients are prescribed a complete protein-vitamin diet and iron supplements.

Quality control of the quality of etiotropic therapy is carried out in the same way as with fascioliasis.

The prognosis is favorable.

Prevention . Veterinary measures are being taken to sanitize infected animals. Sanitary and educational work with the population, especially among those professionally associated with animal husbandry , to prevent the penetration of ants into food.

To prevent dicroceliosis , it is necessary to thoroughly wash and scald meadow vegetation (sorrel, etc.) with boiling water before eating it. In hiking trips, it is recommended to pack products in plastic bags to prevent ants from getting there.

TAPE WORM

TENIARINCHOSIS

Teniarinhoz (ICD10 code - B68.1) is a biohelminthiasis with a chronic course, characterized mainly by gastrointestinal disorders.

Etiology. The causative agent is bovine or unarmed tapeworm (*T aenia saginata*). The length of the parasite is 4-12 m, but larger individuals are also found (Fig. 30). The body consists of a scolex (head) and a long strobila (the body itself), consisting of many segments (proglottids).

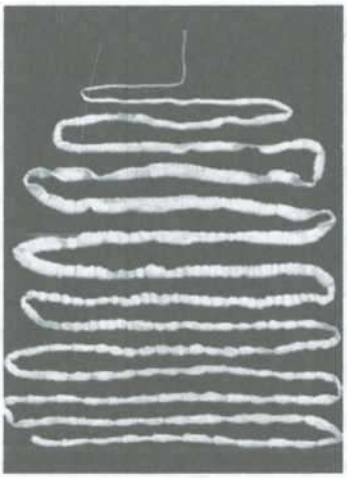

Figure 30. Bull tapeworm *Taenia saginata* (according to N.A. Kholodkovsky, 1916)

The scolex is square-oval in shape with four well-developed suckers and a rudimentary proboscis without hooks. The neck is short. The strobila consists of 1000-2000 proglottids.

The immature proglottids come first. In the middle part of the strobili are almost square hermaphroditic segments; they have a well-developed male and female reproductive system. In the final part of the strobila there are elongated mature segments, which are almost completely filled with a developed closed uterus with eggs, from the central part of which 17-35 lateral branches extend in both directions (Fig. 31).

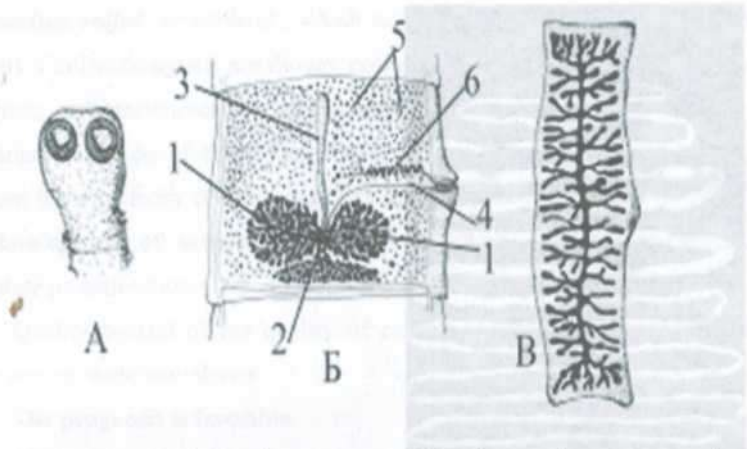

Figure 31, Scolex and segments of Taenia sa ginata: A - scolex; B - hemaphrodite segment; And - a mature segment (1 - ovary, 2 - vitelline gland, 3 - uterus, 4 - vagina, 5 - testes, 6 - ejaculatory canal).

The eggs are oval or spherical in shape, have a diameter of about 28-44 microns. Inside the egg is a six-hooked embryo - the oncosphere. It is covered with a radially striated shell, which is surrounded on the outside by an embryonic shell, protected from above by an egg shell (Fig. 32).

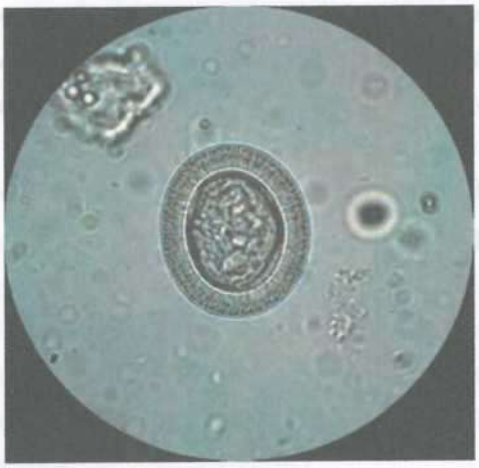

Figure 32. Egg *Taenia saginata* .

Life cycle. The only definitive (final) host for bovine tapeworm is a human, whose small intestine is parasitic with tapeworm. When the strobilus of the bull tapeworm reaches 5 7 meters lengths, mature segments begin to come off and, together with feces or on their own (actively crawling out through the anus), the segments come out. On average, 6-8 proglottids come out per day, each of which contains up to 175 thousand eggs. With active movement, the segment is injured on surrounding objects and the eggs are squeezed out of the uterus through the damaged front edge, seeding the soil and plants.

The intermediate host is cattle (bulls, cows, buffaloes, yaks, etc.). Cattle become infected by eating pasture plants contaminated with parasite eggs. In the intestines of the intermediate host, the egg shells dissolve, and the oncospheres penetrate the capillaries of the intestinal wall with the help of hooks, and then they are carried with blood throughout the body, settling mainly in the connective tissue of the muscles, where after 4-5 months they turn into Finns (cysticerci) (Fig. 33).

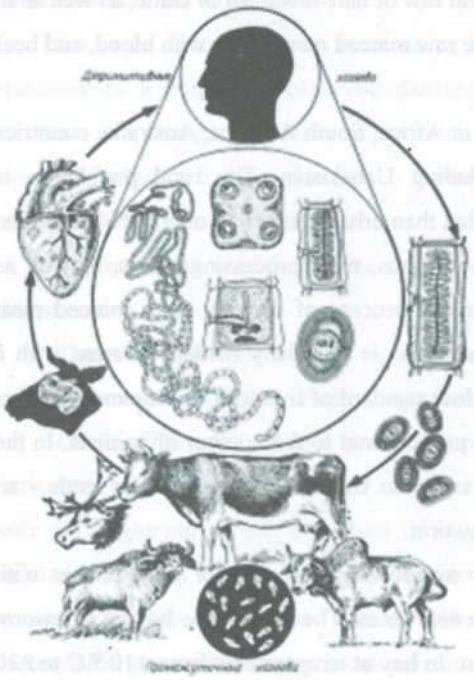

Figure 33. Taenia life cycle diagram sa ginata (according to K.I. Abuladze, 1990).

Cysticercus is an oval vesicle with a clear liquid, ranging in size from 5 to 10 mm, in which there is a scolex with a neck. Life expectancy of cysticerci in the intermediate host is 8-9 months.

Cysticerci enter the body of the final host (human) by eating insufficiently thermally processed Finnose meat of cattle. In the human intestine, the cysticercus scolex turns out of the vesicle, attaches with suction cups to the mucous membrane of the small intestine (usually the duodenum), and a strobilus begins to grow from the neck.

During the day, the strobilus of the parasite lengthens by 7 - 10 cm. Allocation of mature joints begins in 2-4 months. after infection. The life span of the parasite can reach 20 years. Bull tapeworm, as a rule, parasitizes a person in the singular, hence its name "tapeworm" (from the French " s olitaire" - single). Multiple invasion is rare.

Epidemiology. Teniarinhoz is common in areas with developed animal husbandry, where they traditionally eat raw or half-raw meat of cattle, as well as in other regions among people who love raw minced meat, steak with blood, and beef kebabs.

There are foci of teniarinhoza in Africa, South America, Australia, countries of Eastern Europe and Asia, including Uzbekistan. The rural population is predominantly sick (3 times more often than urban residents), over 80% are adults. Among the sick, workers of livestock farms, meat processing plants, as well as housewives who become infected in the process of cooking from minced meat (cutlets, dumplings, etc.) predominate. This is especially evident in areas with a generally low sanitary culture and a low standard of livestock management. In this case, the risk of infection is directly proportional to the number of animals. In the same areas, there is a high risk of infection from an infested human cattle - an intermediate host of an unarmed tapeworm.

Animals become infected by swallowing segments or oncospheres with grass, hay, water, or by licking urine near feces. The eggs of the bovine tapeworm are stable in the external environment. In hay at temperatures from +10 ° C to +30

° C, they remain viable for 21 days, in water - 33 days, in liquid manure - 70 days, on grass - more than 150 days.

Pathogenesis. The pathogenic effect of the unarmed tapeworm is due to the action of its suckers and actively mobile elements of the strobila, which damage the mucous membrane, irritate the intestinal receptors and affect the motor and secretory functions of the gastrointestinal tract as a whole. As a result, catarrh occurs in the small intestine. The passage of the proglottids through the Bauhinian valve may be accompanied by a pain syndrome resembling an attack of appendicitis. Pain also occurs with the introduction of segments into the appendix and the development of inflammatory processes in its mucosa. A large tapeworm can cause intestinal blockage. Cases of penetration of the parasite into the biliary tract and pancreatic duct with their subsequent obstruction, and even into the lungs (aspiration of segments from vomit) are described. But more often dyskinesia of the excretory tracts of the liver and pancreas develops as a result of the neuro-reflex effects of the helminth.

Intensive consumption of nutrients by the parasite, violation of absorption processes as a result of mechanical damage to the mucosa and inflammatory processes in it create a deficiency of the most valuable components of nutrients, as a result of which a feeling of hunger is constantly felt, food intake increases, and body weight decreases.

The sensitization of the organism to the metabolic products of the parasite is also of certain importance.

The constant creeping out of the segments from the anus and their movement over the skin has a depressing effect on the patient's psyche.

Clinic. Often the disease is asymptomatic. Clinical manifestations of the disease are observed after the full development of the parasite and coincide in time with the beginning of the allocation of segments by it. Patients complain of malaise, weakness, dizziness. Dyspeptic syndrome is characteristic - nausea, vomiting, sometimes diarrhea. Cramping pains in the abdomen are possible, cholecystitis, pancreatitis, hypochromic anemia may develop.

In children, teniarinhoz is more severe than in adults and is often accompanied by general toxic symptoms (weakness, fatigue, decreased performance), unstable stool (constipation is replaced by diarrhea), flatulence, hypersalivation against the background of a decrease or lack of appetite. Pain is often noted. Pain in the abdomen they have intermittent cramping in nature, have different localization - near the navel, along the small intestine, in the epigastric region, in the area of the projection of the duodenum, gallbladder and appendix. In children, asthenoneurotic manifestations are not uncommon: increased irritability, headache, fainting, sleep disturbance.

Isolated cases of the development of Meniere's syndrome and epileptiform convulsions are described. In this case, there is a hearing impairment, tinnitus, paroxysmal dizziness, accompanied by balance disorder, nausea, vomiting, increased sweating, changes in the activity of the digestive, urinary and cardiovascular systems.

In a young child, teniarinhoz can cause the development of an acute obstructive form of intestinal obstruction or perforation of the intestinal wall. Sometimes the tapeworm segments, crawling into the appendix, cause the development of appendicitis (Fig. 34).

Figure 34. A segment of the bovine tapeworm in the appendicular process.

Peripheral blood counts in most patients do not go beyond the normal range, sometimes moderate eosinophilia, leukopenia, slight anemia and elevated ESR can be observed.

Complications . Mechanical intestinal obstruction, perforation of the intestinal wall, appendicitis, cholangitis, pancreatitis.

Diagnostics. The usual methods of coprological analysis, used to diagnose most helminthiases, are not very effective in teniarhynchosis, since the uterus in teniids does not have an outlet. Some of the eggs enter the feces only when it breaks during the separation of the segments.

The most common method adopted in mass surveys of the population is a survey on the allocation of segments that are more often seen by patients than the proglottids of other cestodes. Since actively moving segments leave eggs on the prianal skin folds, a prianal scraping is used for diagnosis according to the same method as for enterobiasis (Fig. 35).

Figure 35. Mature segments of a bull tapeworm.

The eggs of all teniids are practically indistinguishable from each other, therefore, differential diagnosis is based on the study of the morphology of the proglottids or the scolex of the helminth. The segments, squeezed between the glass slides, are visible in the light. In the segment of the bovine tapeworm, 17-32 lateral branches depart from the central trunk of the uterus, and in the pork

tapeworm there are 8-12. If the uterus is poorly visible, then before viewing, the segments are kept for some time in a 50% glycerol solution

The tapeworm head, placed between two glass slides, is examined under a low magnification microscope. The differential diagnostic sign of bovine tapeworm is the absence of hooks on the scolex. (Fig. 36).

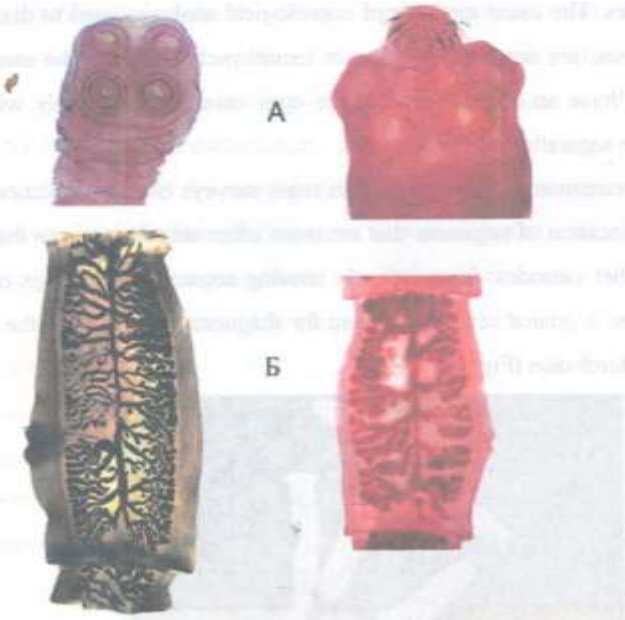

Figure 36. Scolexes (A) and mature segments (B) of bovine (1) and porcine (2) tapeworms. Stained with alum carmine.

Differential diagnosis is carried out with diphyllobothriasis and teniasis.

Treatment. Etiotropic therapy includes the appointment of antiparasitic agents.

The drug of choice is ***niclosamide*** . The daily dose for adults is 2- 3 gr(8-12 tablets), for children under 2 years old - 0,5 gr(2 tablets), 2-5 years - 1,0 gr, 6-12 years - 1,5 gr. Niclosamide tablets are finely crushed into powder and added to 1/3

cup of warm water, stir and drink. It is taken as a single dose, in the morning on an empty stomach or in the evening 2 hours after a light dinner consisting of a liquid, low-fat meal. On the eve of treatment, easily digestible food is prescribed (broth, mashed potatoes, kissels, kefir, ground meat). On the day of treatment, immediately before taking niclosamide, you need to take 1- 2 grdrinking soda.

Praziquantel can also be used . A drug taken in one go inside, during meals, without chewing, with a small amount of water in a daily dose for adults - 25 mg / kg, for children - 15 mg / kg of body weight. The drug is not prescribed for children under 2 years of age, as well as pregnant and lactating women.

If there are contraindications to the use of the above funds, you can use pumpkin seeds (Semina Cucurbitae), peeled from the remnants of the pulp of the pericarp and dried without heating. In terms of activity, they are inferior to modern synthetic drugs, but do not have a toxic effect and do not cause side effects. They are prescribed mainly for poor tolerance of other antihelminthic drugs and children. 2 days before the start of the use of pumpkin seeds, the patient is prescribed an enema daily in the morning, and a saline laxative in the evening on the eve of treatment. On the day of treatment on an empty stomach, an enema is given, regardless of the presence of stool. Pumpkin seeds are used in two ways.

