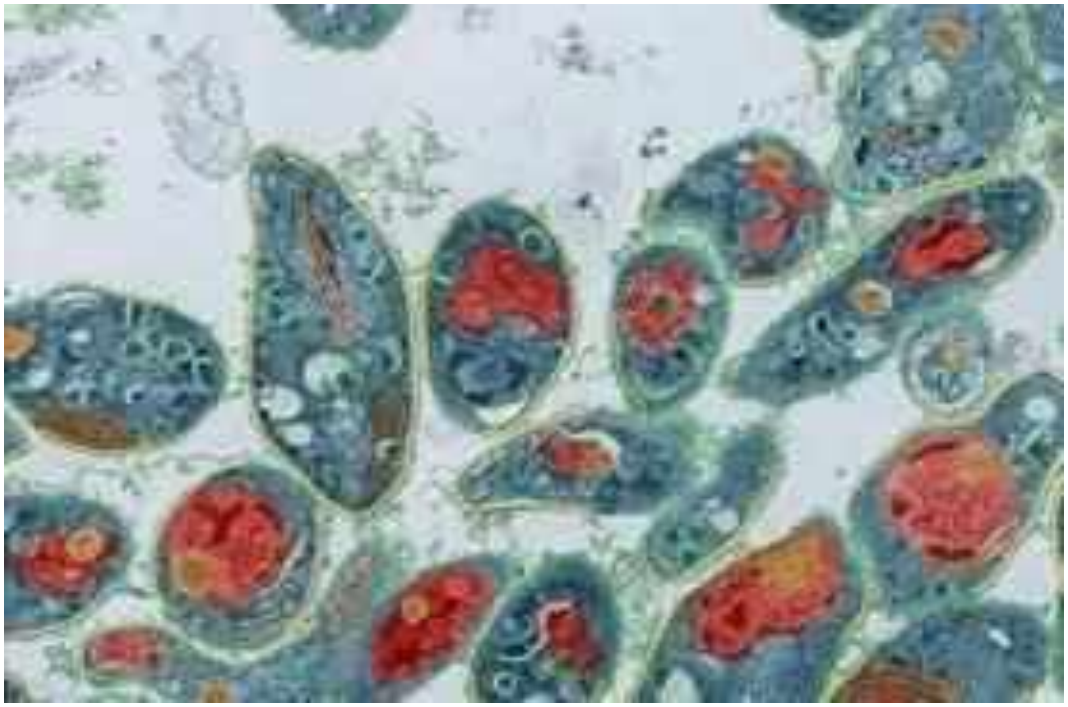
MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHIA STATE MEDICAL AND PHARMACEUTICAL UNIVERSITY
Department of Infectious Diseases

CLINICAL PARASITOLOGY AND TROPICAL MEDICINE



(elective course)
for 6th year medical students
Master of Medicine
specialities 222 "Medicine"

2024

UDC 616.99+616.928/.93](075.8.034.2)

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Approved at the meeting of the Central Methodological Council of the ZSMPHU and recommended for use in the educational process (Minutes No. ___ of _____2024)

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К 49 **Clinical parasitology and tropical medicine** : (elective course): online course scenario for 6th year students of Master of Medicine, specialities 222 "Medicine"/ comp. : O.V. Riabokon, T.E. Onishchenko, O.O. Kornienko [et al.]. – Zaporizhzhia : [ZDMPhU], 2024. - 222 p.

The course material introduces the basic concepts, terms and definitions of clinical parasitology and tropical medicine. The course covers the etiology, epidemiology, pathogenesis, clinical features, diagnosis, and treatment of tropical and parasitic diseases. The online course scenario is designed for 6th year students of the Master of Medicine specialities 222 "Medicine

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Performers:

NAME	Contents.	Signature
Module 1 Clinical parasitology and tropical medicine		
Module 1: Clinical parasitology and tropical medicine. Parasitoses.		
Ass.Professor Onishchenko T.E. Ass. Professor Kornienko O.O.	Topic 1: Features of the course of infectious diseases in countries with tropical climates. Diseases of travellers, principles of prevention. Features of travellers' diarrhoea. Protozoal invasions of the intestine: amoebiasis, balantidiasis, giardiasis (differential diagnosis).	
AssProfessor Onishchenko T.E.	Topic 2. Differential diagnosis of leishmaniasis. Differential diagnosis of trypanosomiasis. Problems of malaria. Complicated forms of malaria. The treatment of malaria caused by resistant strains of pathogens. Modern prevention.	
Trainee teacher Andrieieva V.S.	Topic 3: Other protozoan diseases: babesiosis, toxoplasmosis, pneumocystis, cryptosporidiosis, isosporosis, acanthamebiasis. Leprosy. Monkeypox.	
Trainee teacher Andrieieva V.S.	Topic 4. Worm infestations of tropical and subtropical regions: ankylostomiasis, clonorchiasis, fascioliasis. Paragonimiasis. Strongyloidiasis as an HIV-associated helminthiasis. Dracunculiasis.	
Ass. Professor Onishchenko T.E. Ass. Professor Kornienko O.O. PhD,as. Bilokobyla S.O.	Topic 5. Onchocerciasis, loiasis, hookworm, brugiasis. Differential diagnosis of filariasis.	
Ass.Professor Onishchenko T.E.	Topic 6. Differential diagnosis of schistosomiasis.	
Ass. Professor Kalashnyk K.V.	Topic 7. Zoonoses: scrapie, melioidosis, sodoc, streptobacillosis, listeriosis, foot and mouth disease.	
Module 2. Clinical parasitology and tropical medicine. Diseases caused by viruses and those controlled by IHR.		
PhD, Ass. Professor Kalashnyk K.V., PhD, as. Bilokobyla S.O.	Topic 8: Arboviral infections. Features of the clinical course. Hemorrhagic fevers: Ebola, Lassa, Marburg, dengue, chikungunya, South American haemorrhagic fevers. Phlebotomous fever. Encephalitis: California, Venezuelan, American	

	equine, Japanese, Rift Valley. The concept of rotary typhus.	
As.Venitskaya G.V.	Topic 9: Peculiarities of rickettsiosis: Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia. Q fever. Bartonellosis. Ehrlichiosis.	
PhD, as. Bilokobyla S.O.	Topic 10. Infectious diseases included in the list of events that may constitute a public health emergency on an international scale and fall under the International Health Regulations 2005. Pneumonic plague, yellow fever, West Nile fever.	

Project Manager:

Head of the Department of Infectious Diseases of the ZSMFU, Doctor of Medicine, Professor — Olena RIABOKON

Introduction

Infectious diseases of parasitic etiology and helminthiasis are more common in countries with hot (tropical, equatorial) climates and are atypical for the European continent, but due to the expansion of international relations and the global redistribution of labour resources, migration processes, growing urbanisation, military conflicts, and the rapid development of the tourism industry, they can pose an epidemiological threat to other regions. The course of the discipline "Clinical Parasitology and Tropical Medicine" (elective course) for self-study online is designed in accordance with the curriculum for 6th year students of Master of Medicine specialities 222 "Medicine".

Specific objectives, purpose

The purpose of studying the discipline "Clinical Parasitology and Tropical Medicine" is to find and clarify the current patterns of spread and damage to the human body in a variety of tropical infectious diseases. The student must master the proposed material, acquire basic skills and be able to use the acquired knowledge and skills in practical activities.

The main objectives of the discipline "Clinical Parasitology and Tropical Medicine" are to find and clarify the current patterns of spread and damage to the human body in a variety of tropical infectious diseases:

- Identify the main clinical symptoms that form a characteristic syndrome for the most common tropical infectious diseases and parasitic infestations;

- Make a preliminary diagnosis of the most common tropical and subtropical infectious diseases and parasitic infestations (syndromic and etiological);

- Make a preliminary clinical diagnosis, plan preventive and quarantine measures for the most common subtropical and tropical infectious diseases and parasitic infestations;

- Interpret the patterns and features of the pathological and epidemiological process in subtropical and tropical infectious diseases;

- Perform differential diagnosis of subtropical, tropical infectious and parasitic diseases with non-infectious diseases;

- Interpret the results of specific examination methods in the presence of combined pathology - infectious and non-infectious;

- Determine the tactics of managing patients with the most common tropical infectious and parasitic diseases;

- Determine the tactics of hospitalization and isolation of infectious patients;

- Diagnose emergency conditions and provide pre-hospital care;

- Demonstrate awareness of tropical infectious diseases as weapons of mass destruction;

- Predict the effects of subtropical, tropical infectious and parasitic diseases on human health.

- Demonstrate the ability to maintain medical records in an infectious disease clinic.

- Researching ways of spreading the disease among different risk groups

The student should know:

- the fundamentals of healthcare legislation and policy documents that determine the activities of healthcare authorities and institutions;
- general issues of organizing medical and psychological care in Ukraine;
- organizing the work of hospital and outpatient departments;
- organization of ambulance and emergency care for the adult population;
- organization of dispensary supervision;
- anti-epidemic measures in the event of an infection;
- Principles of diet therapy, physiotherapy and exercise therapy;
- forms and methods of sanitary and educational work;
- modern literature in the field;

List of skills:

1. Be able to justify a preliminary clinical diagnosis of the most common tropical infectious diseases and parasitic infestations.

2. Be able to recognise complications and emergencies in patients with the most common tropical infectious diseases and parasitic infestations.

3. To be able to prescribe a plan for the examination of patients with the most common tropical infectious diseases and parasitic infestations.

4. To perform clinical and laboratory differential diagnosis of tropical infectious diseases and parasitic invasions.

5. Prescribe rational treatment for patients with tropical infectious diseases and parasitic infestations at different stages of medical care.

6. Be able to provide emergency care to patients with tropical infectious diseases and parasitic infestations.

7. Plan basic preventive measures for tropical infectious diseases and parasitic infestations.

8. Be able to justify the preliminary clinical diagnosis of tropical infectious diseases and parasitic infestations.

9. Be able to recognise complications and emergencies in patients with tropical infectious diseases and parasitic infestations.

10. Be able to prescribe a plan for the examination of patients with tropical infectious diseases and parasitic infestations.

11. Perform clinical and laboratory differential diagnosis of tropical infectious diseases and parasitic infestations.

12. Prescribe rational treatment for patients with tropical infectious diseases and parasitic infestations.

13.Be able to provide emergency care to patients with tropical infectious diseases and parasitic infestations.

14. Plan basic preventive measures against tropical infectious diseases

Technical and software equipment:

- A personal computer, laptop, tablet, smartphone or similar device,
- Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
- Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.

- Access to the global network, local network,
- Access to the electronic library of online courses of ZSMFU.

Glossary of terms and glossary for each topic

The theoretical material is presented in information blocks in a sequential viewing mode with links and video fragments: presentations, summaries, and Internet links. Knowledge control: multiple-choice testing.

MODULE 1. CLINICAL PARASITOLOGY AND TROPICAL MEDICINE. PARASITOSES.

Topic 1: Features of the course of infectious diseases in countries with tropical climates. Diseases of travellers, principles of prevention. Features of travellers' diarrhea. Protozoal invasions of the intestine: amoebiasis, balantidiasis, giardiasis (differential diagnosis).

Learning objective: to gain basic knowledge of the peculiarities of infectious diseases in countries with tropical climates.

List of skills:

- adhere to the basic sanitary and anti-epidemic rules for working at the bedside of patients with amoebiasis, balantidiasis, giardiasis;
- take a medical history and assess epidemiological data;
- examine the patient and identify the main symptoms and syndromes of amebiasis, balantidiasis, giardiasis, and substantiate the preliminary diagnosis;
- determine whether the patient has specific complications;
- conduct a differential diagnosis of amebiasis, balantidiasis, giardiasis;
- to draw up medical documentation on the fact of establishing a preliminary diagnosis of amebiasis, balantidiasis, giardiasis (emergency report to the district epidemiological department - F.058);
- draw up a plan for laboratory and additional examination of the patient;
- interpret the results of laboratory tests, including specific diagnostic methods;
- draw up an individual treatment plan, taking into account epidemiological data, clinical form of the disease, severity of the course, presence of complications, allergic history, concomitant pathology, and prescribe prescriptions;
- provide assistance in case of emergency.
- provide recommendations on regimen, diet, examination, and supervision during the recovery period.

Hardware and software:

- A personal computer, laptop, tablet, smartphone or similar device,
- Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
- Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
- Access to the global network, local network,
- Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Amoebiasis, amoeba, balantidiasis, giardiasis, anthroponosis, disinfection, decontamination, decontaminated groups, jaundice, bleeding, peritonitis, liver failure, protozoal diseases.

BALANTIDISIS is an infectious disease that belongs to the group of protozoan infections. New cases of the disease are rarely reported, but 5% of the rural population is carrier of the parasite. It enters the human body through the faecal-oral transmission mechanism from infected animals (pigs).

The causative agent of balantidiasis - Balantidium coli - is a pathogenic microorganism that belongs to eukaryotes, type - infusoria, genus - balantidium. It has a cystic or resistant form, and a trophozoite is a vegetative form. The morphological features of the vegetative form of these protozoa include: large size - up to 200 μm (longitudinal size) and up to 70 μm (transverse size); the presence of a special shell - covered with a thin membrane called a pellicle. It helps to preserve the shape of the parasite; ability to move - on the surface of the pelicle there are cilia that ensure the movement of infusoria in the contents of the large intestine; Balantidium coli has a piriform - a funnel-shaped depression at the front end of the body, surrounded by cilia that help in food capture. The mouthpiece (cytostome) at the bottom of the peristome is connected to the funnel-shaped cytopharynx, through which food is taken in and further digested. Food residues are removed through the cytopig (anal pore) located at the opposite end of the infusoria's body; bilayer structure of the cytoplasm - the ectoplasm is a thin outer layer, while the endoplasm is a cloudy, granular and opaque mass located inside; the presence of 2 nuclei - there are 2 nuclei in the cytoplasm of the parasite (a large nucleus - macronucleus, which contains chromatin and is

responsible for the processes of cell vital activity, a small nucleus - micronucleus, functionally connected with the large nucleus, its main task is to preserve the integrity of genetic information); the presence of 2 contractile vacuoles - 1 of them is located in the terminal part of the infusoria, and the 2nd - in the centre of its body. Both vacuoles are filled with various elements, including starch grains, red blood cells, and other smaller microorganisms. The cyst is smaller, reaching a diameter of 50 μm. Its shell consists of 2 layers that reliably protect the parasite from adverse factors. In the environment, cysts can remain viable for 3-4 weeks. They can survive in soil for more than six months, in disinfectant solutions for several hours, and in gastric juice for 12 hours.

Epidemiology of balantidiasis.

The source of infection is the faeces of convalescent or sick pigs. The mechanism of infection with balantidiosis is faecal-oral. Ways of transmission:

- water (contaminated drinking water, polluted water resources, poorly treated wastewater);
- foodborne - balantidia enter the human digestive tract with food; zoonotic - infection occurs through direct contact with infected animals.

Rats, mice and insects can transmit balantidiasis.

The infection is most often diagnosed in people living in rural areas. Despite its high contagiousness, the majority of those infected are asymptomatic or have mild clinical manifestations.

Pathogenesis of balantidiasis.

Pathogenic microorganisms enter the human digestive tract in the form of spores in water, food or through dirty hands. Further in the intestine, they are transformed into trophozoites, which can exist in the lumen of the colon without causing an infectious process. This condition is called "healthy bacterial carriage". If the living conditions of the balantidia become unfavourable, the parasites are activated and penetrate the mucous membrane of the intestinal wall. The activating factors may include a lack of nutrients (sugars), worm infestations, concomitant chronic pathology of the digestive tract in the acute stage, and other factors. After penetrating the intestinal

mucosa, parasites begin to multiply actively. This process is accompanied by the development of inflammatory changes: areas of hyperaemia and swelling appear; an infiltrate is formed, consisting of different types of immune cells (lymphocytes, histiocytes and neutrophils).

Inflammatory changes are most often localised in the colon, at the level of the ascending sections. As the infection progresses, erosions and ulcers appear on the mucous membrane. The ulcers are located along the folds of the mucous membrane and in places of kinks in the intestinal wall. They have irregular, undermined edges and are often covered with jelly-like, blood-purulent masses of dirty black colour. Ulcers can reach significant sizes (up to several cm^2). Necrotic masses are rejected, leaving deep cavities in their place.

The clinical manifestations of balantidiasis are determined by the form of the disease.

Forms and symptoms of balantidiasis

In the subclinical form, there are no clinical signs of the disease, but rectoromanoscopy reveals characteristic catarrhal haemorrhagic changes and ulcerative defects of the mucous membrane of the distal colon with uneven, undermined edges, sometimes of considerable size; increased activity of liver transaminases and eosinophilia are detected in the blood.

The acute form of the disease is characterised by the development of an intoxication syndrome and colon damage. Intoxication in balantidiasis develops as a result of exposure to toxins, waste products of balantidia and inflammation in the intestine. The intoxication syndrome includes: increased body temperature; general weakness; decreased activity; pain and discomfort in the abdomen, especially in the lower abdominal cavity. The severity of the pain may increase after eating; nausea and vomiting; loss of appetite, anorexia, weight loss.

Intestinal damage is characterised by the development of diarrhoea - stools become liquid and frequent, with an odour indicating putrefaction; mucus and blood appear in the stool; the daily number of stools can increase up to 20 times or more. Insufficient or no therapy leads to chronicity of the process.

In the chronic form of the disease, exacerbations alternate with periods of remission. The duration of exacerbations varies from 7-30 days, and the period of remission lasts several months. In contrast to the acute form, chronic balantidiasis is characterised by less intense clinical manifestations of the disease, with intestinal disorders dominating over signs of intoxication. The chronic inflammatory process can last for years.

The chronic form of balantidiasis with a continuous course is characterised by the gradual slow development of intoxication syndrome and intestinal disorders, with no periods of remission. The most serious complication of this form of the disease is the wasting syndrome: a sharp decrease in body weight, weakness, decreased activity of physiological processes, and asthenia.

Complications of balantidiosis.

The presence of complications is determined by the depth of damage to the intestinal mucosa that causes the development:

- spilled peritonitis, one of the most serious complications of balantidiosis.

- appendicitis - inflammation of the cecum (appendix).

- pneumonia - the penetration of a pathogen into the lungs through the abdominal cavity and diaphragm. Warning signs for a doctor should include coughing, shortness of breath and fever;

- lesions of other organs are rare, but cases of parasitic lesions of the stomach, lymphatic ducts, vagina, uterus, bladder, and liver have been reported).

Diagnosis of balantidiosis.

The final diagnosis is based on the medical history, epidemiological history and the results of clinical and laboratory tests. An objective examination reveals the development of hepatoliver syndrome, liver pain, swollen lymph nodes, fever, tachycardia, low blood pressure, heart rhythm disturbances, weight loss, and exhaustion.

The results of laboratory tests reveal moderate leukocytosis with a shift in the leukocyte formula to rods, eosinophilia, an increase in the erythrocyte sedimentation rate to 30-40 mm/h; a decrease in plasma protein levels, increased α-2 and γ-globulins

with a decreased albumin level, increased activity of liver enzymes; colonoscopy reveals intestinal ulcers of various sizes.

The main method of specific diagnosis of balantidiasis is parasitoscopic. It includes 2 main ways of detecting parasites:

1. Detection of vegetative forms and cysts of the pathogen in faeces - the peculiarity of this method is the need to comply with the deadlines. Faecal samples should be submitted for examination within the first 20 minutes after the act of defecation (vegetative forms of parasites die quickly outside the human body!).

2. Examination of biopsies of ulcer edges or smears of ulcer contents is performed in the presence of ulcerative lesions of the intestine.

The parasitoscopic method allows for the accurate identification of balantidiasis if the material is examined within the required timeframe.