1. Raw or air-dried pumpkin seeds are peeled, leaving their inner green shell intact. 300 grpeeled seeds (for adults) are ground in small portions in a mortar. After the last portion, the mortar is washed with 50 - 60 ml of water and poured into a plate with crushed seeds. You can add 50 - 100 grhoney or jam and mix thoroughly. The patient takes the resulting mixture on an empty stomach in small portions for an hour, lying in bed. After 3 hours, a laxative is given, then after half an hour, regardless of the presence of a stool, an enema is given. Eating is allowed after a stool caused by an enema or laxative. For daily intake, crushed seeds are prescribed for children in the following doses: 3 - 4 years - 75 gr, 5 - 7 years - 100 gr, 8 - 10 years - 150 gr, 11 - 15 years - 200 - 250 gr.

2. A portion of unpeeled pumpkin seeds is crushed together with the peel in a meat grinder or mortar, poured with double the amount of water, and evaporated

for 2 hours over low heat in a water bath, without boiling. The broth is filtered through gauze, after which an oily film is removed from its surface. The entire dose of the decoction is taken on an empty stomach for 20-30 minutes. 2 hours after taking the medicine, a saline laxative is given. The number of seeds for preparing a decoction is determined depending on the age of the patient: for adults - 500 gr for children 10 years old - 300 g, 5 - 7 years - 200 gr, up to 5 years - 100 - 150 gr.

Pathogenetic therapy is aimed at stopping dyspeptic and pain syndromes, normalizing the function of the biliary and enzyme systems of the gastrointestinal tract, and restoring the intestinal microflora.

To correct the function of the biliary system, choleretic agents are prescribed (field artichoke extract, nettle leaves, hepatofalk plant), to restore the intestinal microflora - pro- and prebiotics.

The prognosis is usually favorable.

Prevention. Measures for the prevention and control of teniarhynchosis should be aimed at neutralizing the source of invasion, protecting the environment from fecal pollution and blocking transmission routes. The implementation of a set of preventive measures is carried out by medical and veterinary services with the participation of administrative and economic structures.

In order to identify the source of invasion in endemic areas, mass helminthological examinations of the population are carried out. Good results are obtained by polling the population for the detection of segments of the parasite. Identified patients are subject to deworming.

The protection of the environment from pollution by oncospheres includes measures to improve the sanitary condition of settlements and livestock complexes, the protection of soil and water bodies from contamination by human feces.

To prevent human infection with cysticerci, a mandatory thorough veterinary examination of cattle meat, especially cattle from individual farms, should be ensured.

Personal prevention consists in the exclusion from the diet of raw and insufficiently thermally processed meat of cattle, as well as meat that has not passed veterinary control (not branded).

Sanitary-educational work is of great importance in prevention. The population should know how dangerous it is to eat meat that has not passed veterinary examination. Particularly active sanitary and educational work should be carried out in areas that are unfavorable for taeniasis. For health education of the population, radio and television are used, brochures and posters are published, talks are held, etc.

HYMENOLEPIDOSIS

Hymenolepiasis (ICD10 - B71.0) is an intestinal helminthiasis from the cestodosis group, caused by pygmy tapeworm and occurring with symptoms of a predominant lesion of the upper gastrointestinal tract.

Etiology. The causative agent of the disease is the dwarf tapeworm *(Hymenolepis nana)* **.** Its ribbon-like body consists of a scolex (head), neck and strobili. The length of the parasite is from 5 to 50 mm. The scolex is equipped with 4 hemispherical suckers and a proboscis bearing a corolla of 20-30 hooks. The strobila has 200-300 segments. They are trapezoidal in shape, their width is greater than their length. The middle segments are hermaphroditic, and the posterior ones are mature and contain a uterus with eggs (Fig. 37).

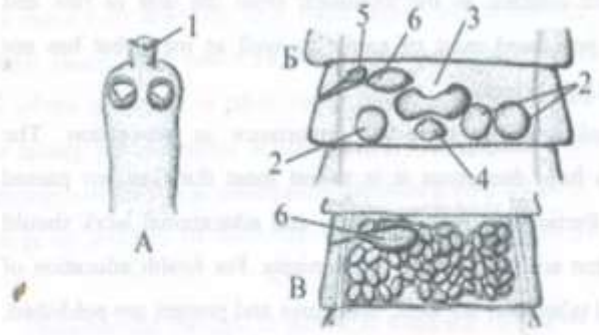

Figure 37. Morphology of the pygmy tapeworm. A - scolex, B - hermaphrodite segment, C - mature segment. 1 - proboscis with hooks, 2 - testicles, 3 - ovary, 4 - yolk gland, 5 - cirrus, 6 - seminal receptacle.

The eggs are oval (40x53 microns), their shell is colorless, two-circuit. The oncosphere is six-hooked, has its own thin shell, from which 6 long transparent threads extend at the poles, holding the embryo in the center of the egg (Fig. 38).

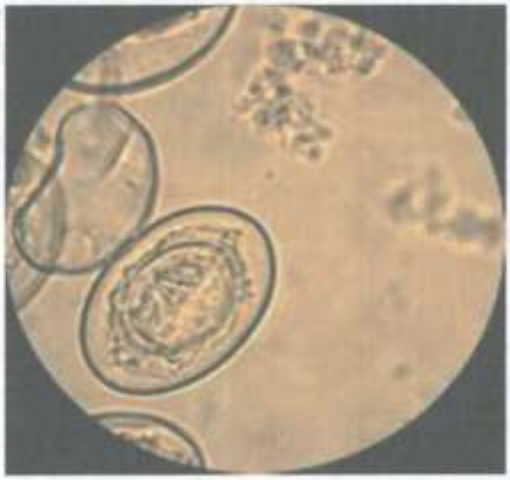

Figure 38. *Hymenolepis* eggs *nana*.

The life cycle begins and ends in the human body, which is both the final and intermediate host for it (Fig. 39).

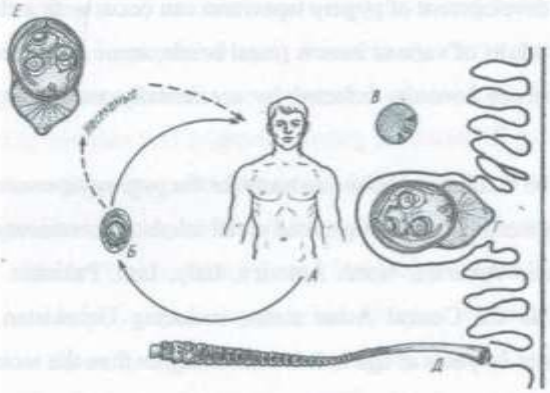

Figure 39. The development cycle of the pygmy tapeworm (according to different authors). A-defenitive host - human; B - egg with oncosphere; B - oncosphere in the intestinal lumen; D - cysticercoid in the intestinal villus; D - sexually mature worm; E, cysticercoid in the gemcele of the intermediate insect host.

A person becomes infected by ingesting mature tapeworm eggs . In the small intestine, the oncospheres are released from the egg membranes and actively penetrate into the villi, where, through a series of transitional stages, they turn into a Finn - a cysticercoid. The latter consists of a swollen anterior part containing a scolex and neck, and a tail-like posterior appendage. After 4-6 days, cysticercoids destroy the villi, exit into the intestinal lumen, attach to the mucous membrane, and after 14-15 days develop to the sexually mature stage.

The terminal proglottids are separated from the strobila and destroyed in the intestine, and the eggs released from them with feces enter the external environment. The lifespan of a dwarf tapeworm is about 2 months.

In case of violations of the digestive organs, weakening of the host's defense mechanisms, and other factors, autoinvasion is observed. At the same time, oncospheres emerge from eggs laid by parasites in the intestines of the same host.

These larvae invade the mucosa of the small intestine and give rise to a new generation of helminths.

Occasionally, the development of pygmy tapeworm can occur with a change of hosts, when larvae or adults of various insects (meal beetle, some fleas) become intermediate hosts. A person becomes infected by accidentally swallowing the above insects with food.

Mice and rats can be facultative definitive hosts for the pygmy tapeworm.

Epidemiology. Hymenolepiasis is registered on all inhabited continents. It is widely distributed in Latin America, North America, Italy, Iran, Pakistan. High incidence is also noted in the Central Asian states, including Uzbekistan. The incidence of children under 14 years of age is 3-3.5 times higher than the incidence of adults.

Hymenolepiasis is a contagious helminthiasis. The source of the invasion is man. Transmission factors are household items contaminated with helminth eggs, toys, food products.

Stability of pygmy tapeworm eggs in the environment is low - a few hours. Therefore, the appearance of secondary cases of invasion in organized groups (kindergartens, nurseries) indicates gross violations of sanitary and hygienic norms and rules. In these cases, more than half of the children can become infected in a year.

Pathogenesis. Leading in the pathogenesis of hymenolepiasis is mechanical damage to the mucous membrane of the small intestine by larvae and mature helminths. Developing in the villi of the small intestine, the larvae cause their enlargement, swelling, degenerative changes and necrosis with the development of ulcerative lesions, which can sometimes reach the muscular layer. With a massive invasion, when hundreds or even thousands of tapeworms parasitize in the intestine, constantly changing their attachment sites, focal necrosis merges, forming extensive defects in the intestinal mucosa. However, due to the high regenerative and compensatory capacity of the small intestine, even in those who

suffer from hymenolepiasis for a long time, the general condition remains satisfactory.

In the process of invasion, the enzymatic and motor functions of other parts of the digestive system (stomach, liver, biliary tract) are disrupted, which leads to impaired digestion and absorption of food products. Dysbiosis develops, the antitoxic, protein- and pigment-forming functions of the liver are disturbed, the secretion of gastric juice decreases, hypovitaminosis C, B_2, PP, etc. occur. Allergic reactions are essential in the pathogenesis of hymenolepiasis.

Clinic. The incubation period lasts about 2 weeks. The severity of clinical symptoms largely depends on the characteristics of the individual reactivity of the organism. Some people do not become infected with hymenolepiasis even in an experiment. Almost a third of those infected are asymptomatic.

The following syndromes are typical for hymenolepiasis:

1) dyspeptic (drooling, loss or lack of appetite, nausea, aching dull pain in the abdomen, unstable stool, diarrhea);

2) astheno -neurotic (malaise, fatigue, decreased activity, memory impairment, working capacity, headache, increased nervous excitability, fainting, somnambulism);

3) allergic (skin itching, rash, Quincke's angioedema, vasomotor rhinitis, asthmatic bronchitis, asthma attacks, eosinophilia);

4) epileptiform (epileptiform seizures, convulsive muscle twitches);

5) anemic (normo- and hypochromic anemia as a result of hypovitaminosis C, P, group B);

6) febrile (low-grade fever, short-term high fever, increased ESR, leukocytosis, neutrophilia, shift of the leukocyte formula to the left).

There is a decrease in the acid-forming function of the stomach, enzymatic disorders in the intestines, and colitis phenomena. Some patients may develop chronic ulcerative gastroduodenitis, disorders in the hepatobiliary system (enlargement of the liver, a slight increase in the activity of aminotransferases, a violation of the protein-synthesizing function of the liver, an increase in bilirubin),

an increase in mesenteric lymph nodes, a decrease in blood pressure, a decrease and deformation of the *R wave* in all leads on the ECG, signs of myocardial dystrophy.

Sometimes the disease acquires a chronic relapsing course, and can last for years, despite ongoing treatment.

Complications . Not typical. Sometimes the development of dysbiosis, mesenteric lymphadenitis, exacerbation of the course of gastric and duodenal ulcers.

Diagnosis is based on the detection of helminth eggs in the feces. In connection with the change of generations of helminths, eggs are released inconsistently. The periods of their release alternate with pauses. Therefore, faeces are examined three times at intervals of 15-20 days. In case of weak invasions, it is recommended to prescribe the patient *niclosamide* in a reduced dose (0.5 - 1,0 gr) together with a 1 grlaxative (purgen) in the evening on the eve of the study. *Niclosamide* destroys the tapeworm strobila, as a result of which a large number of eggs enter the intestinal lumen and are excreted in the feces. It is advisable to use flotation enrichment methods (Fülleborn, Kalantaryan, etc.)

Differential diagnosis is carried out with other helminthiases that occur with a predominant lesion of the gastrointestinal tract (diphyllobothriasis, teniasis, etc.).

Treatment. Features of the biology of the parasite and the possibility of intra-intestinal autoinvasion require persistent and systematic treatment, the use of not only antihelminthic drugs, but also symptomatic therapy in conjunction with a set of preventive measures.

Specific therapy is with *praziquantel* or *niclosamide* . On the days of treatment, a diet is prescribed with the exception of vegetables and fruits.

The drug of choice is ***praziquantel*** . It is administered orally at a dose of 25 mg/kg once an hour after a meal with a small amount of water. With a massive invasion, a second dose of the drug is recommended after 7-10 days. The efficiency reaches 90%.

Niclosamide is less effective (70%). On the eve of treatment, easily digestible food is used - broth, mashed potatoes, kissels, kefir, ground meat.

Niclosamide inside appoint at the rate of 2-3 g / day: children under the age of 2 years - 0,5 gr, 3-6 years - 1 gr; 6-9 years old - 1,5 gr; 10 years and older - 2 gr. Immediately before taking the drug, it is necessary for the patient to give 1- 2 grdrinking soda. Several treatment regimens with niclosamide have been proposed:

1) 6-7 two-day cycles of treatment with intervals between them of 4-5 days. Niclosamide is prescribed 1 time per day in doses: children 1-2 years old - by 0,3 gr; 3–4 years - 0,5 gr; 5–6 years - 1 gr; 7–10 years - 1,5 gr; 11 years and older, as well as adults -2 gr;

2) three seven-day cycles with intervals between them of 5 days. After 1 month spend anti-relapse fourth cycle. Niclosamide in each cycle, only on the 1st day, is prescribed in the age daily dose, on other days, 0,5 grregardless of age (for children 1-2 years old 0,3 gr). This scheme is considered especially effective in the persistent course of invasion;

3) two four-day cycles with intervals of 4 days. During each cycle, the patient takes the 4 gr drug daily in 4 doses with a 2-hour interval between them. For children aged 3-9 years, the daily dose of niclosamide is 3 gr. On the first day of the first cycle, 2-3 hours after taking niclosamide, a saline laxative is given in order to quickly remove the decay products of helminths. On the days of treatment, vegetables and fruits are excluded from food.

From the very first days of treatment in children with hymenolepiasis, a large number of eggs are excreted with feces. Strict adherence to the sanitary and hygienic regime is necessary to avoid environmental pollution and re-infection.

The prognosis is favorable. The disease often ends spontaneously without treatment.

Prevention. The fight against hymenolepiasis includes a complex of treatment-and-prophylactic and sanitary-hygienic measures.

Therapeutic and preventive measures are aimed at identifying and neutralizing the source of invasion.

Coprological examination is carried out in the following groups:

- children and staff of preschool children's institutions, students of grades 0-4 - 1 time per year;
- children and staff re-entering groups (especially closed ones);
- catering workers and groups equated to them;
- visitors to swimming pools - upon admission;
- inpatients (children's hospitals, infectious diseases, gastroenterological departments) upon admission and according to clinical indications;
- contact persons with the definition of the volume, frequency and timing of the survey on the recommendation of the sanitary and epidemiological service.

Identified infested for the period of treatment are suspended from attending preschool institutions, schools.

The rupture of the transmission mechanism is provided by a set of sanitary and hygienic measures.

Measures for the prevention and control of hymenolepiasis are incomparably simpler and more effective than those for enterobiasis due to the low resistance of pygmy tapeworm eggs in the environment.

The leading place among them is occupied by such as thorough wet cleaning of premises, disinfection of bedding, household items, toys, chamber pots, door handles, washbasin taps, etc. using boiling water, hot iron, soap-cresol mixture, etc.

In the complex of sanitary and hygienic preventive measures, an important place belongs to the hygienic education of children and the inculcation of hygienic skills in them: washing hands before eating, after using the restroom, weaning from the habit of biting nails, taking fingers, toys, pencils in their mouths, etc.

ECHINOCOCCOSIS

Echinococcosis (ICD10 - B67) is a zoonotic biohelminthiasis caused by the larval stage of tapeworm *Echinococcus granulosus* , characterized by a chronic course and development mainly in the liver, less often in the lungs and other organs, solitary or multiple cystic formations prone to expansive growth.

Etiology. The causative agent of echinococcosis is the larval stage of development (finn) *Echinococcus granulosus*. Mature individual is a small cestode 2- 11 mm. Her body has a pear-shaped scolex, equipped with four suckers and a proboscis with two rims of hooks. Behind the neck is a short strobila, which usually consists of three proglottids. The first segment is immature, the second is hermaphroditic, and the third is mature (Fig. 40).

Figure 40. Sexually mature form of *Echinococcus granulosus* .

The mature segment contains the uterus, which has lateral projections and contains from 400 to 800 fertilized eggs with six-hooked oncospheres. Their diameter is 30-36 microns. The eggs are similar in structure to those of other teniids.

Finns (syn.: larvocysts, hydatids) have the appearance of a single-chamber bladder filled with liquid. Their sizes range from a few millimeters to tens of centimeters. The walls of the Finn have two shells: outer (cuticular, layered) and inner (embryonic, germinal). The outer layered shell consists of concentrically arranged plates, chemically similar to hyaline and chitin. The germinal membrane has three zones: cambial (parietal), middle, containing calcareous bodies, and internal - zone of brood capsules. Brood capsules contain protoscolexes, which have screwed proboscis with two rows of hooks and 4 suckers. Inside the Finns,

secondary (daughter) and in them tertiary (granddaughter) larvocysts are often formed, in which brood capsules and protoscolexes can also develop.

Outside, around the echinococcal bladder due to the tissues of the host, a pronounced connective tissue capsule is formed. Between the latter and the cuticular membrane there is a narrow space filled with polymorphocellular infiltrate.

The fluid of the blisters is a secretion product of the germinal membrane and contains the necessary substances for the vital activity of the parasite, as well as metabolic products. The life span of a larvocyst is several years.

Within the species *E. granulosus* , various strains are currently isolated by molecular biological methods, which exhibit different pathogenicity in relation to humans. For example, a highly pathogenic "sheep strain" and a low pathogenic "horse strain".

development cycle. The life cycle in the development of echinococcal tapeworm includes a change of two hosts (Fig. 41).

Figure 41. C scheme of the life cycle of echinococcus (A) and the ways of transmission of echinococcus in synanthropic (B) and natural (C) foci. A: 1 - adult parasite; 2 - ocosphere; 3 - larva (echinococcal bladder); 4 - scolex from the echinococcal bladder. B: 1 - final host; 2 - intermediate hosts; 3 - transfer factor; B: 1 - final host; 2 - intermediate hosts; 3 - transfer factor (according to E.S. Leykina, 1967).

The final owners are dogs and all members of the canine family (wolves, jackals, hyenas, etc.). Life expectancy of echinococcus in the final host is 5-6 months. New segments constantly bud from the neck of the cestode, and the posterior (mature) proglottids break away from the strobilus and are released into the external environment either with feces or actively crawling out through the anus. Crawling over the host's body, they pollute the animal's coat with eggs released from the uterus.

Intermediate hosts (a wide range of mammals, including humans) receive eggs orally. In the small intestine, the oncosphere emerges from the eggs with the help of hooks and penetrates into the blood vessels of the intestine and then through the portal vein to the liver, where most of them are retained, and forms finnous blisters. Some of the oncospheres bypass the liver barrier and enter the heart, from where they pass through the vessels of the pulmonary circulation to the lungs, where they linger. Oncospheres can be carried by blood into any internal human organ (brain, spleen, kidneys, bones, etc.), where they also form a larvocyst.

The definitive hosts become infected by eating the organs of the intermediate host, which contain the echinococcal vesicle. In the small intestine of the definitive host, the membranes of the bladder are destroyed, and the protoscolexes are attached to the mucosa. Growth of segments begins from the neck and after three months sexually mature cestodes are formed. The echinococcal bladder contains a large number of protoscolexes and brood chambers, so a huge number of parasites develop in the intestines of canines (tens of thousands) (Fig. 42).

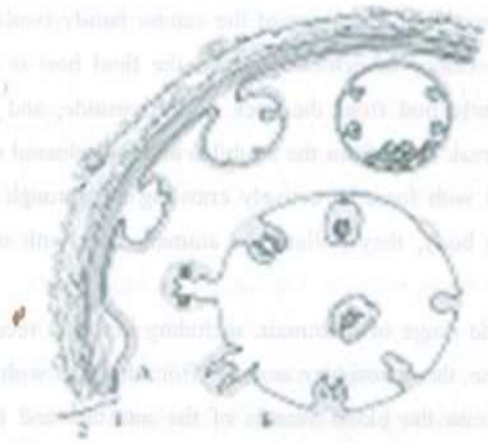

Figure 42. The structure of the wall of the bubble of echinococcus: A - the germinal membrane and the formation of secondary bubbles; B - budding of protoscolexes from the wall of the secondary bladder; 1 - outer (cuticular) membrane of the bladder; 2 - host tissues (according to J. Don ges , 1980).

Epidemiology. Echinococcosis is ubiquitous and distributed throughout the world with varying frequency (from 1 to 220 per 100,000 population per year). The zone of very high endemia includes Argentina, Uruguay, Brazil, Chile, Paraguay, Peru, Mongolia, Pakistan, Afghanistan, Iran and some other regions of Central and Asia Minor. High endemic zones: Tunisia, Algeria, Morocco, Kenya, Somalia, Ethiopia, Sudan, Egypt, Libya, Yugoslavia, Bulgaria, Greece, Italy, Turkey, Cyprus, Spain, Syria and parts of Central and East Asia. In other countries of Europe, North America and the Far East, sporadic cases of human echinococcosis are noted. For Uzbekistan, echinococcosis is a regional pathology that poses a serious problem for public health.

The source of invasion for humans are infected animals: in nature - carnivores (foxes, wolves, jackals, etc.), in synanthropic foci - dogs.

A person becomes infected when removing and dressing the skins of wild carnivores, contact with infected dogs, eating unwashed berries, herbs and

vegetables from gardens visited by infected dogs, when using water from contaminated water sources. Therefore, echinococcosis is more common in hunters and pastoralists, especially when dogs are fed the thermally untreated entrails of infected domestic herbivores. Large and small cattle can be a mechanical carrier of echinococcus eggs; they get on animal hair from the grass of polluted pastures, so you need to wash your hands thoroughly after milking cows and shearing sheep.

Pathogenesis. Once in the human gastrointestinal tract, parasite eggs lose their outer shell under the action of digestive enzymes. The released oncosphere with the help of hooks penetrates into the thickness of the mucous membrane of the stomach or intestines. From there, with the flow of venous blood or lymph, the oncosphere is transferred to the portal system, and is retained in the liver. Part of the oncospheres passes through the liver filter and through the inferior vena cava enters the pulmonary circulation, lingering in the lungs. An insignificant part of the oncospheres, penetrating through arteriovenous anastomoses, enters the systemic circulation, and can be introduced into any organ or tissue of the body (kidneys, abdominal cavity, brain and spinal cord, bones, etc.).

The oncosphere settled in the tissues for 8-15 months. turns into a larva - an echinococcal cyst with a diameter of 5- 20 mm, depending on the structure of the tissues in which it develops. In the lung tissue, parasitic cysts grow faster than in the liver and bones.

In the affected organ, one cyst (solitary lesion) or several (multiple echinococcosis) may develop. Multiple organs may be affected at the same time. Cysts vary in size from 1 2 cmto giant, containing several liters of fluid. Daughter cysts may develop inside the cyst.

A zone of necrosis is formed around the growing cyst, a shaft of cellular inflammatory reaction with a large number of eosinophils. Gradually, the area of inflammation is replaced by scar tissue - a fibrous capsule is formed. The echinococcal cyst grows expansively, pushing away the tissues of the affected organ. The mechanical effect on the tissues, exerted by the growing echinococcal

cyst, leads to atrophy of the adjacent parts of the organ and its pronounced deformation.

Of great importance is the sensitization of the host organism by the products of the metabolism of parasitic antigens, which can sometimes cause intoxication and allergic reactions. When the larvocyst ruptures, the contents may leak into the abdominal or pleural cavity, break into the bronchi, bile ducts or large vessels, leading to severe anaphylactic shock, seeding of other organs and the development of secondary echinococcosis.

The basis of the immune response in echinococcosis is the reaction of lymphoid tissue to the antigens of the pathogen, which is manifested by the production of antibodies. Human defense mechanisms, among other things, are manifested by the construction of a connective tissue capsule (pericyst) around the cyst of the parasite (terminal cyst).

Calcification can develop in the connective tissue capsule (pericyst) over the years, which is well detected by ultrasound and computed tomography. Often calcifications are found in cysts of the liver or cysts of other parenchymal organs, rarely in pulmonary cysts.

Clinic. The disease is usually detected in middle-aged people, but cases of the disease in children under 5 years of age are not uncommon.

The latency period can vary from a few months to decades. Sometimes the whole process is asymptomatic, and Echinococcus hydatida may be accidentally detected during instrumental examination or during surgery for another reason.

In the clinically pronounced stage of echinococcosis, the symptoms, course and prognosis are determined by the volume of the parasitic lesion, the localization and rate of development of cysts, and the reactivity of the host organism. Pregnancy, severe intercurrent diseases, immunodeficiency states, and nutritional disorders contribute to a more severe course of the disease.

The first symptoms of the uncomplicated stage are usually non-specific. Symptoms of general intoxication and allergic reactions predominate. Patients

complain of weakness, malaise, decreased performance. Sometimes there are dyspeptic phenomena; possible fever and the appearance of itchy urticarial rashes.

With liver echinococcosis, the pain syndrome is similar to that with cholecystitis. There is weight loss, loss of appetite, heartburn, belching, vomiting. An increase in the liver is detected, with superficial localization of the cyst, it can be palpated (Fig. 43).

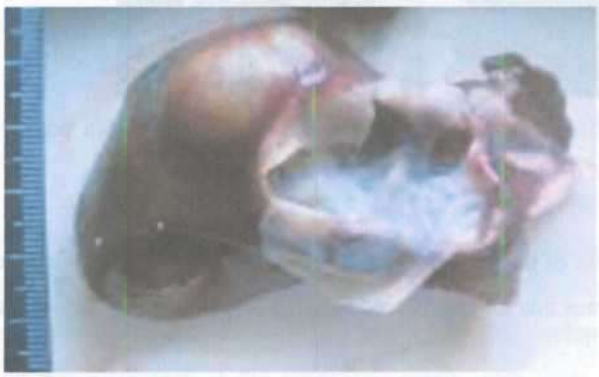

Figure 43. Echinococcal cysts of the liver.

Growing echinococcal blisters in the liver compress the large bile ducts, causing obstructive jaundice

Manifestations of echinococcosis of the lungs are determined by the localization of the cyst. Even a small cyst located near the pleura early manifests itself as a pain syndrome; when it is localized near the bronchial trunk, chest pain, dry cough, hemoptysis appear. Often, uncomplicated lung echinococcosis is detected by chance during an X-ray examination (Fig. 44).

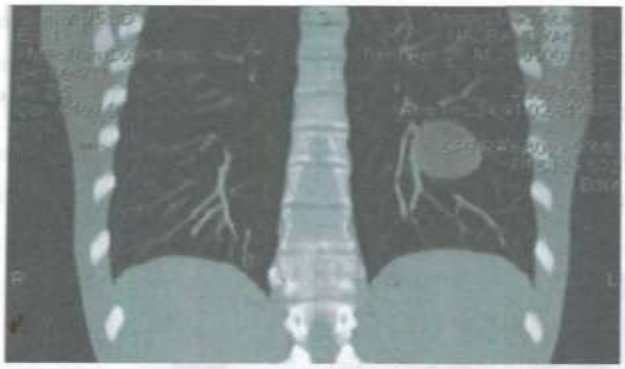

Figure 44. Computed tomography of the chest cavity of a patient with echinococcal cyst of the left lung.

Echinococcosis of the kidneys is often diagnosed only when echinococcosis is detected, sometimes this may be preceded by pulling pain in the lumbar region, dysuric disorders.

Echinococcosis of bones is manifested by pain and swelling in the affected area (Fig. 45).

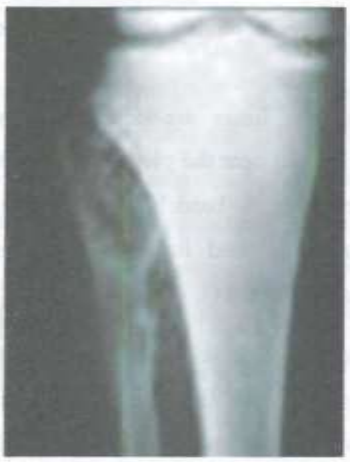

Figure 45. Echinococcosis of the tibia (X-ray).

The symptomatology of other localizations of echinococcosis (brain, mediastinum, mammary gland, intestines) corresponds to the symptoms of a volumetric lesion of one or another organ.

Complications with echinococcosis are common (30%), sometimes being the first clinical manifestation of the disease.

With echinococcosis of the liver, suppuration of the cyst may develop. At the same time, there are sharp pains in the hypochondrium, sometimes jaundice, fever, hyperleukocytosis, an increase in ESR. Jaundice develops when large bile ducts are compressed by cysts. Portal hypertension is rarely observed when the main vessels of the portal tract are compressed by large cysts.

Rupture of an echinococcal cyst is the most formidable complication of echinococcosis. It is accompanied by sharp pains, an allergic reaction, up to anaphylactic shock, sometimes with a fatal outcome. The result of rupture of a viable cyst is the dissemination of the pathogen, the development of secondary multiple echinococcosis of the abdominal cavity, sometimes the retroperitoneal space and other organs (Fig. 46).

Figure 46. Multiple echinococcosis of the lungs, hematogenous seeding.

When the lungs are damaged, complications arise:

- suppuration of the cyst. Accompanied by a lung abscess clinic - fever, chest pain, cough, high leukocytosis, neutrophilia, stab shift, lymphopenia,

increased ESR. At the opening of a festering cyst, purulent pleurisy develops in the pleura;

- compression of the cyst of the bronchus, blood vessels, which causes a persistent cough, hemoptysis;

- opening of the cyst in the lumen of the bronchus. It is accompanied by an attack of painful coughing, the release of light sputum and the discharge of cyst membranes in the form of translucent films ("bulb peel symptom"). The result of rupture of a viable cyst containing scolexes is the dissemination of the parasite with the development of secondary multiple echinococcosis of the lungs, sometimes the pleura.

Diagnosis is based on the data of radio-radiological, ultrasound and immunological studies.