Treatment. In the treatment of balantidiasis: it is recommended to follow a diet that helps to reduce intestinal irritation and severity of symptoms of the disease, which includes (exclusion of spicy and fatty foods; easily digestible foods; plenty of drinking; foods rich in proteins, vitamins and minerals (dairy products, fruits and vegetables, lean meat and fish); exclusion of alcohol and coffee; moderate portions; compliance with hygiene standards.

Etiotropic therapy includes antimicrobial drugs: tetracycline - per os 500 mg 4 times a day for adults, 12.5-25 mg/kg body weight 2 times a day for children over 8 years of age; metronidazole - orally 500 mg 2-3 times a day for adults, for children the daily dose varies between 250-500 mg depending on age; tinidazole - for adults the recommended dose is 2 g per day for 3-5 days, for children -50 mg/kg body weight per day divided into 2 doses). The duration of the course of etiotropic therapy is 5 days. It is recommended to conduct 3 courses of treatment at 5-day intervals.

Pathogenetic therapy includes detoxification and rehydration therapy, electrolyte correction.

Symptomatic therapy: antispasmodics, antiemetics, etc. Vitamin therapy. Due to impaired absorption of nutrients and vitamin and mineral deficiencies, it is

recommended to take ascorbic acid (600-2000 mg), B vitamins, iron preparations, folic acid:

Prevention of balantidiosis includes: sanitary and hygienic measures; avoidance of contaminated water and food sources; heat treatment of food; and sanitary control of pigs. There is no specific prevention of balantidiosis.

The **prognosis** of balantidiosis is determined by the form of the disease, its severity, and the availability of timely and effective therapy. As a rule, the prognosis for patients is favourable, but in case of complications or acute infection with critical dehydration, fatal consequences are possible.

GIARDIASIS, also known as giardiasis, is an infectious disease caused by the parasitic protozoan Giardia lamblia (or Giardia intestinalis). The pathogen lives in the small/duodenum of humans and some animals, causing a variety of symptoms, including diarrhoea (persistent), bloating, abdominal pain and fatigue. Giardiasis is transmitted by the faecal-oral route, usually through contaminated water or food. In the environment, the parasites survive as cysts. Cysts can be found in soil, food, water, or on surfaces that have been contaminated by the faeces of an infected person or animal.

Giardiasis is one of the most common parasitic intestinal diseases in the world. It is prevalent in all countries, but is most common in regions with poor sanitation and hygiene. The life cycle of the parasite includes two stages: a cyst and a vegetative form (trophozoite). Ingestion of 10-100 cysts leads to infection. Under the influence of hydrochloric acid, trophozoites are released from the cysts, which attach to the mucous membrane of the small intestine, destroying the brush border of enterocytes and the structure of villi. This leads to a decrease in the absorption surface. Under the influence of an alkaline environment (bile), trophozoites transform back into cysts, which are then excreted in the faeces.

The reservoir for the parasite is humans (mainly) and many species of domestic (dogs, cats) and wild (e.g. beavers) animals. The infection is usually spread through the diet, mainly through contaminated hands or water, and rarely through food contaminated with cysts. Giardia lamblia is distributed worldwide, with sporadic cases

in developed countries, but epidemics can occur due to contaminated drinking water or the spread of infection in closed environments.

The incubation period of the disease is from several days to several weeks (9-10 days in total). The patient can be a source of infection for others. The cysts remain virulent in a damp, cool environment for several months and are resistant to chlorine.

The clinical presentation of giardiasis can vary depending on the individual and the stage of the disease. Some people may be infected with Giardia and not have any symptoms, while others may have a variety of symptoms. Forms of the disease

- asymptomatic;
- intestinal (manifested in the form of gastritis, causing severe discomfort in the stomach, nausea and dyspeptic disorders);
- reflex (colic is observed in the hypochondrium, a strong taste of bitterness prevails in the mouth, and pancreatitis develops over time);
- astheno-neurotic (characterised by lethargy and increased fatigue, the disease develops against a background of frequent stress and anxiety);
- toxic-allergic (skin rashes, possible asthmatic attacks);
- mixed (combining different forms).

The duration of the disease affects its form and course. For example, up to 30 days is acute, more than 1 month is subacute, and more than 90 days is chronic.

Clinical forms of giardiasis:

Asymptomatic is the most common form of the disease, which in most cases goes away on its own.

Acute - acute gastroenterocolitis. This condition usually lasts 7-14 days. If left untreated, it can become chronic in 30-50% of patients. The main symptoms include diarrhoea (watery, without blood or mucus in the stool) and cramping pain in the epigastric region. Other symptoms may include weakness, bloating, loss of appetite and weight, and sometimes vomiting and fever.

Chronic form - the development of a chronic syndrome of gastrointestinal disorders with impaired absorption (steatorrhoea). Symptoms are similar to acute

symptoms, but less severe and recurrent. The appearance of atypical symptoms includes urticaria, reactive arthritis. Secondary lactose intolerance, cachexia, cholangitis and cholecystitis may develop. After the disease, the immune system is not stable, which determines the possibility of re-infection.

Symptoms of giardiasis:

Diarrhoea (persistent) is the most common symptom of giardiasis. The stools may be loose, greasy or foamy, sometimes with an unpleasant odour. Abdominal pain - localised in the upper abdomen and may be accompanied by bloating.

Nausea and vomiting can lead to loss of appetite and ultimately weight loss. Giardiasis can cause general weakness and fatigue, which may be associated with diarrhoea and weight loss. Bloating and excessive gas production are caused by digestive disorders. In some cases, giardiasis can be a trigger for the development of irritable bowel syndromes, chronic fatigue and food intolerance.

If left untreated, specific complications may develop: skin rashes (urticaria), arthritis, subcutaneous swelling, severe itching (Quincke effect), ophthalmic disorders (visual impairment), weak muscle tone (hypoglycaemic myopathy).

Diagnosis.

The diagnosis of giardiasis involves analysing faeces for the presence of cysts or trophozoites of the parasite. In some cases, several faecal samples may be required, as the number of parasites can vary.

Specific diagnosis of giardiasis:

1. Parasitoscopic faecal examination. This is the most common method of diagnosing giardiasis. Giardia cysts or trophozoites are looked for in faecal samples. In some cases, several faecal samples may be required - 2-3 times with an interval of 1-2 days.

2. A parasitoscopic examination of the duodenal contents, usually performed by probing, can be used to diagnose giardiasis, although it is rare. This method can be used if stool tests and serological tests have not confirmed the diagnosis.

3. Endoscopic method. Morphological examination of the biopsy of the duodenal mucosa or small intestine can be performed in some cases, if there are

indications for endoscopy. Microscopic examination of the biopsy reveals morphological signs of giardiasis: atrophy of small intestinal villi and trophozoites of giardia, which are visible on the surface of the mucous membranes.

4. ELISA test. Confirms the presence of antibodies to giardia in the blood. They may be necessary if there are symptoms of giardiasis, but the faecal test does not show the presence of parasites. However, it should be remembered that after treatment, antibodies will be detected in the blood plasma. The first to be detected in the blood are IgM (on the 10-14th day after infection), then IgG, which are detected in the blood plasma at almost all stages of giardiasis, their level decreases 1-2 months after the death of the parasite. The presence of positive IgG may indicate an ongoing infection, previous giardiasis, or an asymptomatic course of the disease.

5. PCR. It is aimed at detecting Giardia intestinalis antigens or DNA in faecal samples:

Treatment

Treatment of giardiasis usually involves taking antihelminthic, antiprotozoal drugs. Here are some of the most commonly used medicines:

1. Tinidazole. Adults are prescribed 2 g once.
2. Metronidazole. Adults: 250 mg 3 p/day; 500 mg 2 p/day; Number of days of administration: 5-7.
3. Albendazole. Children (3-12 years): 400 mg 1 p/day for 5 days.
4. Mebendazole. 200 mg 3 times daily for 5 days.
5. Monitoring the effectiveness of giardiasis treatment.

The criterion for recovery from giardiasis is the absence of parasites in the faeces 2-4 weeks after the end of treatment. It is important that even after successful treatment, relapses of the disease may occur. They usually occur 2-8 weeks after treatment and may be asymptomatic. This demonstrates the importance of regular medical monitoring and adherence to preventive measures to prevent re-infection.

Prevention of giardiasis includes a number of measures aimed at preventing the infection and spread of the infection.

AMEBIASIS is a protozoan anthropogenic disease that manifests itself mainly as ulcerative damage to the large intestine, as well as the development of abscesses in the liver and other organs in clinically severe cases.

- The causative agent is Enthamoeba histolytica
- Mechanism of transmission - faecal-oral
- Ways of transmission
  - Water
  - Alimentary
  - Contact and household

1873 - Fyodor Alexandrovich Lesh discovered the causative agent of the disease in the faeces of a patient with bloody diarrhoea. He named the discovery Amoeba coli.

1883 - Robert Koch, while working in Egypt in the centre of the dysentery epidemic, discovered amoebae in histological sections of liver ulcers and abscesses.

1903 - Fritz Schaudinn describes the pathogenic dysentery amoeba in detail, naming it Enthamoeba histolytica, and also isolates and describes the non-pathogenic intestinal amoeba Enthamoeba coli.

Epidemiology

Anthropogenic infection

Amoebiasis is one of the most important medical and social problems in the western and southeastern regions of Africa, Southeast Asia, China, and Latin America, where the number of people infected with dysentery amoebae is 20-70%.

In our country, sporadic cases of amoebic dysentery occur mainly in the south.

In the CIS countries, the most disadvantaged are the states of Central Asia and the Caucasus, where 15-35% of the population is carrier.

Because trophozoites die rapidly after being shed from the intestine, asymptomatic cyst carriers are a source of new infections; chronic carriers can shed several million cysts per day. The spread of the infection is facilitated by poverty and other factors that lead to poor personal hygiene. Transmission through food and water is reported in countries and areas of the world with low levels of sanitation.

Risk groups

Risk groups for amoebiasis infection:

- patients with pathology of the gastrointestinal tract,
- residents of non-sewerage settlements,
- persons of decree groups,
- people who have returned from amoebiasis-endemic countries,
- homosexuals.

Pathogenesis of Entamoeba histolitica

Life forms:

- **large vegetative** Entamoeba histolitica forma magna (15 to 45 microns in diameter, can extend up to 60-80 microns).
- **small vegetative** Entamoeba histolitica forma minuta (7-25 μm).
- incystic - cyst (9-15 μm) - oval, stationary behind a double-circular shell.

Amoebae are actively mobile at T +28-48°C due to ectoplasmic

Pseudopodia. It promotes the transformation of small vegetative forms into large ones:

- Overheating, gastrointestinal ischaemia → reduced resistance;
- Changing your diet;
- Carbohydrate diet → ↓ intestinal pH → dysbiosis;
- Immunodeficiency states;
- Intestinal diseases;
- Vitamin deficiency;
- High virulence of the pathogen strain.

Once in the gastrointestinal tract, the cysts begin to divide; in the small intestine, the cyst membrane breaks down, releasing trophozoites. Immature amoebae reach the colon, where they live, feeding on bacteria and organic matter residues. Amoebae are able to enter the portal vascular system and localise in the venules; necrosis of the liver tissue leads to the formation of an abscess. In rare cases, as a result of embolism, abscesses can form in the lungs, brain or spleen.

Amoebic ulceration of the intestinal wall is characteristic: a small area of mucosal damage masks a rather deep zone of necrosis in the submucosa and muscle membranes. The ulcer has the form of a plaque. The signs of acute inflammation are mild, and unlike ulcers in bacillary dysentery, the areas of the mucous membrane between the ulcers look normal. In the chronic process, large masses of granulation tissue or amoebas may form in the cecum, sigmoid, and colon.

Clinical picture

According to the WHO classification, there are :

- asymptomatic
- manifest amoebiasis, among other things:
    - intestinal (Amoebic dysentery and dysentery amoebic colitis) and
    - extraintestinal (liver abscess; pulmonary and other extraintestinal lesions).

Incubation period from 1-3 weeks to 3 months

AMEBIC DYSENTERY (dysentery colitis) is the main and most common clinical form of the disease and can occur acutely and chronically, in severe, moderate and mild forms.

The main clinical signs of the disease are diarrhoea: in the initial period, up to 4-6 times a day, abundant faeces with mucus, then up to 10-20 times a day with blood and mucus, loss of faecal character. The faeces look like "raspberry jelly". The disease usually develops gradually, without signs of general intoxication, and the body temperature is normal or subfebrile. In severe infection, there are pulling or cramping pains in the lower abdomen, which increase during defecation. Painful tenesmus appears. In severe colitis, signs of intoxication increase, which is manifested by fever (usually of the wrong nature), decreased appetite, nausea, and sometimes vomiting. The abdomen in the acute period is soft, painful along the colon.

Remission period

The acute process lasts no more than 4-6 weeks, followed by remission lasting from several weeks to 1 or more months. After remission, the disease recurs and becomes chronic, which can last for years without specific treatment.

The chronic process takes place in the form of recurrent or continuous forms. *In the recurrent form*, exacerbations are followed by remissions, during which patients notice only minor dyspeptic phenomena (mild flatulence, abdominal rumbling, pain without a specific localisation). During exacerbations, the patients' health is not significantly impaired, and their body temperature remains normal. At this time, there are severe pains in the right side of the abdomen, in the ileocecal region (appendicitis is often mistakenly diagnosed), and bowel movements.

Continuous chronic course

In ***the*** case of ***a continuous course of*** chronic amoebiasis, there are no periods of remission. The disease occurs with an increase in the manifestations of the disease (abdominal pain, diarrhoea alternating with constipation, faeces with blood, sometimes fever), and then with their weakening. In case of prolonged chronic intestinal amoebiasis, patients become exhausted, their working capacity decreases, asthenic syndrome and hypochromic anaemia develop, the liver often enlarges, eosinophilia, monocytosis, and in severe cases, cachexia are observed. In chronic intestinal amoebiasis, asthenic syndrome, vitamin, protein and energy deficiency develop. Patients complain of lack of appetite, unpleasant taste in the mouth, and weakness. On examination, the patient's facial features are sharpened, the patient is pale, the tongue is covered with a white or grey coating, the abdomen is usually retracted, palpation is painless or moderately painful in the iliac region. Many patients have severe symptoms of cardiovascular pathology: muffled heart sounds, tachycardia, and pulse lability. Rectoromanoscopy reveals ulcers, polyps, cysts, amoebas.

Intestinal amoebiasis:

Complications of intestinal amoebiasis include:

- perforation of the large intestine wall,
- development of purulent peritonitis,

- bleeding, appendicitis,
- colon strictures,
- amoeba,
- megacolumns, etc.

The most severe complication is perforation and gangrene of the colon, with a mortality rate of 100% among unoperated patients.

Extraintestinal amoebiasis occurs as a complication of intestinal **amoebiasis** as a result of haematogenous or direct introduction of amoebae from the intestine.

Most often, it manifests itself in the form of amoebic hepatitis or liver abscess, which are acute, subacute or chronic. Liver damage can occur during the development of acute amebic colitis, or several months or even years after infection. Acute amoebic hepatitis is more often formed against the background of intestinal amoebiasis. In this case, the liver is enlarged, compacted, moderately painful; the temperature is subfebrile. Hepatomegaly may develop.

Amoebic liver abscess

Amoebic abscesses are characterised by liver enlargement, pain, high fever (up to 39 °C) of remitting, hectic or persistent type with chills and profuse sweating at night. Single or multiple abscesses are more common in the right lobe of the liver. With large abscesses, jaundice may develop, which is a poor prognostic sign. If the diaphragm is involved in the pathological process, high standing of its dome and restriction of mobility are detected. Development of atelectasis is possible. In 10 - 20% of patients, there is a prolonged, latent, or atypical course of abscess (for example, only fever, pseudocholecystitis, jaundice) with possible subsequent breakthrough, which can lead to the development of peritonitis and damage to the chest cavity organs. In case of amoebic liver abscess, indications of previous intestinal amoebiasis are detected only in 30-40% of patients, and amoebae are found in faeces in less than 20% of patients.

Diagnostics

The diagnosis of amoebiasis is made on the basis of epidemiological history, clinical presentation of the disease and laboratory test results.

The results of a parasitological examination are decisive for the diagnosis. The parasitological diagnosis of amoebiasis is made when tissue and large vegetative forms, trophozoites-erythrophages are found in the test material. The material for the study is faeces, rectal swabs taken during rectomanoscopy; biopsy material from peptic ulcers, aspirate of liver abscess contents, with tissue forms being localised mainly in the outer walls of the abscess, rather than in necrotic masses located in the centre.

From the first day of the disease, microscopy of native smears from freshly discharged stool stained with Lugol's solution is performed. In acute and subacute variants of the disease, the vegetative tissue form of the amoeba is sought, and in convalescents and asymptomatic carriers, a small luminal form and cyst are found. Detection of luminal forms and cysts in the faeces alone is not sufficient for a definitive diagnosis. To increase the efficiency of parasitological studies, multiple (up to 3-6 times) examination of freshly excreted faeces (no later than 10-15 minutes after defecation) and other biological substrates, collection of material in a preservative liquid for long-term storage of the preparation, and enrichment methods are used.

In the presence of clinical signs of intestinal amoebiasis and negative results of parasitological studies, serological reactions based on the detection of specific antiamoebic antibodies are used. RIF, ELISA, ELISA, hemagglutination inhibition and neutralisation reactions with paired sera are used (an increase in antibody titer by 4 or more times is a diagnostic titer). Serological tests are positive in 75% of patients with intestinal amoebiasis and 95% of patients with extraintestinal amoebiasis. To establish the diagnosis of extraintestinal amoebiasis, in addition to immunological tests, a comprehensive instrumental examination is performed: Ultrasound, X-ray examination, computed tomography and other methods that allow determining the location, size and number of abscesses, as well as monitoring the results of treatment. Modern testing methods include the detection of dysentery amoebae antigens in faeces and other material using monoclonal antibodies; determination of parasitic DNA by PCR

Treatment and prevention of amoebiasis

Etiotropic therapy

Media sanitation: Yatren 650 mg 3 times daily for 20 days per os; Metronidazole 750 mg 3 times daily for 10 days per os.

Extraintestinal amoebiasis: Metronidazole IV drip 750 mg 3-4 times daily for 10 days; Tinidazole.

Intestinal amoebiasis: Metronidazole 500 mg IV drip 4 times daily;

Tinidazole 30 mg/kg/day for 3 days.

Dispensary observation

Follow-up of patients is carried out for 12 months. Medical observation and laboratory tests are carried out once a quarter, as well as in case of intestinal dysfunction. Persons of maternity leave groups infected with dysentery amoeba are registered with the dispensary until they are completely cured of the amoebic pathogen.

Prevention

Personal - compliance with personal hygiene rules. Measures aimed at disrupting the transmission mechanism include protection of environmental objects from contamination with invasive material (sewage of settlements), provision of good-quality drinking water and food, disinfection of objects contaminated with patient secretions in healthcare and other facilities. Identification, isolation and treatment of carriers and patients. No specific prophylaxis has been developed.

TRAVELLER'S DIARRHOEA is one of the most common and unfavourable health conditions for travellers. The disease can be quite long, painful and debilitating. According to the CDC, the US Centre for Disease Control, 20% to 50% of international tourists (about 10 million people) suffer from traveller's diarrhoea in the first week of their trip, but the disease can develop throughout the entire period of stay in certain countries and even after returning home. A significant proportion of traveller's disease cases occur in Latin America, Africa, the Middle East and Asia. The high-risk group for this disease includes young and elderly people, people with weakened immune systems, chronic inflammatory diseases of the gastrointestinal tract, or diabetes, as well as tourists taking antacids or H2 blockers. Currently, the main cause of traveller's diarrhoea is infectious agents, the most common pathogen being enterotoxigenic

Escherichia coli and other pathogens belonging to a new group of pathogenic bacteria - superbugs. In industrialised countries, the causative agent of acute diarrhoeal disease is usually a virus, and sometimes Clostridium difficile in patients who have taken antimicrobials. Diarrhoea in travellers ("traveller's diarrhoea") returning from a country with poor hygiene is usually caused by a bacterium, rarely a virus or parasite. Approximately one third of travellers are found to have more than one type of pathogenic bacteria.