Radiation (X-ray, ultrasound), radioisotope methods (scanning, scintiography), computed tomography, magnetic resonance imaging allow us to assess the localization and prevalence of the process.

With ultrasound, an echinococcal cyst of the liver is detected as a rounded formation with dense, sometimes calcified walls.

An uncomplicated hydatid cyst of the lungs appears as a rounded shadow with clear contours. When opening a cyst in the bronchus and its partial drainage, a rounded shadow with a liquid level is visible, sometimes the wall is bicontour due to detachment of the chitinous membrane.

CT and MRI can reveal the nature of the lesion, size, topography, complications.

Laparoscopy is of limited value, since the information content of the method is low, and the risk of complications is high. Possible violation of the cyst wall with the development of complications: anaphylactic reaction, dissemination of the pathogen.

Morphological diagnosis is possible in the study of surgical or sectional material. Sometimes the diagnosis can be established microscopically by detecting

protoscolexes and their fragments (hooks) in biological fluids and substrates (Fig. 47).

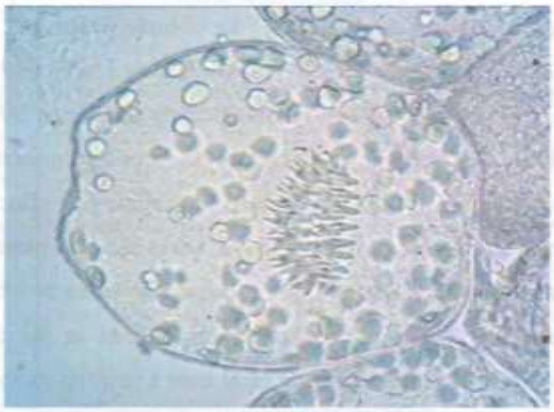

Figure 47. Echinococcus protoscolex.

When cysts break through into the lumen of hollow organs, fragments of the cyst can be found in sputum, duodenal contents, and feces.

Serological methods are highly informative, allowing the detection of specific antibodies to the *E antigen* . *granulosus* . Enzyme-linked immunosorbent assay (EIA), immunoblotting, etc. are used. Biochemical and general clinical results of a blood test with a latent course of echinococcosis remain within the normal range.. Their changes are observed only with the development of complications.

differential diagnosis. Echinococcosis must be differentiated from neoplasms, polycystosis, hemangioma and other focal lesions, including alveococcosis. Often, the initial manifestation of liver echinococcosis is regarded as hepatitis, liver cirrhosis, hepatocholecystitis. Echinococcosis of the lungs and other organs requires differentiation from tuberculosis, neoplasms, systemic mycoses, etc.

Treatment . The main treatment for echinococcosis is surgery. During the operation, all precautions must be taken to remove the cyst without violating its integrity.

For anti-relapse therapy and conservative treatment, ***albendazole is used .***

In the postoperative period, *albendazole* is prescribed inside adults 0.4 twice a day (children over 2 years old 10-15 mg / kg of body weight per day, not more than 800 mg / day) immediately after a meal containing a sufficient amount of fat (at least 30 g). The duration of anti-relapse therapy is at least 90 days without interruption. After 5-7 days, it is necessary to conduct general clinical and biochemical (bilirubin, ALT, AST) blood tests to exclude side effects of the drug. In the future, these studies are carried out in 2-3 weeks and then monthly.

Conservative therapy is carried out in inoperable cases or when the size of the cysts is less than 4 cm. Albendazole is prescribed in the same dosages, however, the duration of treatment should be at least a year (up to 5 years or more). The duration of therapy is determined in each case individually and depends on the degree of changes occurring in the parasitic cyst, as well as on the dynamics of the antibody response.

With the development of purulent complications, antibiotic therapy is carried out in the amount of therapy for abscesses of the liver and lungs.

The prognosis is serious due to possible complications and relapses. With timely diagnosis of single echinococcal blisters localized in the liver and lungs, and their adequate treatment, favorable outcomes are frequent. The rupture of the echinococcal bladder with the development of anaphylactic shock poses a threat to the life of the patient.

Prevention. Careful observance of the rules of personal hygiene when caring for animals, picking berries. Scheduled deworming of dogs, culling and destruction of internal organs of domestic animals containing echinococcal cysts are carried out. In places unfavorable for echinococcosis, a planned medical examination of the population is necessary.

TYPE ROUND WORMS

ASCARIDOSIS

Ascariasis (ICD10 - B77) - intestinal helminthiasis caused by roundworms, occurring with symptoms of lesions of the gastrointestinal tract, intoxication, allergic reactions.

Etiology. The causative agent is the human roundworm *(Ascaris lumbricoides)*. It is a light pink roundworm. The mouth opening is surrounded by three cuticular lips, the caudal part of the male's body is bent to the ventral side (Fig. 48).

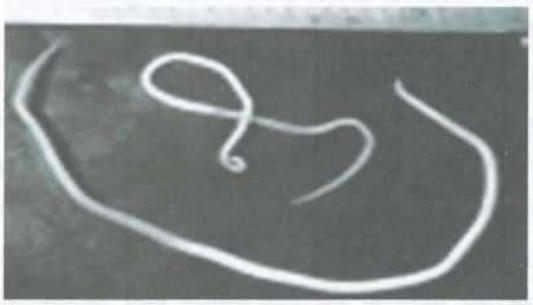

Figure 48. *Ascaris lumbricoides* . Above is the male, below is the female.

The length of females is 25- 30 cm, males - 12- 15 cm.

The eggs are gray-brown in color, covered with a thick multi-layered tuberous shell, oval in shape, size - 50-70 microns. (Fig. 49).

Figure 49. A fertilized roundworm egg.

Life cycle. Mature individuals live in the small intestine. Males fertilize females. Every day, the female lays up to 200 thousand eggs, which leave the intestines with feces. The female can also lay unfertilized eggs (in the absence of males).

The development of the larva in the egg occurs only in the external environment, after which the egg becomes invasive. Under favorable conditions (temperature +25 ° C, the presence of oxygen and sufficient humidity), the formation of the larva is completed within 10-12 days. With fluctuations in temperature and humidity levels (but not less than 8%), the timing of larval development varies from 42 days at a temperature of 13.5 ° C to 9 days at 30 ° C.

Next, the egg must enter the human body, which usually occurs along with contaminated food. In the small intestine, a larva emerges from a swallowed egg, which penetrates the blood vessels of the intestinal walls and begins a complex migration: intestinal vessels → liver → inferior vena cava → right heart → pulmonary artery → lung tissue. Here they destroy the wall of the capillary, ending up in the lumen of the alveolus, where they continue to develop. There they molt twice and increase in size by 8-10 times, from 0.2 0,3 mmto 1.5-2.1 mm, feeding at

this time on blood plasma and red blood cells. After 14 days, the larvae begin to ascend through the airways of the lungs. Their movement speed is very slow. By irritating the respiratory tract receptors, they cause an unconditioned cough reflex. Thanks to coughing, they quickly rise up the bronchi, trachea, enter the larynx, nasopharynx, and when coughing up sputum, they end up in the oral cavity, and from there, together with saliva, they can be swallowed, again ending up in the intestines, where they reach puberty. After 10-11 weeks from the moment of invasion, the female begins to produce eggs, which are excreted with feces into the external environment. The life span of roundworms in the human body does not exceed one year, then they die and are excreted in the feces.

Epidemiology. Ascariasis is one of the most common helminthiases; about 1.4 billion people are affected by it on the globe. On a global scale, foci of ascariasis do not form only in the tundra zone and in the zone of dry deserts and steppes, where artificial irrigation is not used to moisten the soil. Cases of this disease are also registered on the territory of Uzbekistan.

Ascariasis is anthroponotic geohelminthiasis, the source of infection is only an infested person. The eggs excreted in the feces are non-invasive, so infection is impossible even with close household contact. Eggs isolated with feces reach infectivity, developing in the soil (Fig. 50).

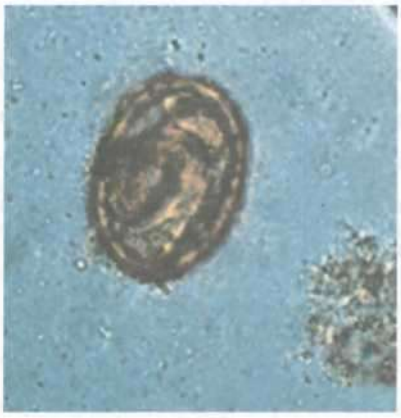

Figure 50. Mature roundworm egg.

Favorable conditions are necessary for their development: optimal temperature (+18–30°C), sufficient humidity and free access of oxygen.

The shell of the eggs is multi-layered, which makes them resistant to various external influences. The outer protein and adjacent carbohydrate layers provide mechanical protection, the inner fat-like layer makes the shell impenetrable to many poisons. Eggs remain viable for months in 3% formalin, 15% H_2SO_4, saturated sublimate solution. In the external environment, they can remain viable for up to 7 years. Ultraviolet rays, high temperature have a detrimental effect on the emerging larvae (at +50 ° C they die within 15 minutes, at +70 ° C - within one second).

The transmission mechanism is fecal-oral. Human infection occurs as a result of ingestion of invasive ascaris eggs when eating unwashed berries (strawberries), vegetables (carrots, radishes, green onions, cucumbers, tomatoes), table greens, as well as through contaminated hands after working in the garden, playing children in gardens. plots, etc. Infection with ascariasis can occur throughout the year, but more often in the summer-autumn period. The most vulnerable groups of the population are children, workers of sewage treatment plants, people involved in crop production, and diggers.

Pathogenesis. There are 2 phases of ascariasis: early - migratory and late - intestinal .

The pathogenetic effect of ascaris larvae at the early (migratory) stage of the disease is due to injuries of various tissues along the movement of the larvae, as well as sensitization of the body by the products of their metabolism. In places of perforation by tissue larvae, hemorrhages are formed; eosinophilic infiltrates form in the lungs, liver and intestinal walls (Fig. 51).

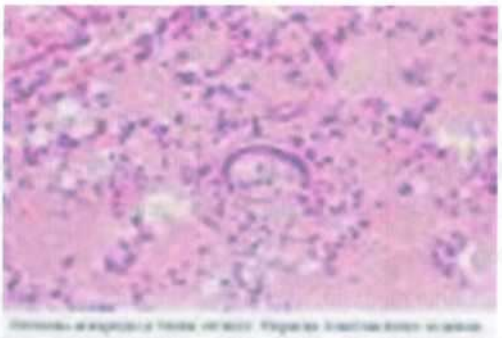

Figure 51. Ascaris larva in lung tissues.

The habitation of sexually mature worms in the intestine at the late stage of ascariasis leads to morphological changes (hypertrophy of the muscular layers of the intestinal wall) and physiological changes (increased intestinal motility, deterioration of absorption and assimilation of proteins, fats; the formation of protein deficiency, deficiency of vitamins A, C). In the small intestine, lactase activity decreases with the development of milk intolerance. Metabolic products of helminths have a toxic effect on the nervous system. Violation of the neuromuscular regulation of the intestine, as well as the closure of its lumen with a ball of ascaris, can lead to intestinal obstruction. Adult helminths are very mobile and able to migrate to the liver, pancreas and other organs, creating conditions for the attachment of bacterial infections and the development of complications of a purulent nature.

The clinic of ascariasis depends on the localization of helminths and the intensity of the invasion. In the clinical course of the disease, two phases are distinguished - early, coinciding with the period of migration of larvae, and late (chronic), due to parasitism of helminths in the intestine.

Early stage of ascariasis at a low intensity of infection, it can be asymptomatic. In clinically expressed cases, there are complaints of general weakness, malaise, fatigue, sometimes an increase in body temperature, the

appearance of itchy rashes on the skin like urticaria. Allergic phenomena last about two weeks, and then subside.

In massive infestations, migrating larvae can cause clinical manifestations of acute pneumonia or bronchitis. There is a cough with an asthmatic component, sometimes with sputum mixed with blood, shortness of breath, chest pain. In some cases, the clinical picture of the migratory stage resembles the course of bronchial asthma or respiratory allergy.

An x-ray examination in the lungs reveals eosinophilic infiltrates, characterized by variability in their size, configuration, position - "flying infiltrates". In the general blood test - eosinophilia up to 35-60%.

Often there is an increase in the liver, spleen, peripheral lymph nodes, dysfunction of the cardiovascular system in the form of a decrease in blood pressure, tachycardia.

The late (intestinal) stage usually proceeds latently. Clinically, the disease manifests itself with a high number of parasites in the intestine. In these cases, signs of damage to the gastrointestinal tract and asthenic syndrome are revealed. Patients complain of a decrease or perversion of appetite, nausea, increased salivation - more often in the morning, on an empty stomach, weight loss. In some cases, there is a tendency to diarrhea or constipation, bloating, less common progressive enteritis. Dyspeptic phenomena may be accompanied by cramping pains in the abdomen.

Pain is noted on palpation of the abdomen. In children, it is often diffuse, in adults it is local, along the midline above the navel, in the ileocecal region, to the right of the midline.

Asthenovegetative disturbances, such as weakness and poor health, are almost always noted. Sometimes there is the development of fainting, increased irritability, headaches, absent-mindedness, memory loss, restless sleep, night terrors, twitching. Less commonly - hysterical and epileptic seizures, Meniere's symptom complex, decreased intelligence.

Complications. One of the most serious complications is intestinal obstruction, which is formed due to reflex spasm or blockage of the intestinal lumen with a ball of ascaris (Fig. 52). Penetration of ascaris into the appendix sometimes causes appendicitis.

Figure 52. Blockage of the human intestine by a ball of ascaris (according to K.T. Ovnatanyan).

With destructive changes in the intestinal mucosa caused by various pathological processes (ulcerative colitis, dysentery, typhoid fever, balantidiasis) or after surgical interventions, ascaris may perforate the intestinal wall and penetrate into the abdominal cavity, leading to peritonitis.

Ascaris crawling into the hepatic tract or their larvae entering the liver lead to the development of biliary or hepatic ascariasis. The liver increases, jaundice, fever appears, attacks of hepatic colic occur. It is also possible the development of ascariasis abscesses of the liver and even its ruptures with the subsequent development of peritonitis. The penetration of ascaris into the ducts of the pancreas can cause an attack of acute pancreatitis. When ascaris enters the respiratory tract, their obturation and the development of asphyxia are possible. When ascaris enters the paranasal sinuses of the skull, sinusitis can develop. In rare cases, due to the action of allergic substances secreted by roundworms on the central nervous system, encephalopathies develop (asthenic syndrome, emotional disturbances and delirium).

There are known cases of disseminated ascariasis, when adult helminths were found in the liver, lungs and heart, which led to serious dysfunction of these organs and even death.

differential diagnosis. In the migratory stage, it is carried out with the migratory stage of other helminthiases, toxocariasis, acute bronchitis, pneumonia. In the intestinal stage, the differential diagnosis is carried out with the development of complications.

Diagnostics. It is impossible to establish the diagnosis of ascariasis only on clinical data. Suspicion of ascariasis invasion in the migratory stage of the development of the disease may arise when migrating infiltrates are detected in the lungs, a change in the position of which is detected by comparing radiographs taken at intervals of several days. Infiltrates are both single and multiple. They can occur in one lobe or throughout the lung. Their shape is varied (round, oval, star-shaped, scalloped), the contours are vague. In the first 2-3 weeks after infection, eosinophilic infiltrates are detected.

The combination of "flying" infiltrates with eosinophilia in the blood, which is observed in 60% of cases, is a strong basis for the assumption of ascariasis invasion. This assumption is strengthened when the patient develops allergic phenomena: urticaria, skin itching, Quincke's edema, etc. Leukocytosis is characteristic of the early stage of ascariasis (with massive invasion it is most pronounced).

In the migratory phase of ascariasis, a parasitological diagnosis is rarely established. Casuistic findings of roundworm larvae in sputum are described (Fig. 53).

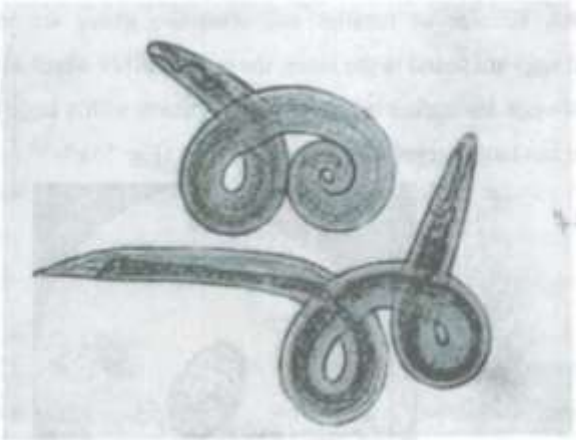

Figure 53. Roundworm larvae isolated from sputum (V.P. Podyapolskaya, V.F. Kapustin, 1958)

In the late (intestinal) phase, the parasitological diagnosis is established when ascaris eggs and sometimes the parasites themselves are found in the feces. In rare cases, roundworms can come out through the mouth and even through the nose.

Most often, fertilized ascaris eggs are found in the feces. They are oval in shape, covered with a large-tuberous surface shell of brown or dark yellow color. Occasionally there are eggs without a protein shell. They are colorless, transparent, resemble plant cells and are difficult to determine (Fig. 54).

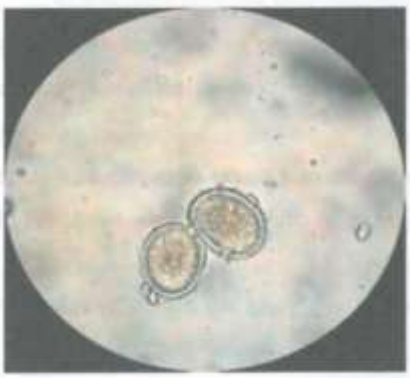

Figure 54. Ascaris eggs with unformed protein coat.

If only females or females and immature males are in the intestines, unfertilized eggs are found in the feces, the outer shell of which is thinner than that of fertilized eggs. Its surface is unevenly hilly; along with a large number of small tubercles, it has large irregularly shaped tubercles (Fig. 55).

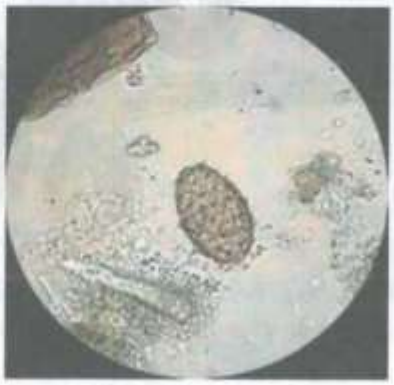

Figure 55. Unfertilized roundworm egg.

When parasitizing in the intestines of only males, immature or old females, there are no eggs in the feces.

Immunological methods in the diagnosis of ascariasis are uninformative and require parasitological confirmation.

In some cases, it is not possible to conduct a clinical differential diagnosis of the intestinal phase of ascariasis with a number of diseases of the gastrointestinal tract.

Treatment. All patients with ascariasis are subject to treatment. On the eve of treatment, cabbage, peas, smoked meats, and fatty meat are excluded from the diet. Fruits are replaced with juices.

The effectiveness of anthelmintic drugs after one course of treatment is more than 90%. Currently, the following drugs are most often used in the treatment of ascariasis:

Albendazole, adults and children over 2 years old - 0.4 g / day once 1.5 hours after a light breakfast.

Mebendazole , adults and children over 2 years old - 100 mg twice a day for 3 days (for a course of 600 mg).

Karbendatsim - adults and children at the rate of 10 mg / kg / day in three divided doses for 3 days.

Pyrantel pamoate - once 10 mg / kg (maximum dose for adults - 750 mg).

Pathogenetic and symptomatic therapy, as a rule, is not required. However, with prolonged intensive invasion, it is advisable to prescribe enzyme preparations and probiotics.

From the 2nd day after the start of treatment, dead roundworms can be excreted in the stool. However, with the use of modern preparations, roundworms can be destroyed in the patient's intestines and not be detected in the feces.

In chronic diseases of the gastrointestinal tract (peptic ulcer, ulcerative colitis, Crohn's disease, pancreatitis, acute and chronic hepatitis), treatment is carried out in the stage of remission of the underlying disease.

In cases of complicated ascariasis (retrograde migration of ascaris into the biliary tract, liver, respiratory tract), surgical or instrumental intervention is required.

The prognosis is favorable.

Prevention is aimed at identifying and treating infested people, protecting the soil from fecal contamination and conducting sanitary and educational work among the population.

In intensive foci, the first deworming is carried out in late spring, the second - in late autumn.

It is unacceptable to fertilize the soil of vegetable gardens, berry plots with untreated animal and human feces. The use of such sources of fertilizer is possible only after composting. In the center of the "compost heap" the temperature rises to 80 ° C, which is detrimental to roundworm larvae in the egg. Measures that provide for sanitary improvement are important: sewerage, dry closets.

Sanitary and educational work includes the formation of personal prevention skills: washing hands before eating, eating only well-washed fruits, berries, and vegetables that are not subjected to heat treatment.

TRICHOCEPHALOSIS

Trichuriasis (ICD10 - B79) is a human intestinal helminthiasis caused by whipworm, and occurring with a predominant lesion of the gastrointestinal tract.

Etiology. The causative agent is whipworm *(Trichocephalus trichiurus)* - so named due to the fact that the front 2/3 parts of the body are thread-like. Only the esophagus is located in this thinned part of the body. In females, the posterior expanded part of the body is curved in the form of an arc, in males it is spirally twisted to the ventral side. The size of females and males of this species is from 3 to 5 cm, males are always shorter (Fig. 56).

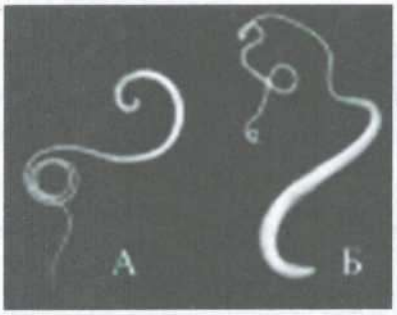

Figure 56. Vlasoglavy. Male (A) and female (B).

The eggs resemble a barrel in shape, they have transparent corks at the poles, the length of the eggs is up to 50 microns (Fig. 57).

Figure 57. Whipworm egg.

Vlasoglav parasitizes in the large intestine, mainly in the caecum, but with massive invasions, helminths can also be found in the distal parts of the large intestine.

Almost the entire front part of the body of the whipworm is immersed in the mucous layer, as if stitching it, and only the expanded tail part remains on the surface. Due to this arrangement, the whipworm feeds on tissue sap and epithelial cells, optionally on erythrocytes (Fig. 58).

Figure 58. Whipworms attached to the intestinal wall (V.P. Podyapolskaya, V.F. Kapustin, 1958)

Life cycle. Whipworms parasitize in the large intestine, mainly in the caecum. After mating, females begin to lay eggs, which, along with feces, enter the environment. At a temperature of 26-28 ° C, access to oxygen and sufficient humidity, the eggs become invasive after 3 weeks.

Eggs with food and water enter the small intestine, where larvae emerge from them, which penetrate the villi of the mucous membrane, where they stay for up to 10 days. Then they re-enter the intestinal lumen and move to the caecum, where they attach to the mucosa, reaching the sexually mature stage in about a month. Eggs first appear in faeces 12 weeks after infestation. One female lays about 5000 eggs per day. The life expectancy of the whipworm is 5-6 years, according to some sources - 5-10 years.

Epidemiology. Trichuriasis is an anthroponotic geohelminthiasis, which is widespread in the world, mainly in regions with a warm climate. Humans are the only source of infection. Eggs excreted in the feces must be incubated in the soil for 17-25 days. The most favorable conditions for the development of eggs are created at a temperature of +26°C - +30°C, air humidity close to 100%, soil moisture 18-22%, and the presence of oxygen. The larva matured in the egg has an annular stylet. In the external environment, whipworm eggs remain viable in the soil for 1-2 years, in a temperate climate they can overwinter under a thick layer of snow.

Humans become infected by ingesting infective eggs. Transmission factors of the pathogen are vegetables, berries, table greens, eaten without heat treatment. Infection can also occur through soil-contaminated hands. The most vulnerable groups of the population are children aged 5-14, adults involved in working with land, fertilizers, workers in sewer networks, treatment facilities, and sewage transport.

Pathogenesis. With a low intensity of invasion, the disease occurs more often subclinically. With intensive invasion (hundreds of individuals), a week after infection, due to parasitism of the larvae, an eosinophilic inflammatory reaction of the mucosa occurs. During this period, a large number of eosinophils and Charcot-

Leiden crystals are found in the intestinal exudate. After the development of the larvae is completed, these phenomena subside. Later, as a result of mechanical damage to the mucous membrane, young helminths and the action of their metabolic products cause hyperemia, edema, infiltration, erosion, hemorrhage and small-focal necrosis of the mucous membrane. Due to traumatization of the intestinal wall, conditions are created for the penetration of bacterial flora and the development of bacterial gastroenteritis. Irritation of the nerve endings of the ileocecal region entails a violation of the motor and secretory functions of the stomach, duodenum, biliary tract, inhibition of the formation of pepsin, which provokes the development of pain in the epigastric region, simulating peptic ulcer.

The helminth feeds on epithelial cells and intercellular fluid, but can absorb red blood cells (facultative hematophage). Each helminth is able to absorb up to 0.005 ml of blood per day. With intensive invasions (200 individuals in children and 500-800 in adults), anemia may develop.

Clinic. With a low intensity of invasion, trichocephalosis proceeds subclinically. With moderate (200 parasites) and intensive invasions (200-1000 or more parasites), symptoms of damage to the gastrointestinal tract and nervous system are observed. There is a decrease in appetite, nausea, vomiting, salivation, flatulence. Patients note pain in the right ileocecal region, resembling pain in appendicitis, intense pain in the epigastric region, simulating the clinic of gastritis, peptic ulcer. Often there is an unstable stool or moderate diarrhea associated with impaired absorption of water and irritation of the interoreceptors. Patients often complain of headache, dizziness, irritability, poor sleep, rarely fainting, convulsive seizures.

The course of trichocephalosis is significantly aggravated by the layering of a bacterial intestinal infection or protozoal invasion. The patient develops hemocolitis, tenesmus, pain all over the abdomen, and body weight decreases.

Hypochromic anemia that develops with trichocephalosis is manifested by weakness, dizziness, shortness of breath on exertion, palpitations, tachycardia, lowering blood pressure, and pallor of the skin. Blood parameters rarely change,

with intense invasions, hypochromic anemia, moderate leukocytosis, and moderate eosinophilia may be noted.

With prolonged parasitism, there are signs of vitamin deficiency - dry mucous membranes, brittle nails, hair. A decrease in the overall reactivity of the body is accompanied by the addition of bacterial infections, exacerbation of chronic diseases.

Invasion lasts 5-7 years with a single infection. In process of release from parasites in connection with the termination of term of their life clinical manifestations subside. In some cases, the period of recovery of impaired functions lasts several months.

Features of trichuriasis in children. In contrast to adults, children with trichocephalosis often have CNS lesions. Sick children lag behind in physical and mental development, become inattentive, sleep poorly, and have reduced appetite. Young children become capricious, whiny, dystrophy, more often suffer from respiratory infections. In the general blood test, as a rule, anemia of one degree or another is recorded. According to some reports, post-vaccination immunity in children with trichuriasis is poorly formed and does not protect against infectious diseases.

Complications rarely develop. Penetration of whipworms into the appendicular process sometimes leads to appendicitis. Anemia, cachexia, intestinal dysbiosis may also develop.

Diagnostics. The diagnosis is established in case of detection of whipworm eggs in the study of feces. Ether-formalin and other enrichment methods are used (Kalantaryan and others). With a low intensity of invasion, repeated studies of feces are required.

Differential diagnosis is carried out with gastroenterocolitis of any etiology, other nematodes, amoebiasis, balantidiasis, peptic ulcer, ulcerative colitis, Crohn's disease.

Treatment of trichuriasis is carried out on an outpatient basis. Hospitalization is required for patients with complicated forms of invasion (anemia, malnutrition, hemocolitis).

The effectiveness of anthelmintic drugs after one course of treatment is 65-95%. Currently, 3 main drugs are used.

Albendozol inside in 20-30 min. after a light breakfast; children 2-12 years old - 10 mg / kg, over 12 years old and adults - 400 mg once.

Mebendazole inside 20-30 minutes after eating; children 1-5 years old - 100 mg 1 time per day, children over 5 years old and adults 100 mg 2 times a day. The course of treatment is 3-5 days.

Karbendatsim - adults and children over 2 years old 10 mg / kg / day in 3 doses every 30 minutes. after meals for 3 days.

Restoration of the intestinal microflora and correction of the function of the gastrointestinal tract is carried out by prescribing probiotics (linex, bifiform, normobact, bifidumbacterin-forte) and enzyme preparations (pancreatin, mesimforte, creon).

With the development of anemia, in some cases, iron preparations and B vitamins are needed.

The prognosis is favorable.

In the **prevention of** trichocephalosis, the sanitary improvement of settlements is of great importance. Use of feces for soil fertilizer only after composting. Prevention of invasion is achieved by observing the rules of personal hygiene, developing hygiene skills in children, eating only thoroughly washed fruits and vegetables, boiled water.

ENTEROBIOSIS

Enterobiosis (ICD10 - B80) is an intestinal helminthiasis caused by pinworms, characterized by an erased and unexpressed course, the most common

symptom of which is perianal itching that occurs at the site of egg laying by sexually mature females.

Etiology. The causative agent of enterobiasis is the pinworm *(Enterobius vermicularis)* . This is a small white worm, spindle-shaped with pointed ends. The length of males is 2- 5 mm, females - 8- 13 mm(Fig. 59).

Figure 59. Pinworms. Males and females.

In a mature state, females increase significantly in diameter, as they contain 5000-17000 eggs. The tail part of the male's body is twisted to the ventral side. The mouth opening is surrounded by a vesicle that forms small cuticular wings. On the esophagus there is an expanded posterior bulb - bulbus .

The eggs are transparent, oval, slightly asymmetrical, 50-60 microns in size. In mature eggs, you can see an already formed larva (Fig. 60).

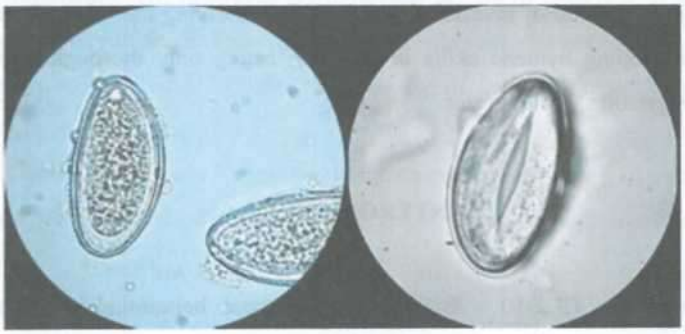

Figure 60. Pinworm eggs: immature (left) and mature.