Etiology and epidemiology. The period of global changes taking place on Earth (climatic, migration, technical and other) requires new approaches to assessing medical risks that any person may face One of these areas of medical development is travel medicine, which has recently become widespread and defined against the background of a significant number of people (about 2 billion) who make various trips every year for tourism, humanitarian missions, globalisation of production or migration of large segments of the population as a result of wars and search for work and safe places to live.

Traveller's diarrhoea is characterised by more frequent stools than usual for the individual, or by loose or watery stools within 24 hours (WHO). The source of contamination in most cases is food or drink contaminated with faeces. The regions with the highest risk of traveller's diarrhoea are: South Asia (60-80%), sub-Saharan Africa, most of Central and South America (20-60%). Caribbean islands, northern and southern Africa, and Eastern Europe (8-20%). The factors that influence the risk of contracting a gastrointestinal infection are: characteristics of the microorganism (pathogenicity, infectious dose, etc.); characteristics of the host (genome, immune defence, stomach acidity, etc.). Etiology of travellers' illness: bacterial etiology (50-80% - EACP, EPCP, ETCP, Campylobacter, Salmonella, Shigella; viral etiology (5-25% - rotavirus, norovirus, adenovirus, hepatitis A virus; parasitic etiology (<10% - giardiasis, amoebiasis, cryptosporidiosis, cyclosporiasis.

Clinical picture: usually begins during the first week of travel. The average duration is 3-5 days; in 2-3% of cases, the duration of the disease exceeds 2 weeks, in 1-2%, the duration exceeds one month.

Traveller's diarrhoea is mild to moderate in more than 90% of cases and resolves without intervention; less than 1% of cases require hospitalisation. Symptoms: abdominal pain, nausea, fever, muscle pain. Invasive bacteria cause an illness that is often more severe than that caused by non-invasive bacteria (e.g. salmonella septicaemia). Cryptosporidium can cause profuse watery diarrhoea that lasts for several weeks. Amoebiasis is often characterised by bloody diarrhoea and can be severe. Severe disease can lead to dehydration and acidosis. Complications: reactive arthritis (salmonellosis, shigellosis and campylobacter infections), Guillain-Barré syndrome (campylobacteriosis), haemolytic uremic syndrome (HUS). Acute diarrhoeal disorders of travellers.

The standard **test** is performed by one of the following methods: bacteriological examination of a stool sample - the pathogen is detected in only about 15% of cases; a combination of a nucleic acid detection test based on gene amplification and stool culture; detects the pathogen in almost 80% of cases. Both tests can detect Salmonella, Shigella, Yersinia and Campylobacter. The nucleic acid test can also detect the cholera vibrio Vibrio cholerae, as well as strains of Escherichia coli that cause diarrhoea: EACP, ECCP, EICP, EICP, EHICP. The nucleic acid detection test is more sensitive and faster than bacterial culture. Specimens positive for Salmonella, Shigella, Yersinia, Campylobacter or EHCP. In a nucleic acid detection test, the pathogen culture is additionally examined and, if necessary, antibiotic susceptibility is determined (without a separate referral). Other tests for acute illness are determined according to the symptom picture

Presentations:

https://docs.google.com/presentation/d/1sXTXEHvWbtjFYGNd6oGv9GWTvd3HJUMN/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for self-monitoring

1. Name the forms of existence of the dysentery amoeba in the human body, describe them.

2. Indicate the resistance of the causative agent of amebiasis, balantidiasis, giardiasis to environmental factors.
3. Describe the source of infection, name the mechanism and main ways of infection with amoebiasis, balantidiasis, giardiasis.
4. Clinical classification of amebiasis, balantidiasis, giardiasis
5. Specify the stages of the pathological process in amebiasis, balantidiasis, giardiasis.
6. Features of the clinical course of amoebic dysentery and extraintestinal amebiasis.
7. Complications are possible with amebiasis, balantidiasis, giardiasis.
8. Plan of examination of a patient with amebiasis, balantidiasis, giardiasis.
9. Methods of specific diagnosis of amebiasis, balantidiasis, giardiasis.
10. Name the main diagnostic features of amebiasis, balantidiasis, giardiasis.
11. Make a differential diagnosis of amoebiasis with shigellosis, protozoal colitis, helminthiasis, ulcerative colitis, acute surgical diseases of the abdominal cavity, and tumours.
12. Principles of treatment of patients with amebiasis, balantidiasis, giardiasis
13. Characteristics of amebicidal drugs.
14. Rules for discharging convalescents from hospital.
15. Prevention of amebiasis, balantidiasis, giardiasis.
16. Terms of dispensary supervision.

Glossary

Amoebiasis is a protozoan anthropogenic disease that manifests itself mainly as ulcerative damage to the large intestine, as well as the development of abscesses in the liver and other organs in clinically severe cases.

Amoeboma is a tumour that develops in the rectum or colon as a result of the parasitic protozoan Entamoeba histolytica, which invades and destroys the intestinal walls.

Anthroponosis is a group of infectious diseases caused by pathogens that naturally can only parasitise the human body.

Zoonosis is a group of infectious diseases caused by pathogens that can naturally parasitise only in the body of an animal.

Declared **groups are** categories of persons at high epidemic risk: workers in the food industry, catering and food trade, employees of educational institutions, workers in the public utilities and service sector, healthcare workers, students of secondary schools and boarding schools, university students, students of colleges, technical schools and secondary vocational schools, persons from socially disadvantaged families, etc.

Protozoan diseases are infectious diseases whose causative agents are classified as protozoa. This group includes the following diseases: amoebiasis, malaria, trypanosomiasis, and leishmaniasis.

Balantidiasis - (Latin: *balantidiasis*, also sometimes called **infusoria dysentery**) is a protozoan intestinal zoonotic disease caused by a parasitic infusoria from the spiral-shaped family - Balantidium. It is characterised by ulcerative lesions of the colon and symptoms of general intoxication, and has a tendency to be prolonged and chronic. Balantidiasis should be considered by the physician in the differential diagnosis of cases of persistent diarrhoea in travellers to Southeast Asia, the Western Pacific Islands, rural South America or communities with close contact with domestic pigs.

Giardiasis is an anthroponotic infectious disease with a predominant functional disorder of the duodenum and other parts of the small intestine, which occurs due to the parasitism of flagellated protozoa - *giardia*.

Test control of knowledge

>>The most frequent haemogram changes in chronic intestinal amoebiasis are as follows

(x) Anaemia, eosinophilia, monocytosis, lymphocytosis, increased ESR

( ) Normal haemogram

( ) Leukocytosis, neutrophilia, increased ESR

( ) Leukaemia, eosinophilia, anaemia

( ) Lymphocytosis, monocytosis

>>Coprocytogram in acute manifestations of intestinal amoebiasis: <<

( ) Red blood cells are arranged in groups in the form of columns, single leukocytes

( ) Red blood cells cover the entire field of view, a small amount of mucus

( ) Red blood cells, white blood cells cover the entire field of view, amoeba cysts

( ) Moderate amount of leukocytes, mucus, amoeba cysts

(x) Large amounts of mucus, erythrocytes, eosinophils, Charcot-Leyden crystals

>>Tissue amebocides include the following drugs: <<

(x) Delagyl

( ) Metronidazole

( ) Yatren

( ) Ampicillin

( ) Azithromycin

>>Detection of small lucent forms of amoebae and cysts in the faeces indicates <<

( ) All of the following are true

(x) Carrying amoebae

( ) Period of remission of chronic amoebiasis

( ) Recuperation period of acute

>>Which research methods are the most informative for the diagnosis of extraintestinal amoebiasis: <<

( ) CT

( ) Radiography

(x) ultrasound

( ) All of the following are true

>>Indicate the nature of the stool in amoebiasis: <<

( ) Scanty, with impurities of mucus balls, blood streaks

(x) Liquid, not profuse, with vitreous mucus stained with blood

( ) Abundant, green in colour, with mucus balls and blood streaks

( ) Melena

( ) Formalised, normal colour, with blood streaks on the surface

>>Indicate the onset of amoebiasis: <<

( ) Sharp

( ) Sharp

( ) Acute with rapid development of the clinic

( ) Latent

(x) Gradual

>>Specify complications of intestinal amoebiasis: <<

( ) Colitis

( ) Intestinal bleeding

( ) Perforation of the intestine

(x) All of the following are true

>>The material for parasitoscopic examination in intestinal amoebiasis is: <<

( ) Blood

( ) Urine

(x) Stool, ulcer content

( ) cerebrospinal fluid

( ) CSF

>>Indicate the characteristic changes in the colon mucosa in acute amoebiasis: <<

(x) Mucous membrane unchanged, ulcers with swollen, undermined edges, bordered by a zone of hyperaemia, the bottom covered with pus and necrotic masses

( ) Ulcers of various diameters, cysts, polyps, amoebas

( ) Mucous membrane is swollen, hyperaemic, haemorrhages, fibrinous layers on the surface of ulcers, intestinal spasm

( ) Impoverished vascular pattern, isolated ulcers, "velvety" mucous membrane, contact and spontaneous bleeding

( ) No changes

>>Specify the causative agent of giardiasis: <<

( ) Salmonella enteritidis

( ) Salmonella typhi

( ) Salmonella paratyphi A

( ) Salmonella paratyphi B

(x) Lamblia intestinalis,

>>Indicate the source of infection in giardiasis: <<

(x) Patient with giardiasis, carrier of the pathogen

( ) Pets

( ) Cattle.

( ) Poultry.

( ) Rodents.

>>The incubation period for giardiasis is most often <<

( ) 1 -3 days

( ) 3 - 7 days

( ) 9 - 14 days

( ) 14 - 25 days

(x) 1-4 weeks

>>Specify the main syndromes of giardiasis: <<

( ) dyspeptic

( ) painful.

( ) asthenoneurotic

( ) allergic

(x) All of the above

>>Indicate the complications of giardiasis: <<

( ) dyskinesia of the bile ducts and gallbladder

( ) gastritis.

( ) pancreatitis

( ) bacterial cholecystocholangitis

(x) All of the above

>>Specify the methods of diagnosing giardiasis: <<

( ) CSF examination.

( ) A smear and a thick drop of blood.

(x) Parasitological examination of faeces and duodenal contents.

( ) Ultrasound of the abdominal cavity

( ) Bacteriological examination of faeces.

>>Specify the natural reservoir of balantidiosis: <<

( ) sheep

(x) pigs

( ) dogs

( ) cats

( ) birds

>>Specify the etiological drug for the treatment of balantidiasis: <<

(x) Tetracycline, tinidazole

( ) Metronidazole

( ) Fazizin

( ) Levomycetin

( ) Delahil

>>Specify the organs affected by balantidiasis: <<

( ) stomach duodenum, small intestine

( ) pancreas, adrenal glands

(x) blind, sigmoid, rectum

( ) hepatobiliary system

( ) genitourinary system

>>Specify specific complications of balantidiosis <<

( ) myocarditis, pneumonia

(x) intestinal bleeding, perforation of ulcers, peritonitis

( ) dysbiosis, acute respiratory failure

( ) cholecystopancreatitis

( ) Cirrhosis, malabsorption syndrome

Topic 2. Differential diagnosis of leishmaniasis. Differential diagnosis of trypanosomiasis. Problems of malaria. Complicated forms of malaria. Treatment of malaria caused by resistant strains of pathogens. Modern prevention.

Learning objective: to acquire basic knowledge and skills in malaria, leishmaniasis, trypanosomiasis. The student must master the theoretical material and be able to apply the acquired knowledge in practice. The presented theoretical materials introduce the basic concepts and terms of the topic under study.

List of skills:

- adhere to the basic sanitary and anti-epidemic rules for working at the bedside of patients with malaria, leishmaniasis, trypanosomiasis;
- take a medical history and assess epidemiological data;
- examine the patient and identify the main symptoms and syndromes of malaria, leishmaniasis, trypanosomiasis, and justify the preliminary diagnosis;
- determine whether the patient has specific complications;
- conduct differential diagnosis of malaria, leishmaniasis, trypanosomiasis;
- to draw up medical documentation on the fact of establishing a preliminary diagnosis of malaria, leishmaniasis, trypanosomiasis (emergency report to the district epidemiological department - F.058);
- draw up a plan for laboratory and additional examination of the patient;
- interpret the results of laboratory tests, including specific diagnostic methods;
- draw up an individual treatment plan, taking into account epidemiological data, clinical form of the disease, severity of the course, presence of complications, allergic history, concomitant pathology, and prescribe prescriptions;
- provide assistance in case of emergency.
- provide recommendations on regimen, diet, examination, and supervision during the recovery period.

Hardware and software:

- A personal computer, laptop, tablet, smartphone or similar device,

- Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar operating system.
- Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
- Access to the global network, local network,
- Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Malaria, leishmaniasis, trypanosomiasis, anthroponosis, zoonosis, declared groups, protozoan diseases, relapse, peptic ulcer, haemoglobinuria, **malarial coma.**

LEISHMANIASIS is a group of protozoal infections characterised by skin lesions (cutaneous leishmaniasis) or predominantly internal organ involvement with fever, splenomegaly and anaemia (visceral leishmaniasis).

Etiology and epidemiology. Leishmania are intracellular parasites that develop in macrophages by simple halving. The causative agent is Leishmania donovani, which has four subspecies - L.d. donovani (Indian variant), L.d. infantum (Mediterranean-Central Asian variant), L.d. archibaldi (East African variant), L.d. chagasi (South American variant). Mediterranean-Central Asian visceral leishmaniasis and East African visceral leishmaniasis are zoonoses. The main source of infection is wild (jackals) and domestic (dogs) animals. The infection is transmitted through mosquito bites. Indian visceral leishmaniasis is an anthroponosis. The main source of infection is humans. The mode of transmission is vector-borne, through mosquito bites. In visceral leishmaniasis, the pathogen enters the body after a mosquito bite and multiplies in macrophage cells of the spleen, liver, bone marrow, lymph nodes, intestinal wall, kidneys, lungs, and is characterised by hyperplasia of the lymphohistiocytic system.

Clinic. It is characterised by three periods: initial, anaemic-splenomegalic, and cachectic (terminal). The incubation period is from 15-20 days to 10-12 months. After the onset of the primary affect (a pale pink slightly pigmented nodule appears at the bite site), the patient develops fever, lethargy, adynamia, pale skin, decreased appetite, and a slight enlargement of the spleen after the incubation period.

In the period of full development of the disease - remitting fever, or temperature in the form of a Rodener curve, severe splenomegaly. The spleen is enlarged, sometimes reaching the pubic junction, anaemia, and enlarged peripheral lymph nodes. In the blood, there is a sharp leukopenia up to $2*10^9$ /l, lymphocytosis, granulocytopenia, anaemia, thrombocytopenia. The acute period is followed by the cachectic period - severe splenomegaly, general weakness, decreased muscle tone, thinning of the skin on the abdomen, so it seems that the spleen is sagging downwards and the abdomen is protruding. Patients have leishmaniasis in the form of nodules the size of a urea nucleus and skin areas with reduced pigmentation. Patients develop complications in the form of pneumonia, necrotising stomatitis, gingivitis, noma, and abscesses.

Diagnosis - bone marrow puncture, liver and spleen biopsy. Serological methods - PCR, ELISA, PCR.

Treatment: pentostam, glucantim (antimony), meglumine antimonate, sodium stibogluconate, amphotericin B, spleen removal. In case of cutaneous forms of leishmaniasis, in addition to the above-mentioned drugs, treatment of the initial stages of ulcers is carried out with laser therapy and cryotherapy.

Cutaneous leishmaniasis is divided into Ancient World Leishmaniasis and American Cutaneous Leishmaniasis. Ancient World cutaneous leishmaniasis is widespread: Mediterranean, Near and Middle East, West and Central Asia, Africa.

Etiology and epidemiology. The causative agent of anthroponotic cutaneous leishmaniasis is Leishmania tropica (minor), and zoonotic leishmaniasis is Leishmania tropica (major). Rural cutaneous leishmaniasis is a zoonotic disease. The source of infection is steppe rodents, wolves, jackals. Transmission occurs through mosquito bites. A person becomes infected when he or she enters the centre of a suspected disease. After returning to the city, a person becomes the main source of infection. Urban leishmaniasis (anthroponosis). In cutaneous leishmaniasis, the pathogen spreads and causes local skin lesions in the form of the following stages: proliferation, destruction (ulceration), and repair (scarring). Leishmania can enter regional lymph nodes from primary lesions.

Clinic. The incubation period is 2-9 months (sometimes 3-5 years). The disease begins with the appearance of papules that slightly rise above the skin of red-brown colour. After 3-6 months, an ulcer in the form of a crater-like pit forms at the site of the papule, its bottom is fine-grained, covered with purulent layers. Around the ulcer there is a moist infiltrate, the edges of the ulcer are unevenly eaten away, the discharge is slight, seropurulent, and the ulcer dries up with the formation of a brown crust. The duration of the primary lesion - from the appearance of the papule to ulcer scarring - is approximately one year.

Zoonotic leishmaniasis is characterised by a shorter incubation period - up to 3-4 weeks. The progression of the disease is faster. Several ulcers are often formed. Ulcers can reach 10-15 cm and leave large scars. In Africa, zoonotic cutaneous leishmaniasis can take on a lepromatous form.

The diagnosis of cutaneous leishmaniasis is confirmed by scraping the contents of the ulcer with Romanowski-Gimza staining. It is important to know how to scrape correctly. It is necessary to take the ulcer area with tweezers so that it is anaemic. after the incision with a scalpel, a thin layer of tissue is removed.

American skin leishmaniasis, a large number of pathogens and their names depend on the country in which they are registered. For example, L. mexicana, L. braziliens, L. peruviana, etc. The pathogenesis and clinic are almost identical to cutaneous leishmaniasis of the Old World, with the exception of chakler's ulcer, which occurs with cartilage damage to the ears, cutaneous and mucosal leishmaniasis (espundia), with damage to the cartilage of the nasopharynx, larynx, and trachea.

The diagnosis is made on the basis of clinical manifestations and a positive Montenegro test (intradermal leishmanin test).

TRYPANOSOMIASIS are tropical vector-borne infections, the causative agents of which belong to the Sarcomastigophora type, Mastigophora subtype, Epomastigophora class, Kinetoplastida order, Trypanosomatina suborder, Trypanosoma genus.

The developmental cycle takes place with a change of hosts. Part of the cycle takes place in vertebrate hosts (humans, animals), and the other part in an invertebrate host, insects. The diseases are common in tropical Africa (African trypanosomiasis, or sleeping sickness) and South America (American trypanosomiasis, or Chagas disease).

The causative agents of African trypanosomiasis go through two stages of development: the trypomastigote and epimastigote stages. Two more stages have been described for the American trypanosomiasis pathogen: the promastigote and amastigote stages.

African trypanosomiasis

Trypanosomes go through two stages of development: trypomastigotes (in the human body) and epimastigotes (in the tsetse fly).

It is known in two varieties:

- Gambian (West African)
- Rhodesian (East African)

Trypanosomes multiply at the site of tsetse fly suction, in the skin. They penetrate the lymphatic vessels to the lymph nodes. After 2-3 weeks, they are hematogenously distributed to parenchymal organs, where they cause inflammatory and autoimmune processes. Trypanosomes are able to modify their antigenic structure during the infectious process and thus escape the influence of immune surveillance factors. The infectious process is long due to the wave-like nature of parasitemia, which stops as a result of the synthesis of a sufficient level of specific antibodies and resumes when a new antigenic variant of the pathogen is formed.

The small vessels of the heart and the brain are affected, leading to inflammatory and degenerative changes (myocarditis, meningitis, demyelinating panencephalitis).

Clinic

Stages of the disease:

- early (haemolymphatic)
- late (meningoencephalitis).