Life cycle. After swallowing, larvae emerge from the eggs in the ileum. In the terminal part of the intestine, they enter the crypts, where they attach to the mucous membrane of the caecum and molt twice. Fixation occurs due to the tight pressing of the head of the vesicle to the host tissues and the suction action of the esophageal bulb. 12-14 days after infection, pinworms reach the stage of puberty. They feed on the contents of the intestine, sometimes they swallow red blood cells that have got there from damaged areas of the mucosa. After copulation, males die, and females begin to produce eggs that accumulate in the uterus. Over the next two weeks, fertilized females descend into the rectum. Overloaded with eggs, they are unable to stay on the mucosa. At night, when the sphincters are relaxed, the pinworms exit through the anus and lay their eggs on the skin of the perianal folds and perineum. Embryos in laid eggs develop rapidly. At a human skin temperature of 35 ° -36 ° C, sufficient humidity and oxygen access, the eggs become infective after 4-7 hours. The eggs are covered with a secretion that causes irritation of the chemoreceptors of the skin, which is manifested by itching. When combing itchy areas of the skin, invasive eggs fall on the hands, and then into the mouth. Eggs spread through underwear and bed linen, settle on the surface of a variety of objects. With this helminthiasis, re-infection very often occurs - reinvasion.

After laying eggs, the females die. Life expectancy is 3-4 weeks.

Epidemiology. Enterobiosis is a contagious helminthiasis. This is the most common helminthic invasion in the developed countries of the world. The share of enterobiasis among other helminthiases reaches 70-95%. The life cycle of the pinworm does not depend on the climatic conditions of the area, therefore enterobiasis is common in all latitudes of the globe, including Uzbekistan. Children in preschool institutions and schools are especially often infected. In adults, enterobiasis is found mainly at the age of 30-39 years in families with children.

Humans are the only source of pinworm infestation. The route of infection is oral. Enterobiosis is considered a disease of dirty hands. Because of the unbearable itching associated with the crawling out of the female pinworms from the rectum,

the infested person soils the hands when scratching. Subsequently, helminth eggs are introduced to other parts of the body - the skin of the abdomen, thighs, face, fall on underwear and bed linen, and when it is shaken, they rise with dust and settle on household items, creating conditions for self-infection of the patient and widespread invasion of others. When examining various objects of the external environment, pinworm eggs are found on dishes, toys, tables, desks, money, food products. Flies play a role in the spread of pinworm eggs. On human skin, underwear, pinworm eggs mature within 4-7 hours to the invasive stage. The optimal temperature for their development is 35-37°C, while they can develop at temperatures from 23°C to 40°C.

In the external environment, helminth eggs survive for 2-3 weeks. They are sensitive to high and low temperatures. At a temperature of -15°C, they remain viable for no more than 40-50 minutes, at a temperature of +55°C and above - a few seconds. The main factors of infection transmission are hands, toys, food products. It is possible to infect children in swimming pools. The possibility of invasion when eggs enter the gastrointestinal tract with dust is not excluded.

Pathogenesis. The pathogenesis is based on the mechanical effect of helminths on the intestinal mucosa, associated with irritation of mechanoreceptors and chemoreceptors during their fixation and movement. Irritation of the ileocecal region leads to a reflex violation of the motor and secretory functions of the digestive tract, resulting in the development of gastritis, gastroduodenitis, enteritis.

Individuals of female helminths can penetrate deep into the mucosa by 2/3 of their body length, contributing to the development of an inflammatory reaction, the formation of granulomas consisting of eosinophils, macrophages, and lymphocytes. The severity of the pathological process depends on the intensity and duration of the invasion. In rare cases, when penetrating into the appendix, pinworms can cause appendicitis (Fig. 61).

Figure 61. Section of the appendix with pinworms. Stained with hematoxylin and eosin.

Sometimes in girls, pinworms can crawl into the vagina, through it into the uterus and fallopian tubes, and even into the pelvic cavity. Ectopic migration of pinworms is the cause of vulvitis, vaginitis, endometritis, which is a serious problem in pediatric gynecology.

Prolonged irritation of the skin of the perianal region and the perineal region can lead to skin maceration and the addition of a secondary infection with the development of pyoderma.

The presence of pinworms leads to a violation of the normal composition of the microbial flora of the intestine, contributing to the development of dysbacteriosis. Infested children are 2.2-3 times more likely to suffer from intestinal infections. Enterobiasis promotes transmission of the single-celled parasite dientameba (*Dientamoeba fragilis*).

The clinic depends on the intensity of infection, the frequency of reinfestations, the age and individual reaction of the infected person. The disease develops gradually.

With a primary massive infection (about 100 eggs), after 2-3 days the child develops abdominal pain, frequent, shaped or mushy stools up to 2-4 times a day without an admixture of mucus and blood. Then, over the next 2-6 days, the

clinical symptoms subside. A month after the initial infection, itching appears in the perianal region.

With a low intensity of invasion, patients have only one symptom - perianal itching, which occurs more often in the evening and at night. Only a few of them report pain in the abdomen. Itching reappears after 28-30 days. Periodicity is associated with the completion of the development of a new generation as a result of reinvasion. As a result of frequent repeated infections, the intensity of invasion increases, while in the intestine there are helminths of different age groups: larvae after the first and second molt, young females and males, mature females. Episodes of perianal itching are becoming more frequent, and over time it can become daily and occur not only in the evening and at night, but also during the day, spreading along the perineum in women to the area of the labia, vagina.

Abdominal pain intensifies, more often in the right ileocecal region, resembling pain in appendicitis; tenesmus, flatulence, loose stools may occur, body weight decreases. Unbearable itching disturbs sleep, causes nocturia, masturbation. Neurological symptoms may be expressed, such as headache, dizziness, memory loss. Children become capricious, whiny, poorly perceive useful skills. Instability of attention, inability to concentrate worsens academic performance. Adults are less productive. Syncope and epileptiform seizures are described in children infested with pinworms.

Due to prolonged irritation and scratching on the skin of the perianal region, dermatitis occurs, sometimes severe, weeping or dry eczema, spreading beyond the perineum.

In some cases, girls develop vaginal discharge, develop vulvitis, vulvovaginitis.

In the general blood test, no changes are noted. In the general analysis of urine in girls, leukocyturia, microhematuria, and even pinworm eggs can be detected, which are mechanically washed away during urination.

Complications. In the structure of rare complications, anal, rectal fistulas, paraproctitis are also described. Vulvitis, vulvovaginitis, urinary tract infections,

proctitis, appendicitis, dermatitis, eczema may develop. In a number of cases, there was a delay in the neuropsychic development of children.

Diagnostics. The basis for the assumption of the presence of enterobiasis can be persistent itching in the perianal region at night, usually subsiding during the day, as well as a combination of this itching with dyspeptic manifestations. Often the appearance of similar symptoms in people who are in close contact with the sick person.

Of decisive importance in the diagnosis is the identification of pinworm eggs in a scraping from the perianal folds, which is performed in the morning before defecation with a cotton swab moistened with glycerin or with a transparent adhesive polyethylene (polyvinyl chloride) tape.

Due to the periodicity of oviposition associated with the change of generations of pinworms, in the case of a low-intensity invasion, the study should be carried out three times with an interval of 7-10 days. The diagnosis can also be made by detecting pinworms on the surface of the feces, which are sometimes noticed by the infested themselves.

Differential diagnosis in some cases should be carried out with atopic dermatitis, seborrheic dermatitis, lichen planus, fungal infection, proctitis, anal fissures, latent fecal incontinence, psychogenic itching, vaginitis and bacterial vulvovaginitis.

Treatment. Before a course of antiparasitic therapy, it is advisable to carry out a thorough wet cleaning of the room using disinfectants, wash children's toys.

In the evening on the day of treatment, it is necessary to take a shower, change underwear, the child should be put to sleep in shorts that fit tightly around the hips to prevent contamination of the hands by pinworms that actively come out at night.

Urom recommended a shower or thorough washing, change of underwear. Bed linen on deworming days should be ironed with a hot iron.

Wet cleaning of the premises during the treatment is carried out daily.

For treatment, you can use a wide range of anthelmintic drugs:

Albendozol inside children 2-12 years old - 10 mg / kg, children over 12 years old and adults - 400 mg 1 time per day once after a light breakfast. Re-admission - after 2-3 weeks. Efficiency - 95%.

Karbendatsim - adults and children over 1 year old 10 mg / kg / day in 3 doses every 30 minutes. after meal. The course of treatment is repeated after 2-3 weeks. Efficiency - 92%.

mebendazole inside after 20-30 minutes. after meals - for children - 10 mg / kg, for adults - 100 mg once. Re-admission - after 2-3 weeks. Efficiency - 90%.

Pirantel - once 10 mg/kg (maximum dose for adults - 750 mg). Re-admission after 2-3 weeks. Efficiency 90-97%.

The prognosis is favorable.

Prevention. A set of measures to combat enterobiasis is aimed at identifying and improving the source of invasion and preventing the transmission of the pathogen.

Children attending kindergartens, schoolchildren in grades 1-4 and staff of kindergartens are subject to an annual examination for enterobiasis. It is forbidden to accept without examination for enterobiasis children who are newly registered in preschool and health institutions, schools, as well as returning to them after a long absence (1 month or more). All children admitted for treatment to hospitals of any profile are subject to examination for enterobiasis. In the clinic and children's consultations, the examination is carried out according to clinical indications. Examination for enterobiasis in adults is necessary when applying for a job in kindergartens, schools, boarding schools, trade enterprises, public catering, swimming pools.

In children's groups, where 20% of infested were identified, all children receive two-time treatment with an interval of 14-21 days.

In the foci of enterobiasis, it is necessary to provide young children with individual pots, systematic treatment of pots with boiling water, providing children with individual towels, decontamination of bedding and underwear from helminth eggs, and twice a day wet cleaning of the room using soap and soda solution.

Sanitary and educational work, education of hygiene skills in adults and children (washing the child in the morning and evening, frequent washing of hands with soap, short-cut nails, fighting the habit of biting nails, sucking fingers, daily change of underwear, cleaning the room).

ANKYLOSTOMIDOZES

Ankylostomiasis (ICD10 - B76.0-9) - hookworm and necatoriasis - helminthiases characterized by damage to the gastrointestinal tract and the development of iron deficiency anemia

Etiology. Crooked head of the duodenum (*Ancylostoma duodenale)* - the causative agent of ankylostomiasis and necator (*Necator a mericanus*) - the causative agent of necatoriasis.

Crooked head and necator are representatives of the hookworm family (Ancylostomatidae). These are small worms of pale pink color, the size of female hookworms is 10-13 mm, males are 8-11 mm. The necator is smaller than the crookhead: females - 9-11mm, males - 5-9mm. In the crooked head and the necator, the anterior end of the body is bent to the dorsal side in the form of a hook. Both worms are hematophages, the mouth opening of both is surrounded by an oral capsule, inside which there are 4 cuticular teeth at the crooked head, and two cutting plates at the necator (Fig. 62).

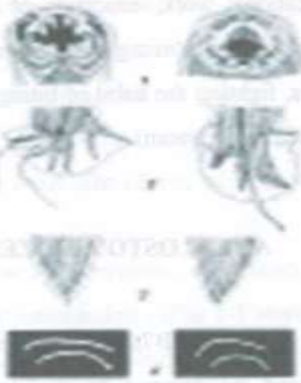

Figure 62. Distinguishing features of Ancylostoma duodenale (left) and Necator americanus (right): 1 - oral capsule (dorsal view); 2 - copulatory bag at the rear end of the body of the male; 3 - rear end of the body of the female; 4 – body shape of dead parasites (females on the left, males on the right); mouth end below (according to Naucke J.).

Both species live in the intestinal lumen, attaching to the mucosa of the upper small intestine.

Hookworm eggs are oval, size - 60 microns, covered with a transparent membrane inside which 4-8 or 16 blast cells (Fig. 63).

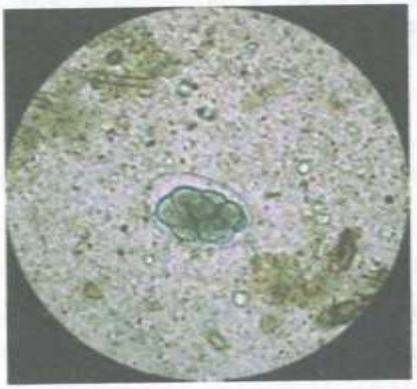

Figure 63. Immature hookworm egg (8 blastomeres) .

Life cycle. After fertilization, the female of the hookhead lays up to 10 thousand eggs daily, some - three times less. Eggs are excreted along with feces into the external environment (fig. 64).

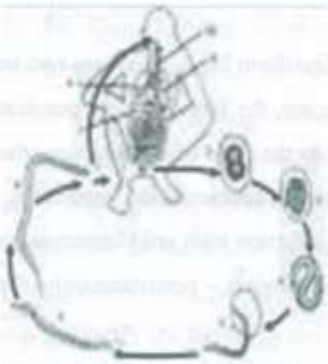

Figure 64. The life cycle of pathogens of human ankylostomidosis (according to Peters and Giles): 1 - parasitism of mature hookworm in the human small intestine; 2-4 development (maturation) of eggs in the soil; 6 - exit of the larvae from the egg and existence in the soil; 7 - formation of invasive filariform larvae; - penetration of an infective larva into human skin or ingestion of an infective larva; 8 - migration of the larva along the lymphatic and circulatory systems to the heart; 9 - entry into the lungs and penetration into the respiratory tract; 10 - migration through the trachea and pharynx and entry through the esophagus into the intestinal tract, where development occurs to the sexually mature stage .

Under optimal conditions (temperature 32 ° C, high humidity, free access to O^2, moist, humus-rich soil), a rhabdite-like larva is formed inside the egg, which leaves the egg shells in a day or two and leads a free lifestyle, feeding on bacteria. In these larvae, as in all free-living nematodes, the esophagus consists of two extensions separated by a constriction. In warm soil, rhabditoid larvae molt twice (on days 3 and 5) and turn into filariform (invasive) larvae. This stage is located in the cap - the cuticle of the second larval stage, which it does not shed. Filariform

larvae remain in the soil without feeding for up to 18 months. At the same time, they can make vertical migrations. When it gets cold in winter, they sink to a depth 1 where they can overwinter. When the temperature rises, they rise to the upper layers of the soil, and even crawl onto plants.

The invasive stage for humans is the filariform larva, there are two ways of infection: percutaneous and oral. In the first case, the larva actively penetrates the skin upon contact of a person with the soil. At the time of introduction, the larva sheds its sheath. Oral infection is possible through contaminated vegetables, fruits, water. Japanese researchers have shown that infection with ankylostomiasis occurs mainly by ingestion of larvae, and with non-catorosis - percutaneously. Cases of transplacental infection are known.

Regardless of the method of penetration, the migration of hookworm larvae in the human body is an obligatory stage in the development cycle of these parasites. The larvae enter the circulatory system through the skin, oral mucosa, and intestinal mucosa. Migration ends in the lungs, where the larvae destroy the walls of capillaries and alveoli. Further, the larva enters the airways, then, together with sputum, ends up in the nasopharynx, oral cavity, and then in the intestine when swallowed with saliva, water, and food.

Once in the intestines (duodenum), the larvae molt again, giving rise to adult worms, which deeply penetrate the mucosa with their front end and switch to feeding on blood. Hookworms reach sexual maturity in 5-6 weeks. At the same time, *Necator and mericanus females* begin to lay eggs. At *Ancylostoma duodenale* there is a period of delayed development: most females begin to release eggs 6-8 months after penetration through the skin into the human body. The duration of invasion ranges from 4 years with necatoriasis, up to 15 years with ankylostomiasis.