The primary lesion is a 2 cm papule (trypanosomiasis chancre) that disappears in a few days, leaving a scar. Soon after, there is a wave-like fever of 38-39 °C, which lasts for weeks with short periods of apyrexia. A rash in the form of trypanids is characteristic - erythema in the form of plaques and rings 5-15 cm in size (more often on the skin of the back, chest, face) and lymphadenitis (of the peripheral lymph nodes, less often mesenteric, peribronchial). Hepatosplenomegaly.

If left untreated, it progresses to the late stage in 3-10 months. Gradually, a picture of progressive meningoencephalitis develops (fatigue, apathy, lethargy, daytime sleepiness and restlessness at night, followed by the development of fibrillary muscle twitching, convulsions, paresis and paralysis with the development of lethargy and cachexia in the terminal stage). The immediate cause of death is bacterial infections, in the acute period - meningoencephalitis and myocarditis.

In Europeans, unlike the local population, the disease is much more severe.

American trypanosomiasis

Trypanosomes go through four stages of development: promastigotes, amastigotes, tripomastigotes and epimastigotes.

Trypanosomes enter the regional lymph nodes through the skin or mucous membranes (most often conjunctiva) and multiply reactive. Then, progressive damage to the autonomic nervous system (ANS) is observed, resulting in impaired innervation of internal organs, primarily the heart, stomach, small and large intestine.

Clinic:

Stages of the disease:

- sharp
- chronic inapparatus
- chronic manifesto.

At the site of penetration of the pathogen, a primary effect (chagoma) occurs - a dark red, limited skin induration with edema, lymphangitis and regional lymphadenitis, which can last up to 2 months with gradual regression. In case of contact with the conjunctiva, acute inflammation with severe edema develops, which can

spread to the temporal region and face (Romani's symptom) with the development of inflammation of the regional parotid lymph nodes.

After 1-2 months, the infectious process is generalised, which is manifested by a rise in temperature to 39-40 °C, which is constant or remitting with possible chills and sweating. The temperature can be maintained for up to 2 months. During this period, the liver and spleen become enlarged, and a small spotted rash may appear on the skin, which disappears on its own in 1-3 weeks. Symptoms of heart damage are constant (weakening of the tones, systolic murmur, expansion of the heart's borders, weakening of the pulse and a decrease in systolic pressure, in severe cases arrhythmia, heart failure, which is often the cause of death). A significant proportion of patients develop meningoencephalitis with possible paresis and paralysis.

With a favourable course, clinical symptoms gradually regress.

The chronic inaparanthous stage develops after the acute stage. Non-sterile immunity is formed. There are no clinical symptoms.

The chronic manifest stage is characterised by the progression of internal organ damage due to autoimmune processes. Clinically, it is most often manifested by cardiovascular lesions or meningoencephalitis (severe myocarditis and rhythm disturbances, cardiac dilatation). The most common causes of death are heart failure and thromboembolism. A characteristic feature of meningoencephalitis is damage to the autonomic ganglia, which is manifested by atonic dilation of internal organs (esophagus, stomach, colon, bladder) due to innervation disorders. Paresis and paralysis may develop. Secondary bacterial complications (pneumonia, pyelonephritis, etc.) may occur.

Differential diagnosis of trypanosomiasis

	African trypanosomiasis		**American trypanosomiasis**
	Gambian	**Rhodesian**	
Pathogen	Trypanosoma brucei gambiens	Trypanosoma brucei rhodesiense	Trypanosoma cruzi Chagas
Region of distribution	West and Central Africa	Central and Eastern Africa	All countries of South America

			(most of all in Brazil, Argentina, Venezuela)
Reservoir	Human (possibly pigs)	Antelopes, to a lesser extent cattle	In anthropurgic foci, there are sick people and carriers in natural foci - about 100 species of vertebrates
Vector	The tsetse fly	The tsetse fly	Fleas of the genus Triatoma
Mechanisms and routes of transmission	Transmission	Transmission	Contact (rubbing in the wound from sucking flea faeces), haemotransfusion, vertical - in childbirth, transplacental route is being studied
Incubation period	2-3 weeks	1-2 weeks	1-2 weeks
Primary affect	Trypanosomiasis chancre	Trypanosomiasis chancre	Chagoma
Fever	wavy	wavelike higher and longer-lasting fever	Permanent or remitting with possible chills, sweating
Rash	Trypanids	Trypanids	Small-spotted

Lymphadenitis	++	+	+
Hepatosplenomegaly	+	+	+
Progressive disaster damage	meningoencephalitis	The signs of meningoencephalitis appear faster and are more severe.	The autonomic nervous system is often deeply affected (serious damage to the myocardium, stomach, intestines). Often meningoencephalitis
The course.	More benign	Rarely lasts more than a year	
Cause of death	Meningoencephalitis, myocarditis, bacterial infections	Meningoencephalitis, myocarditis, bacterial infections	in the acute stage - meningoencephalitis; in the chronic stage - heart failure, thromboembolism.
Chemoprophylaxis	Pentamidine 3-4 mg/kg once every 6 months	Inefficient	Not applicable

Specific diagnosis of trypanosomiasis:

1. Microscopic method - a thick drop and a smear of blood, liquor, chancre puncture, lymph nodes, bone marrow.
2. For the detection of Trypanosoma gambiens, blood concentration techniques are used (microhematocrit centrifugation, leukocyte film technique, anion exchange chromatography technique in mini-columns followed by centrifugation).

3. Serological tests (PCR, ELISA).
4. Biological test on guinea pigs.
5. Skin allergy tests.

Differential diagnosis of trypanosomiasis:

- malaria
- visceral leishmaniasis
- meningoencephalitis
- tuberculosis
- brucellosis
- myocarditis.

Treatment of trypanosomiasis:

All patients need inpatient treatment, bed rest, and vitaminised nutrition.

Etiotropic treatment:

- African trypanosomiasis: at an early stage, IV suramin, eflornithine, IV pentamidine. The course of treatment is 3-4 weeks. In the late stage, nitrofuran drugs or benznidazole + nifurtimox are added to the above therapy. Severe cases and relapses: melarsoprol + prednisolone.
- American trypanosomiasis: therapy is less effective; benznidazole is administered orally at 5-10 (7.5) mg/kg/day in 2 doses for 60 days; nifurtimox is administered orally at 8-10 mg/kg/day in 3 doses for 60-90 days. Treatment of chronic forms gives worse results.

Pathogenetic and symptomatic therapy:

Detoxification therapy, antiplatelet agents, CS, if indicated.

A positive clinical effect in the treatment of African trypanosomiasis was obtained by combining antimicrobials and γ-IFN.

Dispensary observation for 2 years.

Prevention:

Early detection and treatment of patients, disinfestation, sanitary and educational work. Control in the selection of donors in endemic areas.

Chemoprophylaxis.

MALARIA is a group of endemic vector-borne diseases of protozoan etiology. It is characterised by recurrent fever, anaemia, and hepatosplenomegaly.

According to the WHO, malaria is currently the most common parasitic disease, with a large number of severe forms and a high mortality rate. The overall incidence of malaria is 2.6% of all diseases in the world. The death toll from malaria ranges from 1.5 million people annually, with 85% of this figure being malaria deaths in Africa. In terms of percentage, malaria deaths reach 4-5% of total global mortality.

Malarial coma can develop in the first days of tropical malaria. There are three stages: somnolence (precoma), soporas - deeper hibernation with weak gaps in consciousness and deep coma with complete loss of consciousness. During the precomatose period, patients are inactive, monosyllabic and reluctant to answer questions, quickly become exhausted and fall into a soporic state. Many patients have positive meningeal signs. They are caused not only by cerebral hypertension, but are also associated with damage to the tonic centres in the frontal area. In some patients, hyperkinesis phenomena are noted, ranging from clonic limb muscle cramps to general tonic cramps. Abdominal reflexes are reduced, and oculomotor disorders appear: divergent strabismus, nystagmus (horizontal and vertical), and convergence disorders. In coma, the pharyngeal reflex disappears, followed by the corneal and pupillary reflexes. There are floating movements of the eyeballs with the eyelids open (as if the patient is looking at the ceiling). Temperature is 39-40°C, blood pressure is below 70/30 mm Hg, heart sounds are deaf, heart borders are dilated, pulse is threadlike, tachycardia (130-150 per minute), breathing is shallow, rapid from 30 to 50 per minute. There is involuntary urination and defecation. Laboratory examination reveals: a high level of parasitemia - 100000 in 1 μl of blood; different age stages of the parasite in the peripheral blood; hematocrit below 20%, hemoglobin below 50 g / l; increased creatinine, blood urea; blood glucose less than 2.2 mmol / l; acidosis - arterial blood pH below 7.25, alkaline reserve less than 15 μmol / l. With each hour of the patient's stay in a coma, the possibility of saving him decreases. The success of therapy in patients with malarial coma can be achieved only in the first two, at most three days. Long-term consequences of cerebral malaria are observed in 5-10% of patients. The

most severe consequences include: hemiplegia, ataxia, extrapyramidal disorders, mono- and polyneuritis, postmalarial psychosis, characterised by mental weakness, hysteria, and depression.

In addition to malarial coma, tropical malaria can develop acute pulmonary oedema, which is more often the result of acute renal failure. However, there is a rare, especially malignant complication of tropical malaria - primary pulmonary edema, which sometimes develops even at normal temperature. Patients suddenly develop severe dyspnoea of 50-60 per minute, cyanosis, acrocyanosis. This complication is almost not amenable to conventional intensive care.

Haemoglobinuric fever can be of parasitic or drug origin (sometimes called quinine-malarial haemoglobinuria), so it requires different treatment. Hemoglobinuric fever is characterised by massive red blood cell breakdown, when their number decreases to $1x\ 10^{12}$ / l in a few hours, hemoglobin decreases sharply to 13 g / l, reticulocytosis appears (up to 33% of normoblasts) with the development of severe haemolytic jaundice and haemoglobinuria. Hemoglobinuric fever often occurs several hours after taking quinine, primaquine or other drugs that have an oxidative effect and promote haemolysis. It occurs mainly in people with a deficiency of the red blood cell protective enzyme glucose-6-phosphate dehydrogenase.

Hemoglobinuric fever begins suddenly with chills. The temperature reaches 40 °C, intense pain in the kidneys, headache, severe weakness, nausea, and vomiting appear. Haemolysis is accompanied by the development of jaundice, the appearance of urine of the colour of "black coffee" or "red wine" with a characteristic sediment in the form of cylinders, red blood cells, and protein. The main symptom is the appearance of "black coffee" coloured urine, which is due to the content of oxyhaemoglobin in the urine (meta-haemoglobin in the settled urine). When standing, the urine is divided into 2 layers: the upper one is transparent dark red, the lower one is cloudy dark brown, containing detritus (lumps of haemoglobin and red blood cells). Oliguria and anuria follow. There are few parasites in the peripheral blood in drug-induced haemoglobinuric fever.

In some countries (Indonesia, Brazil), chloroquine-resistant pathogens of three-day malaria have been identified. In such cases, amodiaquine 30 mg/kg per day for 3 days is used.

Vivax and ovale malaria are characterised by late relapses due to the preservation of bradysporozoites in hepatocytes. This requires the administration of histoschizotropic drugs that act on the tissue forms of the parasite - primaquine 15 mg (3 tablets) once daily for 14 days.

For the treatment of quinine-resistant tropical malaria (parasitemia++ in 1 μl of blood), one of the combinations is used orally:

- artesunate + amodiaquine, 2 tablets per day for 3 days orally;
- artesunate + mefloquine, 1 tablet for 3 days orally;
- metacalfin, 1 tablet for 3 consecutive days orally;
- Fansidar 3 tablets orally at the same time.

In severe tropical malaria (parasitemia +++ in 1 μl of blood), it is used:

- artesunate - water-soluble artemisin derivatives at a dose of 2 mg/kg once a day + clindamycin 10 mg/kg twice a day for 7 days;

-Artemether 3.2 mg/kg intramuscularly (oil solution) - on the first day, from day 2 to day 5 1.6 mg/kg intramuscularly.

- quinine 10 mg/kg every 12 hours (but not more than 2 grams per day) intravenously drip + doxycycline 5 mg/kg once a day for 7 days.

In case of malarial coma, quinine is the drug of choice, as it is the best able to penetrate the blood-brain barrier. Quinine is a natural alkaloid of the quinine tree bark. Therapy is started with intravenous quinine administration at a single dose of 10 mg/kg body weight every 12 hours. Dilute 1 ampoule (1 ml of 50% quinine solution) in 500 ml of isotonic sodium chloride solution or 5% glucose solution (with low blood pressure in polyglucose) and administer at a rate of no more than 20 drops per minute. A dose of 0.02 mg/kg/min is maintained for 72 hours using an infusion pump. As soon as the patient recovers from a serious condition, the drug is switched to oral administration. When quinine alone is prescribed, the relapse rate remains high, so one of the following drugs is prescribed simultaneously with quinine: artemether,

clindamycin, doxycycline, and others. For example, the combined drug fancidar is administered intramuscularly 2.5 ml once daily for the first 3 days. This drug should be injected deep into the muscle; it should not be added to infusion solutions.

Prevention. Preventive measures are aimed at:

- timely detection and treatment of malaria patients and parasite carriers;
- vector control and protection against mosquito bites;
- chemoprophylaxis.

They are subject to a malaria test (a thick drop and a blood smear):

- all patients with fever with an undiagnosed diagnosis within 5 days, with enlarged liver and spleen, anaemia, especially if they have travelled to malaria-endemic countries in the last 3 years;
- blood recipients with fever within the next three months after a blood transfusion.

People who have returned from malaria-endemic countries cannot be donors for 3 years.

The most important condition for combating malaria is the implementation of hydraulic engineering measures, treatment of water bodies (in places where mosquitoes breed), and treatment of residential and livestock premises with insecticides. An effective insecticide is bactoculicide. It is advisable to stock ponds with gambusia, a fish that intensively eats mosquito larvae. Use mesh on windows and ventilation openings. Use repellents for personal protection.

Prophylaxis with chemotherapy does not prevent infection, but only alleviates the symptoms of the disease. People travelling to malaria-endemic areas should take chemoprophylaxis a week before travelling to the area, during the entire stay in the endemic area and for 4 weeks after returning. In foci of three-day (vivax, ovale) and four-day malaria, delagyl 0.5 g once a week and amodiaquine 0.4 g once a week can be used. With prolonged use, when the total dose of delagyl is more than 100 g, there is a risk of developing resistance. It is necessary to switch to other medications. After the end of the stay in the focal point, persons at risk of three-day malaria are chemoprophylaxis with primaquine (0.5 g - 14 days).

In areas of chloroquine-resistant tropical malaria, mefloquine 0.5 (or 5 mg/kg) once a week is recommended, provided that there are no contraindications (cardiac conduction disorders) and the parasite strains are sensitive to this drug (see Fig. 5), fancidar 1 tablet per week, fancimer 1 tablet per week can be used.

Presentations:

https://docs.google.com/presentation/d/1H-PnTzUlC92gYBUIukuZBdUEDl6Rbii8/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for self-monitoring

1. The source of infection in malaria and the ways of transmission of malaria, leishmaniasis, trypanosomiasis;
2. Pathogenicity factors of malaria, leishmaniasis, trypanosomiasis;
3. Pathogenesis of malaria, leishmaniasis, trypanosomiasis;
4. Stages of the cyclic clinical course of malaria;
5. The main symptoms of malaria;.
6. Specific complications of malaria;
7. 7. Consequences of malaria;
8. The main causes of mortality in malaria;
9. The concept of early and late relapses in malaria;
10. Haemogram of a patient with malaria in the midst of the disease;
11. Plan of examination of a patient with suspected malaria;
12. Methods of specific diagnosis of malaria;
13. Etiotropic therapy of malaria. Doses, route of administration, duration of administration;
14. Rules for discharging a patient with malaria from hospital;
15. Drugs for the treatment of malaria;
16. Emergency care for malarial coma;
17. Clinic of haemoglobinuric fever, possible causes of its occurrence.

18. Etiology, pathogenicity factors of the pathogen of leishmaniasis (cutaneous, visceral, New World), trypanosomiasis, trypanosomiasis;

19. Epidemiology of cutaneous and visceral leishmaniasis, trypanosomiasis; pathogenesis;

20. Clinical and epidemiological features of cutaneous, visceral and New World leishmaniasis, trypanosomiasis;

21. Pathogenesis, timing and clinical manifestations of complications of visceral leishmaniasis and trypanosomiasis;

22. Laboratory diagnosis of leishmaniasis and trypanosomiasis;

23. Principles of treatment;

24. Tactics of treatment in case of emergency;

25. Prognosis of visceral and cutaneous leishmaniasis, trypanosomiasis;

26. Prevention of cutaneous and visceral leishmaniasis, trypanosomiasis.

Glossary

Malaria is an infectious disease caused by protozoa (malaria plasmodium), which are transmitted from a sick person through the bites of malaria mosquitoes. The disease is very common in countries with tropical climates and is characterised by a severe course, general malaise, chills, hyperthermia, disorders of the nervous and other body systems.

Leishmaniasis is a group of protozoan vector-borne diseases of humans and animals characterised by damage to internal organs (visceral leishmaniasis) or skin and mucous membranes (cutaneous leishmaniasis), which are transmitted by mosquitoes.

Trypanosomiases are a group of parasitic infectious diseases of animals and humans caused by protozoa of the genus Trypanosoma. The most common trypanosomiases among humans are: African (sleeping sickness, which combines Gambian and Rhodesian forms); American (Chagas disease).

Anthroponosis is a group of infectious diseases caused by pathogens that naturally can only parasitise the human body.

Zoonosis is a group of infectious diseases caused by pathogens that can naturally parasitise only in the body of an animal.

Declared **groups** are categories of persons at high epidemic risk: workers in the food industry, catering and food trade, employees of educational institutions, workers in the public utilities and service sector, healthcare workers, students of secondary schools and boarding schools, university students, students of colleges, technical schools and secondary vocational schools, persons from socially disadvantaged families, etc.

Protozoan diseases are infectious diseases whose causative agents are unicellular protozoa. This group includes diseases such as malaria, trypanosomiasis, and leishmaniasis.

Recurrence - (from the Latin recidere) is the recurrence of a disease after a seemingly complete recovery (remission). Recurrence in infectious diseases is caused by the fact that the pathogen does not completely disappear from the body during treatment and, under certain conditions, causes the disease to return again in the form of a rapid recovery of clinical signs and symptoms.

Pendine ulcer (cutaneous leishmaniasis, Borowski's disease, rubber ulcer, Pendine ulcer, Baghdad ulcer, etc.) is a group of leishmaniasis manifested by lesions of the skin, subcutaneous tissues and/or mucous membranes. These diseases are caused by leishmaniasis. They are prevalent mainly in the tropics and subtropics and are transmitted through mosquito bites.

Hemoglobinuric fever is a complication that is most common in tropical malaria. In this case, fever and acute haemolysis can be of parasitic or drug origin (due to treatment with certain antimalarial drugs, in people with defects in the enzyme cycles of red blood cells). Haemoglobinuric fever is characterised by massive red blood cell breakdown with severe haemolytic jaundice and excretion of haemoglobin in the urine (haemoglobinuria).

Malarial coma is the most frequent and severe complication of tropical malaria, which causes the main mortality, proceeds rapidly and ends fatally without treatment, based on the development of the complication due to blockage of the vessels

of vital organs (including the brain) by red blood cells containing plasmodium, parasitic blood clots.

Test control of knowledge

>>Source of infection in malaria

( ) sick animal

( ) gametocarrier of malaria plasmodium

( ) mosquito of the genus Anopheles

(x) person

( ) mosquito of the genus Aedes

>>The contingent of patients with the easiest malaria course

( ) children

( ) persons arriving from a non-epidemic region

( ) immunocompromised adults

(x) adults living in the endemic area

( ) pregnant women

>>The manifestation of a typical malarial paroxysm is <<

( ) tracheobronchitis

(x) fever

( ) diarrhoea

( ) hepatomegaly

( ) splenite

>>Choose an atypical symptom of moderate malaria without complications in the acute phase: <<

( ) body temperature rises periodically

( ) enlarged liver

( ) anaemia

( ) enlargement of the spleen

(x) conjunctivitis

>>Changes in cerebrospinal fluid in malarial coma

(x) lymphocytic pleocytosis

( ) neutrophilic pleocytosis

( ) presence of fresh red blood cells

( ) protein reduction

( ) cell-protein dissociation

>> Choose an atypical malaria complication <<

(x) orchid

( ) anaemia

( ) hepatitis

( ) pneumonia

( ) haemoglobinuric fever

>>What malaria has late recurrence <<

( ) tropical

(x) three-day

( ) all types of malaria

( ) four-day and tropical

( ) three-day and tropical

>>What malaria has early and late relapses <<

( ) tropical

( ) tropical and three-day

(x) three-day

( ) all types of malaria

( ) Tropical and four-day

>>The drug of choice for the treatment of malarial coma is: <<

( ) delahil internally

( ) quinine orally

(x) quinine intravenously

( ) metronidazole orally

( ) thienam intravenously

>>Possible changes in laboratory parameters in malaria <<

( ) hyperglycaemia

( ) eosinophilia

(x) an increase in indirect bilirubin

( ) increase in direct bilirubin

( ) significant increase in cytolytic enzymes

>> What is the causative agent of visceral leishmaniasis of Indian Kala-Azar?

(x) L.donovani donovani,

( ) L. donovani infantus,

( ) L.d.donovani archibaldi,

( ) L.tropica minor,

( ) L. tropica major,

>>Name the causative agent of cutaneous leishmaniasis of the urban type: <<

( ) L. tropica major,

(x) L. tropica major,

( ) L.tropica minor,

( ) L. donovani infantus,

( ) L.d.donovani archibaldi

>>Name the vector of cutaneous leishmaniasis: <<

( ) mosquitoes,

( ) jackals,

( ) Bedbugs

( ) damselflies,

(x) mosquitoes

>>Name the source of visceral leishmaniasis infection: <<

( ) rodents,

(x) a person,

( ) jackals,

( ) dogs,

( ) mosquitoes

>>What causes East African Leishmaniasis?

(x) L.donovani chagasi,

( ) L.donovani donovani,

( ) L.tropica minor,

( ) L. tropica major

>>What visceral leishmaniasis is characterised by cutaneous leishmanoids?

( ) East African,

( ) Mediterranean,

(x) Kala Azar, India,

( ) cutaneous leishmaniasis,

( ) at all

>>In what form of leishmaniasis are characteristic changes in the haemogram detected?

( ) Indian Kala Azar,

(x) for all types of visceral leishmaniasis,

( ) East African,

( ) Mediterranean visceral,

( ) New World leishmaniasis

>>In what leishmaniasis is the examination of a thick blood drop, a puncture of bone marrow, lymph nodes, radiography, X-ray, radiological examination used?

( ) cutaneous leishmaniasis of the Old World,

( ) peptic ulcer,

( ) New World cutaneous leishmaniasis,

(x) visceral leishmaniasis,

( ) at all

>>In what leishmaniasis is material from ulcer and marginal infiltrate used?

( ) peptic ulcer,

( ) visceral leishmaniasis,

( ) Leishmaniaceae,

( ) Indian Kala Azar,

(x) for all skin

>>In what form of leishmaniasis are 5-valent antimony preparations used in the treatment?

(x) for all varieties except New World,

( ) only for visceral,

( ) only in case of cutaneous,

( ) only with anthroponosis,

( ) only in case of zoonotic

Examples of case studies

Task 1. A 32-year-old patient has been experiencing chills, hyperthermia up to 40°C, and profuse sweating every other day for a week.

Physical examination: pale skin, hepatosplenomegaly. Pulse rate - 88 per minute. BP - 105/ 75 mm Hg. Heart sounds are slightly muffled.

SAMPLE EXECUTION

1. Preliminary diagnosis: Three-day malaria, moderate severity.

2. Survey plan:

- general blood test

- general urine analysis

- general faecal analysis

- biochemical blood test: bilirubin, ALT, AST, total protein, urea, creatinine, glucose, BUN, RW, HBsAg, ECG, ultrasound of the abdomen, blood test for malaria: thick drop + smear

3. Treatment:

- bed rest

- diet table number 5

- drink plenty of water (up to 3000 ml per day)

- delagil1.0 1st dose, + 0.5 g after 6 hours, per os (first day of treatment, then delagil0, once, for 3 days)

- Primahin0.027, 3 tablets twice daily, per os, for 7 days

- ascorutin (1 tonne x3 times a day, per os)

- diazolin 0.1 x3 times a day, per os

Task 2. A 34-year-old patient, diagnosed with vivax malaria, developed dark (black) urine after taking delagyl.

Observations: T- 37.5°C. The skin is subcharacteristic. The tongue is dry, covered with a white coating. The spleen is palpable 2.5 cm below the left costal arch, the liver - by +1.5 cm. Pulse 80 per minute, rhythmic. Blood pressure -90/60 mm Hg. Heart sounds are muffled. Vesicular breathing in the lungs. No meningeal signs were found.

SAMPLE EXECUTION

1. Preliminary diagnosis: Three-day vivax malaria, moderate severity. Hemoglobinuric fever.

2. Survey plan:

- general blood test
- general urine analysis
- general faecal analysis
- biochemical blood test: bilirubin, ALT, AST, total protein, urea, creatinine, glucose, BUN, RW, HBsAg, ECG, ultrasound of the abdomen, blood test for malaria: thick drop + smear

3. Treatment:

- bed rest
- diet table number 5
- drink plenty of water (up to 3000 ml per day)
- delahil with primahin to cancel
- quinine dehydrochloride 0.65 three times a day, per os, for 3 days
- 5% glucose solution400.0 IV drip
- trisil solution 400.0 in\in drip
- ascorutin (1 tonne x3 times a day, per os)
- diazolin0.1 x3 times a day, per os

Topic 3: Other protozoal diseases: babesiosis, toxoplasmosis, pneumocystis, cryptosporidiosis, isosporosis, acanthamebiasis. Leprosy. Monkeypox.

Learning objectives: to learn and master the etiology, main clinical manifestations, basics of specific and non-specific diagnostics, treatment of protozoal diseases, leprosy, monkeypox. Understand the importance of timely preventive measures against the possible development of these infections.

List of skills:

- Master the general and specific prevention of protozoal infections
- Follow the basic rules of working at the patient's bedside
- Take a medical history with an assessment of epidemiological data
- Know the forms and methods of health education work
- Search for up-to-date literature in the field
- Summarise scientific and practical literature
- Participate in the dissemination of medical knowledge among the population
- Learn about the main anti-epidemic measures
- Know anti-epidemic measures in the event of an infection centre
- Know the safety precautions for invasive manipulations
- Know what to do in the event of exposure to contagious material in the workplace

Hardware and software:

- A personal computer, laptop, tablet, smartphone or similar device,
- Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
- Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
- Access to the global network, local network,
- Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Malaria, leishmaniasis, trypanosomiasis, relapse, complications

CRYPTOSPORIDIOSIS. Zooanthroponous protozoan infection with a faecal-oral mechanism of transmission. It is characterised by a predominantly digestive tract involvement with dehydration, diarrhoea, and usually a benign course. It belongs to opportunistic infections and is severe in people with immunodeficiency conditions, primarily in patients with AIDS. The causative agent is Cryptosporidium from the family Cryptosporididae, class Sporozoasida, an obligate parasite that infects the microvilli of the mucous membrane of the digestive tract and respiratory tract of humans and animals. S. parvum is pathogenic for humans.

Sources of infection include farm animals, contaminated water and food, sick people, convalescents, and carriers of cryptosporidium. The main route of transmission is waterborne: oocysts can be infected for up to 18 months, due to their small size they can freely penetrate most filters, die only at temperatures above 72°C, and are resistant to standard disinfectants. The infection is recorded almost everywhere, and is more common in children under 2 years of age. Seasonality is the warm season. The susceptibility to cryptosporidiosis is low, with children under 5 years of age, livestock workers, veterinarians, patients with immunodeficiency states of various etiologies (congenital or acquired), and tourists being at risk.

Pathogenesis. The pathogen is most commonly found in the mucous membrane of the distal small intestine, lungs, biliary tract, oesophagus, duodenum. As a result of cryptococcal activity, intestinal microvilli are damaged, lactose deficiency develops, which leads to a decrease in absorption with the development of secondary malabsorption syndrome and osmotic diarrhoea, ionic balance is disturbed, and fermentative dyspepsia occurs.

Clinical manifestations. Clinical manifestations are based on acute diarrhoeal syndrome that develops 2-14 days after infection and is characterised by acute enteritis or gastroenteritis. The severity of symptoms depends on the state of the patient's immune system. In patients without immunodeficiency, there are abundant, watery (choler-like) stools with a very unpleasant odour with a frequency of up to 20 times a day. Diarrhea lasts for 7-10 days (2-26 days). The patient may lose from 1 to 15 litres of fluid per day. Profuse diarrhea can be accompanied by moderate spastic abdominal

pain, nausea and vomiting (in 50%), a slight increase in body temperature, lack of appetite, headache, flatulence. In children, the diagnosis is complicated by the presence of catarrhal phenomena.

In people with immunodeficiency conditions (on the background of long-term treatment with immunosuppressants, hormone therapy, chemotherapy, patients with AIDS), the course of the disease is prolonged or chronic, and may recur, leading to significant weight loss and exhaustion. The clinical picture of colitis or enteritis is accompanied by spastic pain in the upper abdomen, fever, intoxication, and dehydration.

Respiratory cryptosporidiosis is a variant of the extraintestinal form, characterised by a severe course, more often fatal. Clinically, it manifests as interstitial pneumonia with dyspnea, prolonged cough, lymphadenopathy, and respiratory failure.

Diagnosis. The material for the study is faeces, sputum, duodenal contents. Immunological methods include immunoblotting, RIF, ELISA. PCR can also be used.

Treatment. Rovamycin, paramomycin, azithromycin (0.5 g once daily) are used as etiotropic drugs. In patients with AIDS, azidothymidine and nitrazoxamide are indicated. Pathogenetic and symptomatic therapy (rehydration, antidiarrhoea drugs, enzymes) is also necessary.

No specific prevention has been developed.

BABESIOSIS. A vector-borne disease of humans and animals caused by protozoa of the Babesidae family, it is considered a rare disease. More than 100 species of the pathogen are known, the most commonly infecting species are B.divergens, B.microti, B.rodhaini, while other species are specific to animals (cattle, dogs, sheep, deer) and are found on all continents except Antarctica. Babesia are small protozoa that parasitise red blood cells. Unlike malarial plasmodium, babesiae do not form pigment, they do not have erythrocytic schizogony and do not form gametocytes; they are located inside the affected erythrocytes in the centre or periphery of the cells. When stained by Gram stain, they look like thin rings with a diameter of 2-3 μm or pear-shaped formations with a diameter of 4-5 μm. Infection occurs through animal bites by

ticks of the genera Dermacentor, Rhipicephalus, Hyalomma. Small rodents are an intermediate reservoir. Human infection occurs through the bite of an infected tick or from a diseased animal, as well as during vertically delivered babies, and through transfusion of infected blood. Tourists, livestock workers, and shepherds get sick during the period of tick activity (spring-summer and summer-autumn seasons). The disease usually develops against the background of immunodeficiency (diabetes mellitus, post-splenectomy condition, AIDS patients, cancer patients, long-term use of corticosteroids). In people with a normally functioning immune system, the disease is asymptomatic, despite the presence of parasitemia, which reaches 1-2%.

Pathogenesis. After a tick bite, the pathogen enters the blood capillaries, then erythrocytes, where babesia multiply. The release of merozoites from red blood cells is accompanied by lysis of blood cells. As a result of babesia waste products and heterogeneous proteins entering the bloodstream, general toxicity and a pyrogenic reaction develop. Clinical manifestations appear when 3-5% of red blood cells are affected. Massive lysis of erythrocytes causes the development of haemolytic anaemia, microcirculatory disorders, haematuria, and acute renal failure.

Clinic. The incubation period lasts on average 5-15 days. The disease begins acutely, with a rapid rise in body temperature above 39 °C with severe weakness, severe headache, nausea and vomiting, pallor of the skin, and a feeling of discomfort in the epigastrium. A characteristic feature of the disease is haematuria, hepatosplenomegaly, and anaemia. The temperature curve is constant or irregular, the duration of fever is 8-10 days. On the 3-4th day of the disease, jaundice and hepatomegaly appear. From 6-7 days, signs of acute renal failure are added. In HIV-infected and other immunocompromised patients, fever can last for 3 or more weeks, with the development of dyspnoea, severe anaemia, profuse sweating, and a high level of parasitemia (20-80%). In immunocompetent patients, the course is benign and may resolve spontaneously.

Complications. Acute renal failure, acute hepatic failure, nonspecific pneumonia, and multiple organ failure may develop.

Diagnosis. Careful collection of epidemiological history, examination of smear and thick drops of the patient's blood for confirmation, indirect immunofluorescence reaction (diagnostic titer 1:256), RBC, PCR can also be used. Patients with prolonged fever in combination with anaemia and hepatosplenomegaly on the background of ineffective antibiotic therapy should be examined for babesiosis. Differential diagnosis is carried out with tropical malaria, sepsis, blood diseases, HIV infection, haemorrhagic fever with renal syndrome.

Treatment. A positive result can be achieved with comprehensive treatment. Combined regimens are used: for mild and moderate forms, it is recommended to use a combination of azithromycin (500-1000 mg) with atovaquone (750 mg twice daily); in severe cases, clindamycin (1.2-2.4 g/day, per os or intravenously) in combination with quinine (650 mg every 6-8 hours) is prescribed. The duration of etiotropic therapy for immunocompetent patients is 7-10 days; in patients with immunosuppression, antiparasitic therapy is extended for 14 days from the date of negative parasitoscopy. In case of severe anaemia, blood products (red blood cell transfusion) should be used; in case of acute renal failure, haemodialysis is indicated. Vitamins and short-term glucocorticosteroids are used as part of complex therapy. The absence of etiological therapy can lead to death (in 50-80%). Treatment of parasite carriers is prescribed in case of prolonged parasitemia (more than 3 months). No specific prophylaxis has been developed.

IZOSPOROSIS. A rarely diagnosed anthropogenic protozoan infection characterised by fever, digestive tract lesions, abdominal pain and weight loss. The causative agent is the protozoa of the genus Isospora, which is widespread in nature. In total, the genus includes more than 200 species, but only two species cause pathology in humans - Isospora natalensis and Isospora belli (I.hominis). The source of infection is humans. The mechanism of transmission is faecal-oral. Modes of transmission: water, food, household. The infection occurs by the alimentary route: protozoa enter the intestine of the patient with contaminated water or food. Transmission of the pathogen through direct contact with a sick person is unlikely,

because oocysts excreted in faeces become invasive only after 2-3 days. The patient can become a source of infection from the end of the incubation period until the end of the disease and for several weeks after the disappearance of clinical symptoms. The natural susceptibility of people is low. Children and people with immunocompromised conditions, including AIDS patients, are most at risk of the disease. The parasites multiply in the cells of the epithelium of the small intestine mucosa, undergo schizogony, sexual reproduction by fission, and the formed oocysts, together with faeces, enter the external environment, where the exogenous stage of their development - the process of sporogony - takes place.

With massive isospora infection, the intestinal mucosa is destroyed with the formation of leukocyte exudate and villi atrophy, which leads to parietal digestion disorders, capillary bleeding occurs, which facilitates the penetration of pathogens into host tissues. The absorption of nutrients and vitamin B12 is impaired, steatorrhoea develops, the level of albumin and other serum proteins decreases, and the absorption of D-xylose is impaired.

Clinic. Isosporosis is characterised by symptoms of enteritis. The incubation period lasts 5-7 (up to 10) days. The disease begins acutely with an increase in body temperature (sometimes up to 39°C), the period of fever can last up to a week. Patients suffer from headache, weakness, nausea, and cramping abdominal pain. Simultaneously with an increase in body temperature or on the 2nd - 3rd day afterwards, watery diarrhoea develops up to 10 times a day, sometimes with small amounts of mucus and blood. Some patients experience vomiting. The disease is usually acute, ending in recovery 7 to 20 days after its onset. In some cases, oocysts can be found in the faeces of people who have no obvious clinical manifestations. Occasionally, severe cholera-like forms are observed. Cases of prolonged chronic course of isosporiasis are observed in immunocompromised individuals or patients with AIDS, and the excretion of oocysts in the faeces can last for 2-12 months. In these cases, the parasites can go beyond the small intestinal mucosa and cause the development of disseminated isosporosis, which can lead to the death of the patient.

Diagnosis. The diagnosis can be made by examining faeces and detecting oocysts (preferably daily for 2-3 days); to increase the efficiency of diagnosis, it is recommended to flotate the test material in a saturated solution of sodium chloride using the Füleborn method, and the Darlington method is also used. Oocysts can be detected in faeces for 2 months, more often within 15-20 days after the disappearance of clinical symptoms of the disease. Duodenal probing and small intestinal biopsy can also be performed for diagnostic purposes; the ELISA method is auxiliary.

Treatment. Symptomatic and pathogenetic treatment aimed at rehydration and elimination of the main symptoms is prescribed. The drug of choice is co-trimoxazole. It is prescribed to adults: 2 tablets 4 times a day for 10 days, then 2 tablets 2 times a day for 3 weeks, metronidazole 500mg 2 times a day for 10 days is also possible. Fansidar, fazizin and other antimicrobial, antiprotozoal and antimalarial drugs are sometimes prescribed, but they are toxic and not very effective. For relapse prevention, patients with AIDS are prescribed co-trimoxazole (960mg every other day for three weeks) or pyrimethamine (25mg daily for 4 weeks).

LEPRA (Hansen's disease, leprosy, St Lazarus disease) is a chronic anthropogenic infectious disease characterised by lesions of the skin, peripheral nervous system, extremities, mucous membranes of the respiratory tract and anterior parts of the eyeball. The causative agent is Microbacterium leprae (discovered by Hansen in 1871), Gram(+), non-motile, resistant to low temperatures and desiccation, has no capsule, does not form spores. The disease is recorded in Africa, India, Nepal, Burma, Brazil, and sporadic cases have occurred in the USA, Russia, and Europe. In 2015, 211973 thousand new cases of leprosy were recorded worldwide. The mechanism of infection is airborne, sometimes contact. The source of infection is a sick person, and close prolonged contact is required for infection (in case of contact with an untreated leprosy patient, the risk of infection is about 10%). Children in families are more likely to become infected before the age of 10. Wild armadillos and lower primates can be a reservoir of infection in nature, but no cases of human infection have been described. Men get sick 2-3 times more often than women.

Pathogenesis. Leprosy is a low-contagious, low-pathogenic disease. Subclinical infection is common. Only one third of people are susceptible to leprosy. Relatives of patients are more likely to be affected, which confirms a genetic predisposition to the disease. The entry gate is the mucous membrane of the upper respiratory tract, and it is also possible for pathogens to penetrate the skin. Accidental infection can occur during tattooing, smallpox vaccination, and surgical interventions. Given the possibility of more frequent trauma to the skin of the lower extremities, it is believed that infection mainly occurs through the skin of the lower extremities. In this case, the pathogenetic significance is attached to microcirculatory disorders, varicose veins and mycoses of the feet - especially epidermophytosis and candidiasis.