Epidemiology. According to the number of infested people, ankylostomidosis takes the second place after ascariasis. They infected more than 900 million people. At the same time, 450 million new cases of the disease are registered annually. Ankylostomiasis are distributed mainly among the population

of tropical and subtropical zones, in the countries of South and Central America, South Asia, Africa, Hindustan, Indochina and on the islands of the Malay Archipelago. In some of these countries, up to 50% of the population is infected.

In the Central Asian republics of the CIS, there are limited foci of necatoriasis and hookworm, in the Transcaucasus - mainly hookworm. In Uzbekistan, this disease has been eliminated.

Ankylostomiasis - anthroponotic geohelminthiases. The only source of infection is humans. Soil is the main transmission factor. Dispersion of larvae in the environment is facilitated by soil contamination with faeces, erosion of cesspools by heavy rains, as well as the use of undisinfected feces for fertilizing vegetable gardens. Mechanical transfer of contaminated fecal particles by pets, birds, flies is possible. A sick person does not pose an immediate danger to others, since he releases immature eggs. The ripening of eggs in the soil and the formation of larvae in them is possible at a temperature of 14-40 °C and high humidity (70-80% for necator larvae and 85-100% for hookworm larvae). The larvae leave the egg shells, molt twice, turning into invasive (filariform larvae). They actively move in the soil in horizontal and vertical directions. When spreading horizontally, they migrate within a radius of 10 м. When the air temperature drops, they are able to go deep into the soil (up to 1 м) and persist for several months. On moistened grass stems, they can rise to a height of up to 22 cm, as a result of which they can get on the skin of a person, even if he wears light shoes. Most larvae die within 6-9 weeks, but under optimal conditions they remain viable for up to 8-18 months.

The main routes of transmission are food, water and contact (through the skin). Cases of transplacental infection are known. Invasion occurs when eating fruits, vegetables, herbs, berries contaminated with hookworm larvae, through dirty hands, as well as when walking barefoot or in light shoes, lying on the ground, when performing agricultural and earthworks . Endemic foci are formed in coal and mining mines at high temperature, humidity and poor sanitary conditions.

The natural susceptibility of people is high. Risk groups include children, summer residents, agricultural workers, miners, tourists visiting endemic regions.

Pathogenesis. In the pathogenesis of ankylostomidosis, the leading role belongs to the sensitizing effect of larval antigens, their mechanical effect on the tissues of those organs through which they migrate, as well as the hematophagy of parasites.

In the lungs, pinpoint hemorrhages occur with infiltration by eosinophils and leukocytes. During the period of migration of individuals, the patient develops toxic-allergic and pulmonary syndromes.

After reaching puberty, the intestines enter the chronic (intestinal) phase of the disease with the development of gastrointestinal disorders and anemic syndrome. Hookworms are obligate hematophagous, feeding only on blood. When fixing in the intestine, the cutting appendages of the oral capsule capture parts of the mucosa, damaging small vessels and capillaries, causing microbleeding. Bleeding ulcerations up to 2 cmin diameter are observed at the site of attachment of the parasite. Anticoagulants secreted by the esophageal glands of worms slow down blood clotting, which leads to prolonged bleeding. The daily loss of blood during parasitism of one individual is 0.03-0.05 ml, hookworm - 0.16-0.34 ml. The level of blood loss and iron loss depends on the intensity of the invasion. In humans, it can parasitize from a few copies of hookworm (low intensity) to several tens (medium intensity), hundreds and thousands of copies (significant intensity).

An important manifestation of pathogenesis is the loss of protein and lymph with the development of hypoalbunemia, malabsorption of amino acids. Mechanical damage to the mucous membrane of the small intestine contributes to the introduction of microbial flora, the development of catarrhal inflammation, sometimes erosions and ulcers in the form of duodenitis and peptic ulceration. Violated motor and secretory functions of the gastrointestinal tract.

Clinic. The incubation period of the disease lasts 40-60 days. In most cases, the invasion is asymptomatic, a clinically pronounced course is observed with moderate and high intensity of invasion, as well as in patients with immunodeficiency.

When the larvae enter through the skin, itching and burning of the skin, erythematous, papular, vesicular or pustular rashes occur, which can persist for several months. Often in the skin there are foci of infection due to the introduction of pathogenic microflora (streptococci, staphylococci) with the development of purulent-inflammatory processes in the skin.

When the larvae migrate through the respiratory tract, catarrhal phenomena may develop, shortness of breath, wheezing, pain behind the sternum appear, less often hoarseness, up to aphonia. There may be a development of bronchitis, pleurisy, pneumonia. Eosinophilia is observed in the blood.

If hookworm enters the gastrointestinal tract, duodenitis may develop with heartburn, lack or increased appetite, sometimes taste perversion (desire to eat, for example, clay), nausea, vomiting, diarrhea, pain in the epigastric or hepatic region. Often parasitism hookworm provokes the development of gastric ulcer and duodenal ulcer.

In children with ankylostomidosis, signs of damage to the central nervous system are noted, lethargy, lag in mental and mental development appear.

Anemia develops 3-5 months after infection. Weakness, dizziness, fatigue, pallor of the skin and mucous membranes, angular stomatitis, dry skin, brittle nails and hair, puffiness of the face, and sometimes swelling of the legs are noted.

With prolonged iron deficiency anemia, dystrophic changes in the heart muscle increase, accompanied by shortness of breath, palpitations, and increased heart rate. In severe cases, hemorrhages in the retina are noted. Eosinophilia in the blood, compared with the acute period, is somewhat reduced. Anemia is characterized by a decrease in the number of erythrocytes (up to $1x10^{12}$ /l), hemoglobin (up to 70-90 g/l) and color index (up to 0.3-0.5), microcytosis, moderate reticulocytosis are recorded.

Ankylostomidosis is especially dangerous in pregnant women, when developing anemia leads to a threat to the life of the fetus and the pregnant woman.

Complications: decompensated anemia.

Diagnosis is based on detection of hookworm eggs in faeces using unified flotation methods: Fülleborn, Kalantaryan. Considering that hookworm eggs develop rapidly in faeces and become similar to trichostrongylid eggs when crushed, the material should be quickly delivered to the laboratory. When using the Fulleborn method, the film is microscoped after 10-15 minutes of settling. A thick Kato smear due to the clearing of eggs in glycerin is not always reliable. Highly informative is the cultivation of larvae by the Harada and Mori method in a test tube on filter paper, followed by viewing and studying the morphology of helminth larvae (Fig. 65).

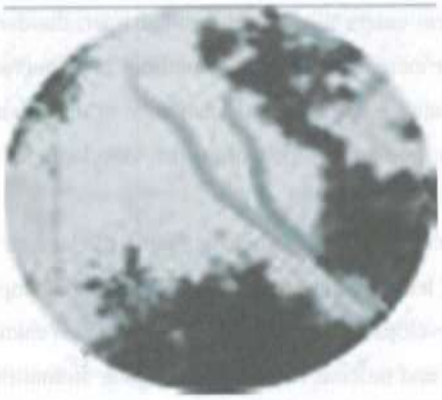

Figure 65. Ankylosomidae larvae.

Serological methods are not used in clinical practice.

In the peripheral blood, there is a decrease in erythrocytes to $1x10^{12}$ / l, hemoglobin - up to 70-90 g / l, color index - up to 0.3-0.5; microcytosis, moderate reticulocytosis, leukocytosis up to $15-20x10^{9}$ /l, eosinophilia - (in the initial period up to 50-60, then decreasing to 10-15%), accelerated ESR.

In a biochemical blood test, hypoalbuminemia, a decrease in the level of serum iron, and an increased iron-binding capacity of blood serum are determined.

Differential diagnosis is carried out with gastroenterocolitis of any etiology, other nematodes, amoebiasis, balantidiasis, peptic ulcer, ulcerative colitis, Crohn's disease.

Treatment is usually carried out on an outpatient basis or in day hospitals. With the development of intense invasion and decompensated anemia, hospitalization is necessary. In etiotropic therapy, the following drugs are used:

Albendazole, adults and children - 0.4 g / day once after a light breakfast.

Mebendazole for adults - 0,1 gr twice a day, children 2-3 years old - 0,025 gr, 4-7 years old - 0,05 gr, 8-10 years old - 0.075, over 10 years old - 0.1 g / day in the morning and evening for 3 days.

Carbendacym - adults and children 10 mg / kg / day in 3 doses every 30 minutes. after meals for 3 days.

In young children and in severe anemia, antiparasitic treatment should be carried out only after therapeutic measures aimed at correcting anemia. In severe cases (hemoglobin less than 40 g / l), it is advisable to transfuse red blood cells or whole blood before prescribing antiparasitic therapy.

Antianemic therapy. With the development of anemia, iron preparations are prescribed in age doses, if necessary, folic acid.

Correction of protein metabolism. In case of severe hypoalbuminemia , ***albumin*** 5-10-20% solution is administered intravenously in a dose of 200 ml at a rate of 50-60 drops / min or ***fresh frozen plasma*** in / in a drip of 20 ml / kg / day. at a rate of 50-60 drops / min, the frequency of injections is determined individually.

Forecast for life favorable, except in young children with severe anemia.

Prevention. Prevention and control measures consist in identifying and curing all those infected, carrying out general sanitary measures, including protecting the soil from fecal pollution and neutralizing sewage. Small areas around cesspools of latrines, field ditches in places of temporary accommodation of people and other places of sewage collection are treated with table salt at the rate of 0.5-1 kg per 1 m2 every 5-10 days. It is important to observe the rules of personal hygiene, careful processing of vegetables and fruits. Wearing shoes in endemic areas and avoiding contact with the soil of exposed areas of the body.

Successful results in the prevention of ankylostomiasis can only be achieved with active and systematic sanitary and educational work.

TOXOCAROSIS

Toxocariasis (ICD10 - B83.0) is a larval, chronically proceeding tissue zoonotic geohelminthiasis. The disease is characterized by a long and recurrent course, polymorphism of clinical manifestations, with a predominant lesion of the internal organs and eyes.

Etiology. Human toxocariasis is caused by canine toxocara (*Toxocara canis*) , extremely rare - feline toxocara (*T. mystax*) . In animals, toxocara parasitize in the small intestine. These helminths have a phylogenetic relationship with roundworms. Toxocar sizes are 12 - 19 cm in dogs and 3 - 7 cm in cats. These nematodes have wide cuticular "wings " at the head end on the sides. (Fig. 66).

Figure 66. Head end *of Toxocara canis* .

Toxocara canis eggs are almost round, brown, with a fine-meshed shell, 66-85 x 64-78 microns in size (Fig. 67).

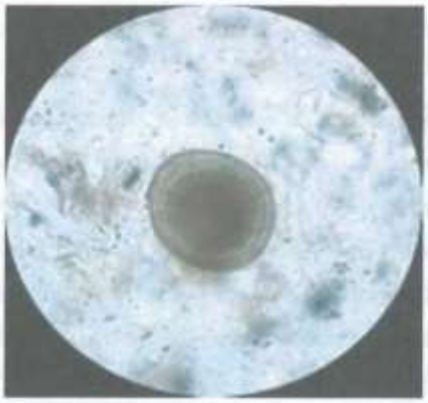

Figure 67. An immature *Toxocara canis* egg .

Life cycle. Sexually mature Toxocara live in the small intestine of dogs, cats and some other predatory mammals. After mating, females lay thousands of eggs every day, which are passed into the environment with feces. Like roundworms, Toxocara eggs are shed immature and mature to the invasive stage in the soil. The optimum temperature is 24-30°C, air humidity - 85%, soil - 20% and higher. Under these conditions, the development of the larva in the egg ends within 5-8 days. At lower temperatures, the formation of the larva is completed in 2-4 weeks.

There are several ways to infect animals:

- ingestion by dogs and cats of invasive eggs with soil contaminated with Toxocar eggs (oral route);
- intrauterine infection with larvae of puppies through the placenta from an infested pregnant bitch (transplacental route);
- larvae through the milk of a lactating dog (transmammary route);
- eating the tissues of paratenic (reservoir) hosts by the dog, which are primarily small mammals - rodents that have swallowed the mature eggs of the parasite. Toxocara larvae are found in the tissues of such hosts.

After toxocara larvae enter the body of a dog or cat, they migrate (intestine - liver - right heart - lungs - upper respiratory tract - oral cavity - intestines), after

which they grow and become sexually mature. Their life expectancy is 8-10 months.

Epidemiology. Toxocara infection is widespread both among animals and among people in almost all countries of the world. The incidence of dogs, the main hosts of Toxocara, is very high worldwide, reaching 90% in some regions. The increase in the number of dogs in cities, their high prevalence of toxocara invasion, the significant fecundity of helminths, and the stability of eggs in the external environment are the determining factors for the wide spread of invasion.

Toxocara infection in humans varies from 2.6% in Belgium to 80% in the Caribbean.

The source of invasion for humans in the synanthropic focus are dogs (rarely cats), which seed the soil with helminth eggs. Humans cannot be a source of infection, since the parasite does not reach the sexually mature stage in the human body and does not release eggs into the external environment.

Human infection occurs as a result of ingestion of Toxocara eggs with food and water. The risk group for the development of toxocariasis includes children 1.5-5 years old, in contact with the soil and dogs; children suffering from geophagy; persons who have professional contact with animals and soil (veterinary workers, employees of nurseries, circuses, zoos, public utilities workers, sellers of vegetable shops, workers of vegetable bases); mentally retarded and mental patients with a tendency to copro- and geophagy; homeowners; pet owners.

Pathogenesis. In the human intestines, larvae emerge from the eggs, 0.1 mm in size. They perforate the intestinal wall and penetrate the lymphatic and blood vessels and enter all organs and tissues, primarily the liver, as well as the lungs, eyes, spleen, skeletal muscles and brain . Once in capillaries narrow in diameter (0,02 mm), the larvae get stuck in the parenchyma of the organs, where they linger, remaining viable for many months. With the weakening of the body's defenses, their activation and further migration through the body are possible. Gradually, the larvae encapsulate and die. Based on their life cycle, toxocara in humans tend to get into the lungs.

Morphologically, toxocariasis is an eosinophilic granulomatosis as a manifestation of a delayed-type allergic reaction. In the center of the granuloma there is a zone of necrosis with a larva, along the periphery - a large number of eosinophils, histiocytes, neutrophils, macrophages, lymphoid and epithelioid cells. With massive infection, numerous granulomas form in the lungs, liver, pancreas, myocardium, mesenteric lymph nodes, brain, and organ of vision. In the process of migration, the larvae injure tissues, cause hemorrhages, necrosis, and can be conductors and inoculators of pathogenic microbial flora.

The leading role in the pathogenesis of toxocariasis is played by the sensitization of the body by the waste products of the helminth - metabolic and somatic antigens with the development of hypersensitivity reactions. Increased production of specific antibodies of the IgE class leads to the activation of mast cells, basophils, neutrophils and their release of a number of mediators - histamine, prostaglandins, leukotrienes, responsible for the development of immediate allergic reactions. Activation of Th 2 cells, an increase in IL5, IL3, GM - CSF contribute to the activation of eosinophils, an increase in their number in the vascular bed and in tissues. Eosinophils are concentrated in the localization of Toxocara larvae, where they break down and release granules containing enzymes. Under the action of eosinophilic peroxidase, the vascular endothelium and endocardium are damaged. Thrombi are formed due to the main basic protein, which inactivates heparin. The generation of active oxygen radicals and the production of cationic proteins make eosinophils the central damaging factor during their accumulation in tissues. Sensitized T-lymphocytes that have accumulated around the larva secrete lymphokines, attract macrophages and other cells that are involved in the process of granuloma formation.