The resistance of the infected organism and the virulence of the pathogen determine the form of the disease. It is assumed that a minor infection stimulates cellular immunity, resulting in the development of a tuberculoid variant of the disease. Massive infection reduces the immune potential, resulting in the development of the lepromatous type. The hormonal background is also important: the first manifestation may occur shortly after puberty, during pregnancy or after childbirth. During these periods, new lesions may appear and old ones may become active. Immunity in leprosy is cellular in nature, it is minimal in the lepromatous form of leprosy and maximal in patients with tuberculoid type.

Clinic. According to the variants of the course, there are lepromatous type (LL), tuberculoid type (TT), borderline lepromatosis (BL), borderline tuberculoid (BT), borderline leprosy (BB), and undifferentiated leprosy is separately distinguished. The incubation period ranges from 6 months to several decades, with an average of 3-6 years, and is asymptomatic. In the prodromal period, moderate vegetative and vascular disorders are possible: swelling of the extremities, paresthesias, cyanosis, decreased sweating, and fever.

The lepromatous variant is the most contagious and has a severe course. The skin lesions look like spots, papules, nodules (lepromas), plaques of different colours, the surface is smooth or with fine scaling, without clear contours, with a seal in the centre, and continuous infiltrates may form. Localisation is more common on the face

(nose, auricles, eyebrows, cheeks), limb joints, lower legs, and buttocks. Alopecia of the outer third of the eyebrows is typical.

In addition to the skin, the mucous membranes of the upper respiratory tract, the anterior parts of the eyeball, and internal organs are affected. On the infiltrated surface of the affected tongue, dense papules with a flat surface covered with a whitish coating due to epithelial exfoliation ("silver tongue") may appear. When the nervous system is affected, severe pain along the nerve endings is gradually replaced by a decrease in all types of sensitivity (pain, temperature, tactile). Eye damage is possible - keratitis, episcleritis, iridocyclitis, iritis. As a result of inflammatory infiltration, opacity, ulceration and scarring of the cornea after lepromatous keratitis without treatment, partial or complete loss of vision may occur. If the epiglottis is involved, voice loss up to aphonia occurs. In the later stages of the disease, ulcerative changes in the lepromas lead to perforation and deformation (saddle nose, facies leonine, tongue shortening, palatopharyngeal ankylosis), against the background of atrophy of the facial muscles, the face becomes mask-like (St Anthony's mask), and atrophy of the circular muscles of the eye leads to incomplete eyelid closure (lagophthalmos).

The endocrine glands are also affected, especially the sexual sphere: premature menopause, decreased sexual function up to impotence, development of orchitis, epididymitis in men, adnexitis and oophoritis in women, resulting in frequent infertility. The general picture of leprosy, and especially the lepromatous type, is characterised by exacerbations - leprosy reactions. They are triggered by concomitant diseases, neuro-emotional factors, colds, surgery, treatment regimen violations, iodine abuse, and are more common in spring and autumn. Very often, leprosy reaction begins during pregnancy, childbirth and lactation. They are clinically manifested by activation of all leprosy manifestations. Relapses of exacerbation reactions are accompanied by damage to the lymphatic vessels, impaired rheological properties of blood and platelet aggregation, which leads to lymphostasis and the development of elephantiasis (hence one of the old synonyms for leprosy - elephanthyasis graecorum).

The *tuberculoid variant* has a more benign course, characterised by the formation of myatrophies, trophic ulcers, and mutations due to damage to the

peripheral nervous system. Tuberculoid leprosy is characterised by early changes in pain, temperature and tactile sensitivity not only in the areas of the rash but also around it, where the skin is not clinically affected. It usually begins with the appearance of a depigmented spot or small papule with clear contours and hypersensitivity within its boundaries, then the rash merges and forms elements in the form of discs, rings, and semi-rings. Along the periphery of the rash foci, there is a zone of doughy consistency that rises above the skin surface due to merging lichenoid, papular elements. Along with the rash elements, thickened nerve trunks are palpable, resulting in muscle atrophy. The muscles of the extremities are more often affected, contractures, neurotrophic ulcers, and shortening of the phalanges develop. After the rash regresses, depigmentation and skin atrophy remain, and hair follicles and sweat glands disappear.

The borderline type is characterised by the predominance of neurological symptoms. The disease is manifested by the appearance of a scanty, unstable and predominantly spotted rash of various sizes and shapes on the skin. The spots are often erythematous and squamous with a brownish-yellow or rusty tint, with clear contours, and may be depigmented or hyperpigmented. Within a few months, paralysis, paresis, muscle atrophy, contractures, trophic ulcers on the soles of the feet, atrophy of the sweat glands and hair follicles develop, and the face becomes mask-like due to amygdala.

Undifferentiated leprosy - in case of early detection of the initial skin manifestations of the disease (leprosy), when a certain type has not yet formed. The most typical location in adults is the buttocks, lumbar region, thigh, shoulder; in children, the rash is located disseminated on the trunk and limbs, except for the "immune" zones. At the same time, in the corresponding area of irradiation, sensitivity is lost up to anaesthesia, reflexes are reduced, various motor, vascular and trophic disorders, paralysis, contractures, amyatrophy, deep, including penetrating ulcers appear. The clinical picture of polyneuritis with neurological symptoms is typical of undifferentiated leprosy.

Diagnosis. It is based on the assessment of: epidemiological history, clinical findings (rash, eyebrow loss, amyotrophy, contractures, polyneuritis); results of

functional tests for sensory and sweating disorders (Minor test, histamine test, nicotinic acid test); bacterioscopic index (detection of bacteria in smears from the affected skin and nasal mucosa, from the point of the affected lymph nodes; Fite-Faraco, Ziehl-Nielsen stain); results of pathological examination of a skin sample from the edge of the lesion (detection of M. Leprae); study of immunological reactivity of the body (lepromine test: intradermal injection of 0.1 ml of lepromine). Lepromin test (Mitsuda reaction) in healthy people, in patients with tuberculoid and borderline type will be positive, in case of reduced or absent resistance - negative. Malignant lepromatous type is characterised by a negative lepromin test along with the detection of a large number of Hansen's bacilli in the mucous secretions of the cartilaginous part of the nasal septum.

Undifferentiated leprosy is characterised by unstable results of humoral and cellular immune reactions, including the lepromine test. Therefore, the lepromin test in this case is of prognostic significance: if its results are negative, the process can transform into a severe lepromatous form, and if the results are positive, it can transform into a tuberculoid type.

The bacterioscopic index is the number of bacteria in the smear (the index value is directly proportional to the degree of infectivity of the patient with leprosy):

*0 = none detected in 100 fields of view;

*1+ = 1-10 bacteria per 10 fields of view;

*2+ = 1-10 bacteria per field of view;

*4+ = 10-100 bacteria per field of view;

*5+ = 100-1000 bacteria per field of view;

*6+ = more than 1000 bacteria per field of view.

It is more difficult to confirm the diagnosis of lepromatous and undifferentiated types when leprosy mycobacteria are absent. In these cases, it is possible to use the complement binding reaction (CBR) and the precipitation reaction, a biological test on guinea pigs.

Differential diagnosis is carried out with mycoses, sarcoidosis, lichen planus, vitiligo, onchocerciasis, toxicoderma, cutaneous leishmaniasis, tertiary syphilis,

tuberculosis of the skin, systemic lupus erythematosus, erythema nodosum, frambesia, and Kaposi's sarcoma.

Treatment. Complex treatment is carried out with the participation of narrow specialists (ophthalmologist, orthopedist, surgeon, neurologist, physiotherapist). Patients are hospitalised in a leprosarium. According to WHO recommendations, sulfonated drugs such as dapsone, leprosan, sulfetron, avlosulfone, in combination with clofazimine and rifampicin, and ethionamide and ofloxacin may be prescribed as etiotropic therapy. A monthly combination therapy regimen is used for up to two years, until M. Leprae disappears from the biopsy. If necessary, corticosteroids, NSAIDs, multivitamins, immunomodulators, drugs to improve nerve conduction, physiotherapy and surgery are prescribed. The average duration of therapy is 3-3.5 years. Outpatient treatment is possible if the patient is free of mycobacteria for 6-12 months after the course of treatment. With timely diagnosis in modern conditions, leprosy is a curable disease.

Complications: blindness, leprosy mutations, contractures, paralysis, nephritis, hepatitis, pneumonia, infertility, facial disfigurement. There is no specific prevention. Patients are isolated and contact persons are given preventive treatment based on a lepromine test. Newborn babies of infected mothers are isolated and put on artificial feeding. Children are usually born healthy. In endemic areas, sanitary and educational work is carried out, as well as mass examinations to actively identify patients. Contact persons are monitored for at least 7 years. Prevention of morbidity among medical personnel is based on strict adherence to sanitary and hygienic rules.

MONKEYPOX (lat. variola vimus, English monkeypox) is a rare natural and focal zoonosis characterised by vesicular pustular rash, fever, intoxication, found in the humid tropical forests of Central and West Africa. The causative agent is a DNA-containing virus of the genus Orthopoxvirus (OVM), family Poxviridae, first isolated in 1958 from sick monkeys, grows and multiplies on chicken embryos, very similar to smallpox virus (SV). The source of infection can be monkeys, African squirrels, some wild animals, and a sick person during the rash period. The most susceptible to

monkeypox are children and people who have not been vaccinated against smallpox. Humans become infected through direct contact with an infected animal or a sick person, or by eating the internal organs of sick animals without heat treatment.

Pathogenesis. The pathogenesis of monkeypox is based on systemic damage to internal organs (mainly lungs), suppression of the immune system and interferon (IFN) system. The disease is characterised by changes in biochemical parameters that indicate impaired kidney, liver and lung function. The upper respiratory tract is the entrance gate to the infection. The pathogen is transmitted by airborne droplets and through household items, toys and other things infected with the contents of smallpox pustules. The primary site of virus propagation in humans is the cells of small bronchi and bronchioles, and then the lungs are involved in the pathological process. Subsequently, the virus penetrates the regional lymph nodes and multiplies in them. From the affected lungs and regional lymph nodes, the virus enters the bloodstream and is killed by phagocytic cells of the internal organs rich in reticuloendothelium. Reproduction of the virus in the affected cells leads to a second wave of viremia at the end of the incubation period or at the beginning of the disease, with the pathogen penetrating the skin epithelium, mucous membranes of the upper respiratory tract and internal organs from the blood. As a result of intensive viral multiplication, specific lesions develop in the lower layers of the epithelium of the skin and mucous membranes. The death of monkeypox patients is caused by viral infection and, to a much lesser extent, by bacterial infection.

Clinical course. The clinical course of monkeypox in humans is characterised by 4 periods: incubation, prodromal, rash and convalescence. The incubation period lasts 7-21 days. The onset is acute, sudden, with a rise in body temperature to 39-39.5°C, weakness, headache, nausea, arthralgia, pain in the muscles of the back and lower extremities. Important symptoms of monkeypox include pain in the lower back, frontal part of the head, occipital or generalised headache. All manifestations are accompanied by severe intoxication. This period lasts from 48 to 120 hours. Lymphadenitis develops almost immediately (in 85-90% of patients), which is the main difference between monkeypox and smallpox. The submandibular, cervical, hyoid, and

inguinal lymph nodes are affected. On the 3-4th day of the disease, the temperature drops to subfebrile and a rash appears on the skin of the face, then on the upper extremities and trunk, with a characteristic centrifugal location. The development of the rash element is staged: from a macule (6-10 mm) to a vesicle with navel-like involvement, then pustulation and crusting with a final scar. During vesicle suppuration, the body temperature rises again to 39-40°C, and infectious and toxic shock may develop. In severe forms, the number of rash elements reaches 100 or more. Enanthema may develop with the formation of painful erosions and ulcers. The condition improves from 9-10 days, crusts form. The total duration of the disease is 2-4 weeks. Sometimes there are relapses of the disease, mild forms are possible. The mortality rate is 10-15% (in severe forms of smallpox, 50-100%). The development of complications is due to the addition of secondary bacterial infection (pneumonia, sepsis, encephalitis, abscesses, phlegmon).

Diagnosis. To confirm the diagnosis, it is important to have epidemiological data (stay of an unvaccinated person in an endemic area, contact with exotic African animals). Laboratory methods include real-time PCR (100% specificity) and virological methods (isolation of the virus from infected 12-day-old chicken embryos). A complete blood count in the first days of the disease reveals lymphocytosis, in the suppurative stage - leukocytosis with a left shift, and an increase in ESR. Urinalysis may show albuminuria, cylinders, erythrocytes.

Differential diagnosis is made with smallpox, varicella, and vesicular rickettsiosis. In smallpox, there is no lymphadenitis, the size of the rash elements is smaller, prone to fusion, and the final scars are more pronounced. Vesicular rickettsiosis is not transmitted from humans, characterised by a primary affect 7-10 days before the onset of fever, with regional lymphadenitis, and healing lasts 3-6 weeks. Chickenpox is characterised by 3-4 waves of rash in 24-48 hours, with no stages.

Treatment. Treatment in a boxed infectious disease hospital, with strict isolation for 17 days (due to the difficulty of differential diagnosis with smallpox). No etiotropic therapy has been developed. Currently, the development of drugs based on

nucleoside analogues is being actively discussed. Careful skin and mucous membranes should be taken to prevent secondary bacterial infection: treatment of pustules with 2% potassium permanganate solution, application of tetracycline eye ointment behind the eyelids during the period of pustulation, treatment of the oral cavity with 3% borax glycerin or 1% sodium bicarbonate solution, control of cleanliness of linen. According to the indications, detoxification, analgesics, vitamins, broad-spectrum antibacterial drugs, sleeping pills. The discharge criterion is clinical recovery.

Prevention. Vaccination against smallpox is a method of specific prevention against VVM. In case of disease, quarantine is established in the area for 17 days, and contact persons are monitored in quarantine.

ACANTHAMOEBA. Protozoa, caused by pathogenic amoebae of the genus Acanthamoeba, usually manifests as acanthamoebic conjunctivitis and keratitis, dermatitis or granulomatous encephalitis. There are 6 species of amoebae that are pathogenic to humans: A. hatchetti (causing eye lesions), A. palestinensis and A. astronyxis (causing CNS lesions), A. polyphaga, A. Culbertsoni, A. castellanii (causing various lesions, including skin). The pathogen is an aerobic unicellular organism that lives freely in soil, water supply systems, stagnant fresh water of natural and artificial reservoirs, especially in sewage contaminated water. The best conditions for Acanthamoeba are water temperatures above 28°C and the presence of various organic substances. Under unfavourable conditions (lower ambient temperature, drying out of the reservoir, exposure to disinfectants and antiseptic substances), it can transform into cysts and remain viable. Upon ingestion, acanthamoebiasis pathogens revert to a vegetative state and become capable of parasitism. The source of infection is contaminated soil or water. Most often, the infection occurs through water and food, less often through contact with the environment (ingestion of pathogen cysts on the skin and mucous membranes of the eyes). Quite often, pathogenic amoebae can be detected in nasopharyngeal mucus and faeces of a healthy person without the development of a pathological process. With a decrease in the activity of the general or

local immune system (HIV infection, diabetes mellitus, malignant neoplasms, etc.), the number of protozoa increases with the development of disease.

Clinic. There are 3 main forms of acanthamoebiasis, characterised by different localisation and symptoms of the infectious process:

* Keratitis is a predominant lesion of the cornea, in which the patient complains of burning in the eyes, light intolerance, sandy sensation, blurred vision, lacrimation, and itchy eyelids. The progression of the disease without proper treatment leads to disc-like opacities, uveitis, iridocyclitis, and perforation (formation of a through hole) of the cornea.

* Acanthamoebiasis of the skin is a fairly common clinical form that can be of primary and secondary origin. In the case of primary development of the pathological process, the disease is self-limited (when water contaminated with amoebae cysts gets on the injured skin). Clinically, the pathological process is characterised by the appearance of small papules (nodules) or dark blue spots on the skin of the face, back, and chest. They have an average size of 0.5 to 3 cm in diameter. With the progression of the pathological process, spots and nodules turn into crusted ulcers. Subsequently, without adequate treatment, abscesses of muscles, lymph nodes, liver, lungs and other internal organs may form. The secondary process develops as a result of acanthamoebiasis of the brain.

* Central nervous system damage in acanthamoebiasis causes the development of granulomatous encephalitis, which occurs when acanthamoeba is haematogenously introduced into the brain from primary sites (cornea or skin), and from the moment pathogens enter the brain until the first clinical signs of the disease appear, it can take from several weeks to several years. The pathological process is localised in the brain tissue, basal ganglia, and vasculature. If the body's resistance is normal, granulomatous inflammation develops; if it is reduced, a necrotic process occurs. The initial period of acanthamoebiasis has no specific symptoms, characterised by intermittent subfibrillation, drowsiness, headache, lumbosacral muscle cramps, and convergence disorder. Progression of the process leads to coma and death.

Diagnosis. Diagnosis is based on the detection of vegetative and cystic forms of acanthamoebae in the test material. In case of keratitis, lacrimal fluid, corneal washings and scrapings are examined; in case of dermatitis, the content of infiltrates, skin biopsies; in case of granulomatous encephalitis, cerebrospinal fluid is examined. The culture method, serological tests, and biological samples are also used to verify the diagnosis of acanthamoebiasis. Due to the absence of specific symptoms, narrow specialists should be involved - ophthalmologist, neurologist, dermatologist - to exclude keratitis, encephalitis and dermatoses of other etiologies.

Treatment. The basis of adequate therapy is the use of antiprotozoal agents, antibiotics to which pathogenic amoebae are sensitive. Therapeutic approaches differ depending on the clinical form:

*Keratitis - mandatory refusal to wear contact lenses, topical remedies include hourly instillation of eye drops containing glucocorticoids, antibiotics (gentamicin, polymyxin, neomycin) and antifungal component (amphotericin, ketoconazole), applications with appropriate ointments. If there is a threat of corneal perforation, surgical treatment with keratoplasty is recommended.

*Skin lesions - systemic antibiotic therapy with aminoglycoside drugs, local topical application of ointments with neomycin, polymyxin is performed.

*Acanthamoeba encephalitis is fatal in most cases. Intravenous administration of amphotericin B is prescribed, and a combination of trimethoprim and sulfamethoxazole, aminoglycosides may be used.

Prevention consists in following personal hygiene rules and limiting contact with contaminated water bodies that are habitats for acanthamoebae.

PNEUMOCYSTIS JIROVECII. A protozoal disease caused by Pneumocystis jiroveci, which is one of the most common causes of pneumonia in cancer patients receiving immunosuppressive therapy, AIDS patients (80% of all opportunistic infections), and primary immunodeficiencies. The causative agent is Pneumocystis jiroveci, a member of the unicellular fungi Ascomycetis fungi, widely distributed among humans and animals (from 1 to 10% of healthy people are carriers

of pneumocystis). They are transmitted by airborne droplets. The natural habitat under normal conditions is the lungs of humans and some mammals, but the pathological process occurs only in immunocompromised individuals. The life cycle is structured in such a way that 2 main forms of this microorganism can be detected in the alveolar tissue: small uninucleated trophozoites (1-5 μm) and cysts (10 μm) with a thick wall and containing 2 to 8 cells (1-2 μm) called sporozoites. When a mature cyst ruptures, the sporozoites continue their developmental cycle in the alveoli, turning into trophozoites, or are released into the external environment (with mucus droplets when coughing).

Pathogenesis. The leading role in the development of the pathological process is played by disorders of cellular immunity mechanisms, severe dystrophy of type I alveolocytes and reparative hypertrophy of type II alveolocytes, which gradually disrupt the gas exchange functions of the alveolar epithelium, characterised by the development of hypoxaemia, respiratory alkalosis, and increased alveolar-capillary permeability. With a defect in cellular immunity and a decrease in the functional activity of alveolar macrophages, the pathogen actively multiplies and its endogenous stages (trophozoites, precysts and cysts) fill the alveolar cavity. The alveolar exudate takes on a typical foamy appearance, and microscopy reveals pneumocysts, cellular detritus, leukocytes, and fibrin. As a result of these processes, severe respiratory failure of the restrictive type develops. Extra-pulmonary lesions are rare, associated with impaired cellular immunity, and may affect the internal organs of the gastrointestinal tract, heart, thyroid gland, skin, lymph nodes, and bone marrow.)