Clinic. Toxocariasis is characterized by a long (up to several years) relapsing course. Clinical relapses are due to the periodic release of parasite larvae from granulomas and the resumption of their migration. There are visceral and ocular forms of toxocariasis.

Visceral toxocariasis. There are two variants of the course of the disease:

- inapparant;
- clinically pronounced.

Clinically pronounced visceral toxocariasis is recorded mainly in children aged 1.5-4 years, adults get sick less often and get sick more easily. Boys are infested 2 times more often than girls, probably due to more active contact with the soil, picacism (the desire to take the earth into the mouth).

Allocate acute and chronic stages of invasion. The disease often begins acutely - without a prodromal period, only in rare cases there is a short period of mild malaise.

The main clinical symptoms of the acute stage of toxocariasis are recurrent fever, lymphadenopathy, pulmonary syndrome, enlarged liver, less often spleen, skin rashes in the form of urticaria or polymorphic rash, Quincke's edema.

The temperature is often subfebrile, less often febrile, usually rises at noon or in the evening, accompanied by chilling, and with a decrease - profuse sweating. There is an injection of the vessels of the conjunctiva.

The defeat ***of the bronchopulmonary system*** is observed in 20-50% of patients, characterized by a variety of clinical manifestations (airway catarrh, bronchitis, pneumonia, broncho-obstructive syndrome).

In the classic version, eosinophilic pneumonia develops (with Loeffler's syndrome). Sick children have coughing fits, more often at night, often - asthma attacks with severe shortness of breath, cyanosis. During auscultation, dry whistling rales are heard - single or over the entire surface of the lungs, sometimes - foci of moist rales. In the general blood test, leukocytosis and eosinophilia are noted (always more than 10%). X-ray examination reveals "flying" infiltrates, increased lung pattern. When examining the function of external respiration, signs of bronchial obstruction are often revealed: a decrease in FEV1, the Tiffno index, and the average volumetric expiratory flow rate.

Hepatomegaly is considered a typical sign of toxocariasis. It occurs in 40-80% of patients and is characterized by a moderate enlargement of the liver. Enlargement of the spleen is noted in 20-40% of cases.

Ultrasound of the liver registers linear structures of increased echo density, rounded foci of low density, foci of calcification, an increase in lymph nodes in the gates of the liver, spleen, para-aortic lymph nodes. Puncture biopsy of the liver reveals eosinophilic infiltration of the stroma, eosinophilic granulomas, and sometimes a picture of acute hepatitis of an immune nature. In rare cases, a granulomatous tissue reaction, Toxocara larvae, can be detected. There are descriptions of pyogenic liver abscesses, which are associated with a delay in the microbial flora around parasitic granulomas.

In 60-70% of children, there is an increase in ***lymph nodes,*** up to the development of lymphadenopathy. Palpable lymph nodes are painful, mobile, without inflammatory changes.

CNS ***involvement*** is rare. In the acute stage of the process, patients complain of headache, insomnia, irritability. In severe cases of invasion, meningoencephalitis, arachnoiditis, and recurrent myelitis develop. There are signs of cerebral and focal lesions of the brain and spinal cord - paresis, paralysis, generalized and focal convulsions, various mental disorders. In rare cases, pathomorphological examination of the brain reveals vasculitis, eosinophilic granulomas. Toxocara larvae are found in the brain and cerebrospinal fluid.

An increase in total IgE is characteristic .

Inapparent (asymptomatic) toxocariasis is manifested by the presence of antibody titers of 1:800 and above, constant eosinophilia of more than 10% without clinical manifestations.

Ocular toxocariasis. The ocular form of toxocariasis is rarely combined with visceral lesions and develops mainly in children over 12 years of age and adults. With toxocariasis of the eye, the retina is more often affected, the choroid itself, where the inflammatory process is formed, and specific granulomas. Usually one eye is affected, but bilateral lesions are also possible. Clinically, diffuse endophthalmitis, posterior granulomatous chorioretinitis, eosinophilic abscesses in the flat part of the ciliary body, cataracts can be observed. In severe cases, the

process can progress to panophthalmitis and retinal detachment. Ocular toxocariasis can lead to blindness.

Diagnosis The most typical change in laboratory parameters in toxocariasis is an increased content of eosinophils in the peripheral blood. The relative level of eosinophils can range from 10-15% to 40-80%. Leukocytosis, hypergammaglobulinemia, hypoalbuminemia, and accelerated ESR are also characteristic. With a long course of the disease, anemia develops. With liver damage in 20% of patients, an increase in the activity of aminotransferases is observed.

The leading role in the diagnosis of toxocariasis is assigned to immunological research methods, since one of the stable signs in toxocariasis is an increase in the content of specific I gE in the blood serum.

, the level of I gM increases , later - I gG. The most effective, sensitive and specific method for determining anti-toxocariasis IgG is enzyme-linked immunosorbent assay (ESA) using excretory-secretory antigen *T. canis* . The sensitivity of ESA in patients with toxocariasis is about 78%, and the specificity is 92%. It is believed that the diagnosis of toxocariasis can be confirmed serologically if the patient has a characteristic clinical picture (usually damage to the lungs or eyes), persistent eosinophilia of more than 10%, and an antibody titer of 1:800 or higher.

As an additional method for diagnosing toxocariasis, it is proposed to determine specific IgE to toxocariasis antigen and circulating toxocariasis complexes in blood serum.

The diagnosis of ocular toxocariasis is mainly established retrospectively, with a histological examination of the removed granulomas. Serological reactions in ocular toxocariasis are uninformative. Many of the symptoms of toxacariasis are non-specific and can also occur with other diseases.

Differential diagnosis is carried out with schistosomiasis, ascariasis, opisthorchiasis and other diseases accompanied by high eosinophilia (Leffler's syndrome, tropical eosinophilia). Eye lesions in toxocariasis must be differentiated

from retinoblastoma and chorioretinitis of another etiology (tubercular, toxoplasmic, cytomegalovirus).

Treatment. With asymptomatic toxocariasis, accompanied by an increase in eosinophils in the peripheral blood and low titers of antibodies to toxocara, specific therapy is not carried out. Patients should remain under dispensary supervision.

Treatment of patients with toxocariasis is carried out on an outpatient basis or in a hospital. Children under 3 years of age, as well as patients with severe forms of invasion, are treated in a hospital.

Before starting treatment, it is necessary to conduct a patient examination, including a clinical blood test, urinalysis, ECG, a biochemical study (total protein, bilirubin and its fractions, urea, ALT, AST, γ-GT, alkaline phosphatase, iron). Carbamatebenzimidazole derivatives are used to treat toxocariasis.

albendazole, inside after meals, adults - 0,4 gr2 times a day, children - 10 mg / kg / day in 2 divided doses, for 14-21 days.

Less effective mebendazole inside 10 mg / kg / day in 3 doses, courses of 3-6 weeks.

When using these drugs, it is necessary to monitor peripheral blood parameters to exclude adverse reactions.

The effectiveness of the use of antiparasitic agents is about 50% and repeated treatment is often required.

If necessary, pathogenetic therapy is carried out. Antipyretics are indicated for high fever. In case of severe bronchial obstruction, bronchodilators are prescribed (salbutamol, fenoterol, ipratropium bromide / fenoterol, cromoglycic acid / fenoterol). To stop allergic manifestations, antihistamines are used.

Prevention. The complex of preventive and anti-epidemic measures includes: planned deworming of dogs, equipment of special places for their walking, protection of children's playgrounds, parks, squares from visiting animals; compliance with the rules of personal hygiene (washing hands after contact with animals, soil, thorough washing of greens, vegetables that may contain soil

particles); fight against the habit of picacism, the destruction of cockroaches - mechanical carriers of toxocar eggs.

DIROFILARIOSIS

Dirofilariasis (code according to ICD10 - B74.8) is a tissue filariasis that manifests itself in humans during the invasion of dirofilaria by the formation of a mobile tumor under the skin in various parts of the body, as well as under the conjunctiva of the eye.

Etiology. The causative agent is *Dirofilaria repens* . This is the only type of filaria, cases of infection with which are registered in the CIS.

Dirofilariae are thin filamentous white nematodes (from Latin diro - hard, repens - creeping). The length of sexually mature females of *D. repens* reaches 135-170 mm, males - 50-70 mm, the maximum body thickness is about 1.2 mm. The body of dirofilaria is covered with longitudinal cuticular ridges and delicate transverse striation. The size of the larvae (microfilaria) varies from 275 to 362 microns in length, with a diameter of 6-8 microns. The sheath is absent, the posterior end is narrowed and does not contain nuclei of somatic cells.

Life cycle. In the body of dogs - the final owners of sexually mature helminths live in the subcutaneous connective tissue. Females give birth to larvae (microfilariae) in the blood, which, without changing morphologically, circulate in the circulatory system of the animal. When a mosquito (intermediate host and carrier) sucks blood, microfilariae enter its intestines with blood. From the intestine, they actively migrate into the body cavity and develop in the Malpighian vessels to the larvae of the invasive stage, after which they migrate to the head section of the insect. The development of larvae in the body of a mosquito occurs at an ambient temperature above +14 °C. At an optimum temperature of +23-28 °C, their development lasts 8-17 days.

120 days after infection, fertilized females are found in the dog's body, which are located in the subcutaneous connective tissue, usually paired with males. 190-200 days after invasion, microfilariae can be detected in the peripheral blood vessels of the dog in the evening and at night. The life expectancy of adult dirofilaria in the definitive host is 2-7 years, microfilaria - 2-2.5 years. *The* average duration of the pre-patent period *D. repens* in the body of a dog is 193 days.

Epidemiology. The source of invasion in synanthropic foci is infected domestic dogs, less often cats, and in natural foci - wild canids. Carriers are mosquitoes of the genera *Culex* , *Aedes* , *Anopheles* . *More effective carriers of* dirofilaria are mosquitoes of the first two genera. An increase in the number of stray animals, their mass migration in nature and in settlements, urbanization processes and climate warming largely contribute to an increase in the risk of human infection with dirofilariasis.

Data on the incidence of animals and exact data on the frequency of invasion among humans are not available. Identification of cases of invasion *D* . *repens* is random. In humans, female helminths are able to reach puberty, but cannot give birth to microfilariae, so a person is an accidental owner of dirofilariae and cannot participate in the spread of dirofilariasis.

The first clinical manifestations of the disease are usually recorded in the winter, which is associated with human infection in the summer months of the year. Dirofilariasis is a widespread infection of dogs throughout the world; sporadic cases of human injury are recorded everywhere. They are known in the south of Europe (in Italy, Hungary, the countries of the Balkan Peninsula, Turkey, etc.), in Ukraine, Georgia, Armenia, Kazakhstan and Uzbekistan. Large foci of dirofilariasis are located in the tropical regions of North and South America.

Pathogenesis. From the moment of infection to the formation of a nodule in which the helminth is localized, it usually takes about 1 month, but there are cases when the incubation period has been prolonged to 12 months and even several years. Primary inoculation of the invasive larvae of *D. repens* can occur in various human tissues: under the skin, mucous membranes, subcutaneous fat, and have

different localization, which is determined by the site of the mosquito bite. It should be emphasized that dirofilariasis is characterized by pronounced mobility of the parasite and the possibility of its migration, the speed of which is up to 10–15 cm/day. Despite the invasion and possible migration of helminth larvae, the development of a general reaction of the macroorganism in dirofilariasis is atypical.

In humans, unlike animals, microfilariaemia does not develop, which is the main (but not the only) reason for the absence of general manifestations of the disease. Due to the lack of sensitization of the body, eosinophilia is also rarely detected. Toxic-allergic reactions do not develop even if the parasite is present for a long time (for several months). And only in the case of the death of the helminth and the development of suppuration in patients, intoxication phenomena can develop. Although the number of clinical observations is limited, it is believed that the duration of parasitism of *D. repens larvae* in human tissues may be only a few months, while in animals it can be several years.

Clinic . Clinical manifestations of *D. repens invasion* can be very diverse, due to the primary localization of the parasite and its subsequent migration. Most often, dirofilaria larvae are localized in the eye area (conjunctiva, eyelids, etc.), on the trunk, in the area of \u200b\u200bthe mammary glands, limbs, men in the genital area (scrotum), and more rarely in the mouth, root of the tongue, pharynx , in the omentum and mesentery. The complexity of early detection of dirofilariasis is associated with the peculiarity of the onset of the disease. As a rule, its first sign is the appearance of an inflammatory skin infiltrate 3–5 cm in diameter at the site of parasite inoculation.

Sometimes patients note itching and burning of varying intensity, while patients feel stirring and crawling in the area of the inflammatory infiltrate. A migrating skin infiltrate may persist for 2–3 months and disappear periodically, which is an important clinical sign.

With the localization of the parasite in the eye area, patients may experience hyperemia and swelling of the eyelids, lacrimation, blepharospasm, ptosis, pain at

rest and on palpation. The most severe cases of the disease occur with damage to the eyeball. In patients in such cases, a decrease in visual acuity is often detected, and they see "moving worms".

Diagnostics. Primary diagnosis of dirofilariasis is difficult. In almost all patients, erroneous diagnoses of fibroma, lipoma, atheromas, cysts, tumors, lymphadenitis, etc. are initially established.

With subcutaneous localization, parasites can be detected by ultrasound (Fig. 68).

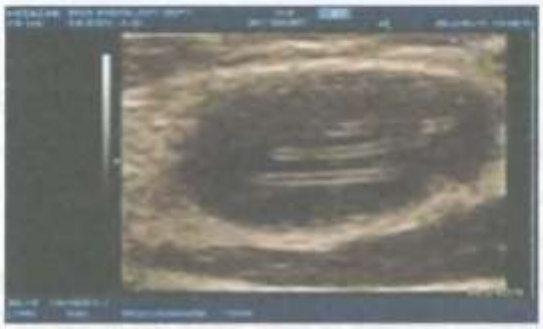

Figure 68. Dirofilaria under human skin on ultrasound.

The final diagnosis in most cases is established only after several weeks and even months from the moment the first signs of the disease appear. This often occurs during surgical procedures. Of exceptional importance in the diagnosis is the complete surgical extraction of the helminth with its subsequent study.

differential diagnosis. Subcutaneous dirofilariasis should be differentiated from diseases of the skin and subcutaneous tissue, manifested by the formation of tumors, abscesses, boils, etc.

Treatment. Surgical removal of helminths (Fig. 69).

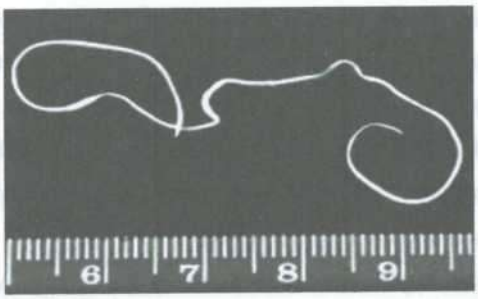

Figure 69. Dirofilaria female repens extracted from under human skin.

Since microfilariae are not found in human blood, the appointment of microfilaricidal drugs is not indicated. The use of anthelmintic drugs (diethylcarbamazine, ivermectin) did not justify itself, because if the helminth dies, the patient may develop a pronounced toxic-allergic reaction.

Prevention consists in protecting against mosquito bites, conducting seasonal disinsection treatment of water bodies and examining dogs in veterinary institutions for dirofilariasis. In some countries, prophylactic treatment of dogs with anthelmintic drugs is recommended.

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