Clinic. The incubation period is 1-8 weeks. Patients with AIDS may have a prodromal period of 2 to 10 weeks. The edematous (first) stage lasts 7-10 days: gradual development of subfebrile, moderate dyspnoea during exercise, dry cough, and signs of intoxication. Auscultation reveals unstable, differently calibre, moist rales.

The atelectatic stage lasts 3-4 weeks, is characterised by fever up to 39-40°C, dyspnoea at rest, increased dry cough (productive cough is rare), and sputum production is possible. Dry, less frequently moist rales and crepitations in the basal regions are often heard in the lungs. Examination reveals tachypnea, tachycardia,

cyanosis, hepatosplenomegaly. Dyspnoea gradually increases, up to 40-60 respiratory movements per 1 minute, pneumothorax, cardiopulmonary insufficiency are possible.

The emphysematous stage is characterised by a decrease in dyspnoea, improvement of the general condition, normalisation of temperature, and a boxy sound on percussion examination.

Pneumocystis pneumonia (PCP) in AIDS is usually characterised by a long chronic course. Initially, no auscultatory symptoms are detected, and the radiological picture may also remain without pathological changes. With the progression of the disease, bilateral root infiltrates appear, which then transform into focal or interstitial changes. Occasionally, solitary nodules are detected, which can be necrotised with the formation of a large central cavity.

Complications: RDS, pneumothorax, pulmonary infiltrates abscessing due to secondary bacterial and fungal flora, exudative pleurisy.

Diagnosis. To detect Pneumocystis jiroveci, sputum examination is required (usually obtained after ultrasonic inhalation with hypertonic NaCl solution), and if the result is negative, bronchoalveolar lavage (BAL) is performed. The BAL method is a common method of diagnosing PCa, with a diagnostic value of more than 90%, and transbronchial biopsy using fibrobronchoscopy is also possible. Chest X-rays at an early stage of the disease show almost no pathology, while later on, root diffuse cloudy infiltrates in the form of a butterfly can be seen. CT scan of the chest cavity can reveal thickened interlobular septa in the lung tissue and the symptom of "frosted glass" (reduced transparency of the lung tissue with a vascular pattern on this background). Direct RIF with monoclonal antibodies, RNIF to detect the pathogen in lavage fluid or sputum, and quantitative PCR are also used. It is imperative to test a patient with HIV when a PCR is detected.

General blood test - anaemia, leukopenia, thrombocytopenia, eosinophilia, significantly increased ESR (40-60 mm/h). The LDH test is prognostic: the level of lactate dehydrogenase reflects the level of lung damage, correlates with respiratory failure, and activity over 500 IU/l is an unfavourable sign.

Differential diagnosis is carried out with pulmonary tuberculosis, pulmonary embolism, other pneumonias (fungal, chlamydial, cytomegalovirus, mycoplasma, legionella and other bacterial etiologies).

Treatment. Treatment is carried out in an infectious diseases hospital. The main etiotropic drugs are trimethoprim-sulfamethoxazole (Biseptol, Bactrim) and pentamidine isothionate. Trimethoprim-sulfamethoxazole is administered intravenously 20mg/100mg/day in 500ml of 5% glucose solution for 2 weeks, then orally for another 2 weeks. Pentamidine is prescribed for long-term therapy of PCP, a dose of 4mg/kg/day intravenously or intramuscularly in 250 ml of 5% glucose solution for 3 weeks. It is advisable to prescribe folic acid preparations at the same time. For inhalation, an aqueous solution of pentamidine isothionate is available (600 mg per inhalation for 3 weeks). If within 4-5 days a patient with PCP without HIV infection (in patients with HIV on day 8) has no effect from the prescribed therapy, it is possible to use alternative drugs: alpha-difluoromethylornithine (DFMO) 18 g/1m2 of body surface /day for 8 weeks, trimethrexate, eflornithine, atovaquone. In AIDS, anti-relapse chemotherapy is mandatory, primarily when the CD4 cell count reaches 200 or less cells per 1 μL. To prevent relapses, bactrim, dapsone, and the aerosol form of pentamidine are used.

Prevention. In wards with immunocompromised patients, if a patient with pneumocystosis is detected, isolation is required. Chemotherapy is used to prevent the development of pneumocystosis.

TOXOPLASMOSIS is a zoonotic parasitic disease of humans and animals, characterised by damage to the nervous system, myocardium and eyes, lymphadenopathy, and hepatosplenomegaly. The disease is included in the list of HIV-associated diseases, can be activated in the late stages of HIV infection, and causes mostly severe encephalitis. The causative agent is Toxoplasma gondii, an obligate intracellular parasite of the class Sporozoites, subclass Coccidia. Toxoplasma has a complex life cycle, goes through phases of sexual and asexual reproduction. There are three main periods of development: tachyzoite (trophozoite), tissue cyst (bradyzoite),

and oocyst (sporozoite). Sexual development (gametogony) of the parasite occurs in the epithelial cells of the small intestine of the final host - domestic cats and other felines - with the formation of immature oocysts, which are excreted in the faeces into the environment. A few days later, they form sporozoites in the soil that can become invasive for a year. Asexual reproduction of toxoplasma occurs in the macrophage system of intermediate hosts - mammals (including humans) and some bird species. In the process of asexual development, trophozoites (reproduction stage) and tissue cysts (persistence stage) are formed. The intermediate host does not release the pathogen into the environment. Toxoplasma is widespread in nature, and due to its high susceptibility, the population's infection rate ranges from 10 to 90%. An important feature is the possibility of intrauterine infection with severe developmental abnormalities. In most people, infection occurs at an early age. In an infected person with normal immunity, tissue cysts are formed (mainly in the muscles and brain), which contain live, slowly dividing protozoan forms that remain in the human body for life.

Human infection occurs through:

1) food (vegetables, fruit), water and hands contaminated with oocysts excreted in cat faeces; consumption of meat from infected animals without sufficient heat treatment containing protozoa (most often pork and lamb), unpasteurised milk; contact with contaminated soil;

2) rarely - transmission of tachyzoites from a pregnant woman to the fetus through the placenta during parasitemia (vertical infection, exclusively during primary infection);

3) very rarely - accidental contact with tachyzoites (occasionally transfusion of blood or blood products - mainly leukocyte concentrate, organ transplantation, laboratory infection, skin damage with a contaminated instrument).

Toxoplasmas are found in all human biological fluids, but due to the low number of pathogens, there is no human-to-human transmission (except for the transplacental route in pregnant women). Toxoplasmosis is not transmitted by sexual or airborne droplets.

Pathogenesis. In 95% of cases, toxoplasma infection occurs orally: cysts or oocysts enter the gastrointestinal tract, penetrate the epithelium of the lower small intestine, and then the mesenteric lymph nodes. As a result of the pathogen accumulation, mesadenitis occurs, which, in the context of a decrease in the body's defence barriers, leads to parasitemia with subsequent dissemination. The formed cysts exist for life in the liver, spleen, muscles, brain, and eye tissues. The process of toxoplasma multiplication is accompanied by the release of toxins and allergens. The acute or latent form is followed by a chronic form.

In congenital toxoplasmosis, parasitemia results in the formation of a primary focus in the placenta, and the severity of the consequences for the fetus depends on the gestational age. Early infection results in stillbirth, spontaneous miscarriage, severe malformations, or the development of a generalised form of toxoplasmosis. In the last trimester of pregnancy, asymptomatic forms with late onset of clinical symptoms prevail.

Risk factors for the development of severe form (generalised form, ocular form) are immunodeficiency states of any etiology, especially immunosuppressive therapy after organ transplantation; chemotherapy of malignant tumours; immunosuppressive treatment for other reasons; HIV infection (toxoplasmosis of an internal organ indicates AIDS); embryonic period. The development of immunosuppression can lead to reactivation of the disease: tissue cysts rupture and latent forms contained in them turn into invasive tachyzoites (endogenous invasion).

Clinic. The incubation period of the acquired form is from 2 weeks to 2 months (average 4 weeks). Parasitaemia lasts 1-3 weeks The risk of fetal infection is 17-25% in the first trimester of pregnancy, 25-54% in the second trimester and 60-90% in the third trimester. The patient does not infect contact persons. The clinical course depends on the type of invasive protozoan form, source of infection, pathogenicity of the strain, state of the immune system and intensity of infection. In immunocompetent individuals, it is usually asymptomatic or mild (85% of cases). Depending on the mechanism, acquired and congenital toxoplasmosis are distinguished.

Congenital toxoplasmosis occurs as a result of an acute infection in a pregnant woman. Generalised, manifest forms occur in 25-30% of cases, in other cases the course is latent. The severity of the disease depends on the state of the newborn's immune system and the availability of preventive treatment for the pregnant woman. Clinically, congenital toxoplasmosis is characterised by CNS lesions (encephalitis, convulsions, calcifications, hydrocephalus), thrombocytopenia, eye lesions (nystagmus, anophthalmia, microphthalmia, eye muscle paralysis), and jaundice.

Acquired toxoplasmosis is latent in most cases (80-90%) and is detected by chance during serological examination. Acute toxoplasmosis is characterised by lymphadenopathy and intoxication. One group of lymph nodes (cervical, jaw, supraclavicular, axillary, mesenteric) is more often affected, generalised lymphadenopathy is less common. Depending on the symptoms, there are encephalitis, typhoid, and mixed forms. After the primary infection, non-sterile immunity is formed, and after the acute process subsides, the transition to a secondary chronic form is possible.

Chronic acquired toxoplasmosis has a latent course and is diagnosed serologically. Clinical manifestations may include lymphadenopathy, prolonged subfebrile, various allergic reactions, arthralgia, myalgia, carditis, haemorrhagic syndrome (haemorrhages, bleeding), eye lesions (chorioretinitis, uveitis), CNS lesions (neuroses), and vegetative disorders.

In most cases, toxoplasmosis in pregnant women is subclinical and diagnosed serologically. Infection in the first trimester of pregnancy leads to spontaneous miscarriages, intrauterine death of the fetus or severe congenital malformations; in the second trimester, 20-30% of newborns have various manifestations of congenital pathology (chorioretinitis, seizures, brain calcifications, microcephaly, anophthalmia). In the case of infection in the third trimester and intrapartum, about 5-10% of newborns have clinical manifestations of active infection, and signs of CNS and eye damage occur several years later in infants without clinical manifestations of infection.

Toxoplasmosis in patients with HIV/AIDS: when the CD4 cell count drops below 200 cells/mL, the latent and chronic forms may turn into generalised forms.

Clinically, the disease is severe, with predominantly nervous system involvement (encephalitis, panencephalitis, myelitis, arachnoiditis), pneumonia, myocarditis, chorioretinitis, small intestine and other internal organ damage.

Questions for students' self-control

1. General and specific prevention of protozoan diseases
2. Universal precautions to prevent monkeypox infection
3. Post-exposure prophylaxis of leprosy
4. Activities with an increased risk of HIV infection
5. Universal precautions for healthcare workers
6. Safety precautions for invasive manipulations
7. Clinical observation of persons receiving drug prophylaxis
8. Rules for filling in medical records.
9. Measures to be taken in the event of contamination with infectious material in the workplace

Test control of knowledge

>>In immunocompetent individuals, toxoplasmosis is more often characterised by <<

( ) Eye damage

(x) Asymptomatic

( ) Nervous system disorders

( ) Myocardial damage

( ) Manifesto course

>> Vaccination to prevent toxoplasmosis <<

( ) is performed on women of reproductive age

( ) is administered to persons with immunosuppression

( ) is given to children

( ) is carried out by veterinarians

(x) not developed

>>A 32-year-old woman with an asymptomatic course of the disease gave birth to a stillborn child with severe microcephaly for the second time. What disease should the doctor think about first of all?

(x) Toxoplasmosis

( ) Listeriosis

( ) Histoplasmosis

( ) Syphilis

( ) Brucellosis

>>The most typical morphological element in lepromatous leprosy: <<

( ) erythematous spot;

( ) exudative papule;

( ) hillock;

( ) bubble;

(x) a node.

>>Which of the following AIDS-associated parasitoses can be contracted directly from a person with HIV/AIDS?

( ) Leishmaniasis

( ) Toxoplasmosis

(x) Pneumocystosis

( ) Strongyloidosis

( ) Malaria

>>Which of the following factors is not associated with the development of pneumocystis pneumonia?

( ) Prematurity of children

( ) Congenital immunodeficiency

( ) HIV/HCV infection

( ) Immunosuppressive chemotherapy

(x) Presence of chronic gastro-duodenal pathology

>>In which of the following materials can pneumocysts not be detected?

( ) Laryngeal and tracheal swabs (laryngoscopy)

( ) Sputum smears

(x) Blood smears

( ) Lavage fluid

( ) Bronchial secretion

Example of a case study

A 43-year-old man complained of ring-shaped lesions on the skin of the right cheek, hair loss in this area. He considers himself sick for 2 months. At first, small red papules appeared, which gradually merged to form a ring-shaped shape. 2 years ago, hair began to fall out in this area. On the skin of the right cheek there are small reddish-blue flat, polygonal, peeling papules that have merged in the form of a ring. Hair loss is noticeable at the site of the lesion. Pain, tactile, and thermal sensitivity in this area is reduced.

1. Your probable diagnosis and its justification;

2. Treatment and preventive measures.

ANSWER:

-Tuberculoid leprosy (usually debuts with skin lesions (mainly spots and tubercles) prone to regression in the early stages, nerve trunks are involved in the process with the development of various neurological disorders, the degree of systemic disorders is not significant).

-Treatment (complex): antibacterial, immunotropic, general tonic drugs; physiotherapeutic methods; proper nutrition; treatment of concomitant diseases

Topic 4. Worm infestations of tropical and subtropical regions: ankylostomiasis, clonorchiasis, fascioliasis. Paragonimiasis. Strongyloidiasis as an HIV-associated helminthiasis. Dracunculiasis.

Learning objectives: to acquire basic knowledge and skills in parasitology, which will allow to predict the course of the disease and anticipate the development of complications, master diagnostic methods and be able to use the knowledge gained in practice.

List of skills

- Take a medical history with an assessment of epidemiological data.
- Examine the patient and identify the main symptoms of infections.
- Conduct differential diagnosis with other parasitic, infectious and non-infectious diseases with similar symptoms.
- Conduct a targeted examination according to the diagnosis.
- Prescribe highly effective medications for treatment.
- Create a plan of preventive measures.

Hardware and software:

- A personal computer, laptop, tablet, smartphone or similar device,
- Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar operating system.
- Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
- Access to the global network, local network,
- Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Continuous surveillance, HIV-associated helminthiasis, source of worm infections, ICF, Parasitology

ANKYLOSTOMIDISIS

Infections caused by some helminths from the Ancylostomatidae family, which belong to the subclass "Diseases caused by hookworms", are quite widespread in the world.

Ankylostomiasis (synonym - Ancylostomiasis, hookworm discases).

Etiology.

Ankylostomiasis unites 2 helminthiasis, similar in epidemiological and clinical manifestations. The causative agent of ankylostomiasis is Ancylostoma duodenale; necatorticollis is Necator americanus.

Size of female Ancylostoma duodenale: 10-13 * 0.4-0.6 mm; male size: 8-11 * 0.4-0.5mm

Size of female Necator americanus: 7.6-13.5*0.3-3.5mm; size of male: 5.5-10*0.2-0.25mm.

Necator eggs are similar to hookworm eggs.

Epidemiology

This infection affects more than 1 billion people on the planet. Ancylostoma duodenale is predominant in the Middle East, North Africa, and India, while Necator americanus is distributed in North and South America, Central and Southern Africa, Southeast Asia, China, and Indonesia.

In the absence of proper epidemiological surveillance, these helminths are prone to form foci of infection in underground mines, even in temperate climates, as the temperature inside (in the mines) is constantly high, which favours the development of the pathogen. The source of infection is a sick person who excretes immature hookworm eggs. Larvae in the external environment can develop at temperatures ranging from 14 to 40°C (optimal 27 to 30°C) in the presence of high humidity and sufficient aeration. Hookworm infection is most commonly transmitted through vegetables, fruits, herbs and dirty hands.

Infection with necatosis occurs through contact with the soil, most often when walking barefoot or lying on the ground. However, infection is also possible through exposed areas of the upper limbs if the person is wearing shoes, as the larvae can crawl out of the soil onto the grass. Direct contact with the patient does not pose a danger to others.

Pathogenesis

Hookworm larvae enter the host mainly through the mouth and develop in the intestine, namely the 12th cecum, without migration. The necator larvae penetrate the

skin and enter the capillaries, migrating through the large and small circulation. Finally, having reached the lungs, they enter the gastrointestinal tract through the respiratory tract, larynx, and pharynx, where they develop into adult worms in 4-5 weeks. In addition to feeding on blood, helminths secrete special anticoagulants that contribute to prolonged bleeding.

Clinic

When hookworm larvae penetrate the skin due to their migration through the body, the initial clinical picture is presented by pathology of the chest cavity organs, eosinophilic infiltrates in the lungs and vascular pneumonia with fever and high (up to 30-60%) eosinophilia. Tracheitis, laryngitis, sometimes up to aphonia, are observed.

The intestinal phase manifests itself 30-60 days after infection, followed by intestinal syndrome (vomiting, unstable stools, abdominal pain, general malaise, weakness).

Diagnostics

Hemogram: hypochromic anaemia with clinical manifestations (general weakness, dizziness, shortness of breath, fatigue, weight loss, decreased appetite, and rarely - increased appetite). Often a perverted taste (eating lime, paper, licking metal objects)

The blood smear showed microcytosis, aniso-poikilocytosis, and erythrocyte hypochromia.

Red blood cells are found in the faeces (coprocytogram). The Gregersen's reaction to "hidden" blood is almost always positive.

The specific diagnosis is aimed at detecting ankylostomid eggs in faeces or duodenal contents by means of a native smear on a large glass slide.

Complications: infection of the skin in the area of the entrance gate with the development of inflammatory reactions; allergic manifestations:

- laryngospasm, sometimes Quincke's edema;
- hypochromic anaemia;
- In children, with prolonged parasitisation, there may be a delay in physical and mental development.

Treatment

1. Albendazole (Aldazol, Wormil, Nemozol) - 400mg once an hour after a meal, preferably after a light dinner.
2. Mebendazole (Vermox) -100mg*2p/day for 3 days.
3. Efficacy monitoring 3 weeks after treatment, three times with an interval of 2-3 days.
4. Pyrantel - pamoate (Helmintox, Combantrin) - 11mg/kg body weight (daily dose divided into 3 doses, with meals) for 1-3 days.
5. Levomizole (Decaris, Ketrax) - 150 mg once, 1 hour after meals.

Prevention

1. Identification and treatment of patients.
2. Carrying out sanitary measures.
3. Compliance with personal hygiene rules.
4. Do not walk barefoot in ankylostomiasis foci, rest on the ground or lawns.

FASCIOLOSIS

Fasciolosis is a bihelminthiasis with predominantly hepatobiliary system involvement and a tendency to chronicity of the process.

Etiology

Pathogens: 2 species of fasciola - Fasciola hepatica; Fasciola gigantica.

In Ukraine, there are sporadic cases of fasciolosis caused by Fasciola hepatica.

Trematode size: 30mm*10-12mm, which mainly develops in the liver and biliary tract. In the human body, the age of fascioles lasts from 3-5 to 20 years.

The final host is cattle, horses, and rarely humans, which excrete fasciolar eggs into the environment in their faeces.

The intermediate host is a freshwater mollusk, in whose body larvae mature and actively enter the water where they turn into adolescent larvae, or invasive larvae.

Epidemiology

Human infection occurs through ingestion of contaminated water and consumption of contaminated vegetables and herbs. Infection usually occurs in the summer months.

In Ukraine, there are endemic foci in the western region (Lviv oblast). A sick person is not a source of infection.

Pathogenesis

Entering the gastrointestinal tract with contaminated food and water, adolescents turn into larvae that migrate through the intestinal wall into the abdominal cavity and then through the capsule and liver parenchyma to the bile ducts. In 3 to 4 months after maturation, sexually mature helminths moving through the bile ducts injure and sometimes lead to obstruction, with the development of mechanical jaundice. Allergic reactions due to the absorption of metabolic products.

Clinic

In the fasciolosis clinic, acute and chronic phases of the disease can be distinguished.

The incubation period is 2-8 weeks.

The acute stage of the disease, which lasts 3-4 months, is characterised by a picture of acute allergy with liver damage. The majority of patients have a manifest course - remitting fever, symptoms of intoxication, hepatomegaly, eosinophilia, anaemia, sometimes jaundice and allergic rash Complaints of weakness, decreased appetite, headache, vomiting, pain in the right hypochondrium. During this period, patients are diagnosed with eosinophilia (30% to 80%). The chronic period is characterised by symptoms of dyskinesia, hepatocholecystitis with a tendency to periodic exacerbations. The size of the liver increases, jaundice appears, bilirubin levels can rise to 100 μmol/l with a slight increase in aminotransferase activity. Patients lose weight, and hypochromic anaemia occurs. In the case of bacterial infection, a cholangitis clinic develops - hectic fever, neutrophilic leukocytosis, a significant increase in ESR, increased levels of total bilirubin (up to 200 μmol/l) and alkaline

phosphatase, dysproteinemia with a decrease in the percentage of blood albumin against the background of increased levels of α and α2 globulin fractions.

Prognosis: in the presence of primary or acquired immunodeficiency, the course of fasciolosis is particularly severe.

Diagnosis of acute forms:

serological methods: PCR, ELISA. The use of ELISA is promising.

Diagnostics of chronic stages - parasitological examination - detection of fasciola eggs in duodenal contents and faeces.

To detect adult parasites, ultrasound is used, as well as endoscopic retrograde cholangiography, which detects filling defects in the choledochus; CT with contrast.

Treatment:

The drug of choice for the treatment of fasciolosis is triclabendazole in a single dose of 10-12 mg/kg body weight. The efficacy is close to 100%.

Bitinol 25 mg/kg body weight per day for 10 days or 30 mg/kg per day every other day (5 times in total). These drugs affect both adult and young fascioles. Therefore, they can be prescribed for both acute and chronic fasciolosis. They are low-toxic, rarely cause side effects, and are used in paediatric practice. However, these drugs are not yet registered in Ukraine.

Medical examination. Follow-up is carried out for 1-2 years. The first year - a three-time control examination 4-6 months after treatment, then every 3 months. In the second year - once a quarter.

Prevention:

- detection and treatment of sick people;
- examination and treatment of dogs;
- compliance with personal hygiene rules (hand washing, handling greens, vegetables, berries containing soil particles);
- Compliance with public hygiene rules (protection of playgrounds, parks and squares from animal visits).

CLONERHOUSE

Clonorchosis is a biological helminthiasis that is accompanied by damage to the hepatobiliary system and pancreas.

The causative agent is the Chinese two-spotted bug (Clonorchis sinensis), belonging to the family Opisthorchide of the class Trematoda of the type Plathelhelmintes.

Epidemiology

Human infection occurs when eating insufficiently cooked fish containing invasive metacercariae. Men of working age are more likely to be affected. Clonorchiasis is seasonal - summer and autumn, as with opisthorchiasis.

The disease is endemic in nature: these are the regions of China, Korea, Vietnam, and Japan. There may be imported cases in Ukraine.

Pathogenesis.

The cyst shell of the metacercariae dissolves when it enters the small intestine of humans or domestic and wild animals, and the larvae enter the liver through the portal system, or choledochus, where they turn into sexually mature two-earthers, which begin to lay eggs after a month of being in the host. Similar larval development can also occur in the pancreatic ducts.

The first 1-2 months are characterised by toxic and allergic phenomena. Subsequently, the clinic is caused by direct damage to the biliary tract, which leads to the development of cholangitis and cholestasis, and subsequently causes carcinogenesis; biliary dyskinesia (biliary dysfunction).

Clinic:

It has an acute onset, characterised by various allergic manifestations: fever, generalised lymphadenopathy, polymorphic skin rash, often accompanied by itching, and hepatomegaly.

Indigenous people living in endemic regions suffer from the disease without any clinical symptoms. In non-natives, the disease becomes manifest with hyperleukocytosis (25-30*109/L); hyper-osinophilia (up to 60-70%), and an increased ESR (up to 40 mm/h).

With the progression of the disease, untreated patients develop symptoms of cholangitis, choledochal obstruction, which can lead to the formation of biliary cirrhosis.

A typical complication of clonorchiasis is purulent cholangitis.

Diagnosis:

The main method is microscopic examination of faeces (detection of eggs), more likely in the chronic period.

Serological diagnostic methods (ELISA) are not used in Ukraine.

Treatment:

Etiotropic therapy: Praziquantel 75mg/kg body weight daily dose divided into 3 doses after meals within one day. The maximum single dose is 2g.

Pathogenetic and symptomatic therapy: acute period: detoxification and anti-allergic (Loratadine, Cetrin, Citrileve) therapy. Antibiotics - in case of bacterial flora.

Dispensary observation is not regulated.

Prevention:

Sanitary and educational work in natural clonorchiasis areas. Eat only sufficiently heat-treated fish.

PARAGONIMOSIS

Paragonimosis is a biogeolithiasis caused by the pulmonary fluke Paragonimus westermani and characterised by predominant lung involvement.

Etiology

Helminth of the genus Paragonimidae of the class Trematoda of the type Plathelhelmintes - size: length 8-12 mm, width 5-6 mm, covered with cuticle with spines, produces oval eggs, size 60-80 * 50-55 microns, yellow-brown in colour with a lid.

In water, after 1-3 months, myracidia are released from the lung fluke eggs, which penetrate the first intermediate host - freshwater gastropods, where sporocysts, redia and cercariae develop. The latter are able to penetrate additional intermediate

hosts, such as freshwater crayfish and crabs, where they incubate and develop into invasive metacercariae. The final hosts are pigs, cats, dogs, rodents and humans.

Epidemiology

Paragonimosis is endemic in Southeast Asia (China, Indonesia, Taiwan, Philippines) and South America (Colombia, Ecuador, Peru). Imported cases may be reported in Ukraine.

The source of infection is the final host, which excrete eggs in urine and faeces.

A person becomes infected by eating meat from insufficiently heat-treated freshwater crayfish and crabs, as well as by swallowing river water while swimming, which contains invasive metacercariae.

Pathogenesis

The invasive metacercariae enter the duodenal section of the small intestine, where the cyst membrane dissolves and the larvae penetrate the intestinal wall into the peritoneum, and then through the liver, pancreas and diaphragm into the pleural cavity and lungs.

During the migration stage, an allergy pattern develops.

After 2-3 months from the onset of the disease, adult worms form in the lungs from larvae that are encapsulated. The disease progresses to the chronic stage, which is characterised by the formation of parasitic cysts. In case of cyst destruction, adult helminths and their eggs may be introduced into the mesenteric lymph nodes, prostate, skin, brain, and other organs.

Clinic:

The incubation period is 2-3 weeks.

The following clinical forms of paragonimosis are noted:

- acute abdominal;
- acute pleuropulmonary;
- chronic pleuropulmonary.

Acute abdominal paragonimosis in many patients is accompanied by an acute abdominal clinic (with aseptic peritonitis, enteritis, hepatitis with hepatomegaly, elevated bilirubin and a slight increase in transaminases).

Acute pleuropulmonary paragonimosis is characterised by the progression of toxic and allergic phenomena (bronchial obstructive syndrome, urticular rash; temperature reaches 38-39°C, haemogram shows eosinophilia (up to 15-30%).

The chronic stage of the disease develops in 2-3 months. In the clinic: chest pain, cough with purulent bloody sputum, shortness of breath, night sweats, weight loss, hyperthermia.

Complications: Pulmonary haemorrhage and pneumothorax may occur in the unfavourable course of the disease. It is possible to develop parasitic cysts in the brain substance, which leads to cerebral paragonimosis in the form of encephalitis or meningoencephalitis.

Diagnosis:

Migration stage: ELISA (detection of antibodies against helminth antigens).

Chronic stage: excretion of helminth eggs in faeces, pleural exudate and cerebrospinal fluid.

In case of pleural paragonimosis, a tissue biopsy is performed.

Treatment:

1. Etiotropic therapy:
    a. Praziquantel 75mg/kg - a daily dose of 3 doses after meals for 1-2 days.
    b. Triclabendazole (but not registered in Ukraine).
2. Detoxification.
3. Antihistamine.

A month after treatment, sputum examination is required three times! If no helminths or eggs are found, patients are removed from the dispensary register.

DRACUNCULOSIS

Etiology

Dracunculiasis (also known as rishti disease) is a disabling parasitic disease caused by Dracunculus medinensis, a long threadworm. It is transmitted exclusively when people drink water contaminated by water fleas that are infected with the parasite.

Dracunculiasis is a disease that is on the verge of elimination: in 2017, only 30 cases of the disease were registered.

The disease is usually transmitted when people with limited or no access to drinking water supplies drink water contaminated with fleas that are infected with the parasite.

Out of 20 countries endemic in the mid-1980s, only two countries reported cases in 2017 (Chad (15) and Ethiopia (15)).

From the time of infection, 10-14 months pass, after which the developmental cycle is completed by the release of an adult from the human body.

Dracunculiasis is rarely fatal, but infected people cannot live normally for months. The disease affects people living in rural, poor and isolated communities who consume water mainly from open water bodies.

Transmission, life cycle and incubation period

Approximately one year after infection, a painful blister forms - in 90% of cases on the lower leg - and one or more worms are released, accompanied by a burning sensation. To relieve the pain, patients often immerse the infected part of their body in water. The worm then releases thousands of larvae into the water. These larvae become infectious when they are swallowed by tiny crustaceans, or copepods, also called water fleas. People drink contaminated water and swallow infected water fleas. The water fleas die in the human stomach and the infective larvae are released. They then penetrate the intestinal walls and migrate through the body. The fertilised female worm (60-100 cm long) migrates under the skin tissue until it reaches an exit point, usually on the lower limbs, forming a blister or swelling from which it is eventually released. Migration and release (incubation period) of the worm takes 10 to 14 months after infection.

Prevention:

There is no vaccine or cure for this disease. However, prevention is possible, and it is thanks to preventive strategies that this disease is on the verge of being eliminated. Here are some of these strategies:

- strengthening of surveillance to detect any case of the disease within 24 hours of the worm's detection;
- prevention of transmission of infection from each worm by treating and cleaning the damaged skin area and regularly applying bandages until the worm is completely released from the human body;
- prevention of drinking water contamination based on recommendations to patients not to enter the water;
- Ensuring greater access to safe drinking water supplies to prevent infection;
- filtration of water from open water bodies before drinking;
- controlling vectors of infection through the use of the larvicide Temephos;
- Promoting health education and behaviour change.

The path to liquidation

In May 1981, the Inter-Agency Steering Committee for the Joint Action for the International Decade for Drinking Water Supply and Sanitation (1981-1990) proposed the elimination of hookworm as an indicator of the success of the Decade programme. In the same year, WHO's decision-making body, the World Health Assembly (WHA), adopted a resolution (WHA 34.25) recognising that the International Decade for Drinking Water Supply and Sanitation provided an opportunity for the elimination of draculiasis. Subsequently, WHO and the US CDC formulated a strategy and technical guidelines for the eradication campaign.

According to WHO recommendations, in a country where rickettsial disease has recently been interrupted, active surveillance should be carried out for at least 3 years. This is to ensure that no cases are missed and that the disease does not re-emerge.

STRONGILOIDISIS is an anthroponosis, percutaneous and oral geohelminthiasis characterised by a chronic course and the presence of two stages -

early (migration) and late - with damage to the gastrointestinal tract, hepatobiliary and nervous systems.

Etiology

The causative agent is Strongyloides stercoralis.

Male 0.7 mm long, 0.04-0.06 mm wide, female 2.2 mm long, 0.03-.07 mm wide parasitise the human small intestine. Eggs are oval, transparent.

Epidemiology

The disease is common in regions with warm and humid climates (Eastern and Southern Africa; Southeast Asia; South America).

Infection occurs through contact with the soil: when working in the garden, walking barefoot on the ground, on the beach (percutaneous route), as well as through food (when consuming vegetables and fruits) and water.

In the body, the larvae migrate through the blood and lymphatic vessels, then settle in the upper part of the small intestine and grow to adults.

Adult worms lay eggs that hatch into larvae in the intestines. These larvae can migrate through the body, causing a variety of allergy symptoms.

If the patient's immune system is severely impaired, the migration of strongyloides can be delayed for a long time, and the number of migrating larvae increases.

All this can cause severe disorders in the patient's body.

Strongyloidiasis is particularly severe in patients with AIDS, as hyperinvasion develops. In this case, the parasitic process is generalised, affecting various organs and tissues. A large number of parasites accumulate in the host's body, the disease becomes progressive with exhaustion of the body, and also becomes malignant with the development of destructive changes in the intestinal mucosa, and may be complicated by the development of peritonitis.

The most dangerous complication of strongyloidiasis is the generalisation of the disease (disseminated strongyloidiasis); the spread of larvae throughout the body leads to myocardial damage, meningoencephalitis and septicaemia.

Treatment:

1. Etiotropic therapy:
    a. albendazole 400 - 800 mg/day in 1-2 doses, 3-5 days;
    b. Carbendacim and mebendazole 10mg/kg/day - 3-5 days (1-2 courses).
2. Pathogenetic therapy:
    a. desensitising drugs;
    b. antispasmodics.

Treatment control in 1-2-3 months - no larvae are detected in faeces and bile.

Dispensary supervision - 6 months with monthly monitoring of the examination.

Prevention:

- detection and treatment of patients;
- compliance with the rules of personal hygiene;
- carrying out sanitary measures.

Presentation:

https://docs.google.com/presentation/d/1vBxPlqevR3UDeamOUmoqKojF7IeZLhco/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for students' self-control

1. The concept of parasitology
2. State the main features of fasciolosis.
3. Methods of laboratory diagnosis of strongyloidiasis, treatment features.
4. When strongyloidiasis becomes malignant and generalised.
5. Clinical manifestations of ankylostomiasis, including the pathogenesis of the disease.
6. Describe clonorchiasis.

Glossary

Continuous surveillance - active surveillance for at least 3 years to ensure that the disease does not reappear in areas endemic for the disease.

HIV-associated helminthiasis is a particularly severe course of helminthiasis in the setting of immunosuppression, which can lead to death.

The source of worm infestations is an infected person or animal, whose body is a natural habitat for the parasite.

ICLD - International Commission for the Certification of Dracunculiasis Eradication.

Parasitology is the science that studies parasitic diseases, the main biological feature of which is the inability to reproduce in the body of the final host.

Worm infestations in tropical and subtropical regions:

- ankylostomiasis:
- fasciolosis;
- clonorchiasis;
- paragonimosis;
- dracunculosis;
- Strongyloidiasis as an HIV-associated helminthiasis.

Test control of knowledge

>>What complications can clonorrhoea lead to, EXCEPT: <<

( ) cholangitis;

( ) PBC (primary biliary cirrhosis);

(x) myocarditis;

( ) cholangiocarcinoma;

( ) LWD.

>>Cestodoses (tapeworms) include all, EXCEPT: <<

( ) diphyllobothriosis;

( ) hymenolipedosis;

( ) tenariinchosis;

( ) tenebrosis;

(x) clonorchiasis.

>>What is the mechanism of transmission of infection in dirofilariasis?

(x) transmission;

( ) food;

( ) percutaneous;

( ) aerogenic;

( ) contact and household.

>>Nematodes (roundworms) include all of the following EXCEPT: <<

(x) Strongyloidiasis;

( ) trichinosis;

( ) dirofilariasis;

( ) ascariasis;

( ) enterobiosis.

>>Fasciolosis pathogens parasitise in: <<

( ) vessels;

( ) muscles;

(x) bile ducts;

( ) colon;

( ) pancreatic duct.

>>Transmission of heartworm to humans occurs as follows: <<

( ) by airborne droplets;

( ) percussion;

( ) when swimming in a contaminated water body;

(x) by transmission;

( ) by food.

>> In dracunculosis, the transmission factor of the infection is: <<

( ) eating fish that has not been sufficiently cooked;

( ) there is no correct answer;

(x) drinking contaminated water;

( ) eating raw beef;

( ) eating raw pork.

>>Anything is used to treat ankylostomiasis EXCEPT: <<

( ) pyrantel;

( ) albendazole;

(x) piperazine;

( ) mebendazole;

( ) levomisole.

>>What group of pathogens does the causative agent of clonorchiasis belong to?

( ) rickettsia;

( ) are the simplest;

(x) helminths;

( ) chlamydia;

( ) mycoplasma.

>>What is the mechanism of transmission of ankylostomiasis?

( ) air-droplet;

( ) faecal-oral;

( ) transmission;

( ) percutaneous;

(x) C and D are true.

>>To which class of helminths does the causative agent of paragonimosis belong: <<

( ) cestodoses;

( ) nematodes;

(x) trematodes.

>>The final host of the paragonimosis pathogen can be all of the following EXCEPT: <<

( ) person;

(x) cattle;

( ) cats;

( ) dogs;

( ) pigs.

>>What complications can paragonimosis lead to, EXCEPT: <<

( ) pulmonary bleeding;

(x) mechanical jaundice;

( ) pneumothorax;

( ) encephalitis;

( ) meningoencephalitis.

>>Which of the parasites is on the verge of elimination (on the globe): <<

( ) clonorchiasis;

( ) dirofilariasis;

(x) dracunculosis;

( ) trichinosis;

( ) all of the above.

>>All clinical manifestations are characteristic of strongyloidiasis, EXCEPT: <<

( ) functional disorders of the nervous system;

( ) loose stools, sometimes with mucus and blood;

( ) cough, shortness of breath, blood in the sputum;

(x) urticaria is predominantly located on the scalp and distal extremities;

( ) dyspeptic syndrome, urticaria, blood eosinophilia.

Topic 5. Onchocerciasis, loiasis, hookworm, brugiasis. Differential diagnosis of filariasis.

Learning objective: to acquire basic knowledge and skills in clinical manifestations, differential diagnosis, specific examination methods, patient management tactics and basic preventive measures for onchocerciasis, loiasis, hookworm, brugiasis, filariasis.

List of skills:

1. Follow the basic rules of working at the patient's bedside
2. Take a medical history with an assessment of epidemiological data
3. Know the forms and methods of health education work
4. Search for modern literature in the field of study
5. Summarise scientific and practical literature
6. Participate in the dissemination of medical knowledge among the population
7. Draw up a plan for laboratory and additional examination
8. Choose research methods that are adequate to achieve the goal and objectives
9. Interpret the results of different research methods
10. Use modern research methods
11. Determine the medical tactics of patient management
12. Determine the tactics of hospitalisation and isolation of patients
13. Participate in the organisation of basic preventive measures

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar operating system.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Filarial doses, mechanism of transmission, filarial helminths, source of infection, echinococcosis, Timor-Leste and Malay bruguiozoon,

tropical pulmonary eosinophilia, bruguiozoon, elephantiasis, source of infection in echinococcosis, filarial life cycle

ONCHOCERCIASIS, also known as river blindness, white water disease, and Robles' disease, is a helminthiasis that occurs as a result of infection with the helminth Onchocerca volvulus. Manifestations of the disease include severe itching, subcutaneous blisters and blindness. Onchocerciasis is the second most common infectious disease after trachoma in terms of the number of cases of blindness. The disease is classified as a subgroup of subcutaneous filariasis, a group of filariasis. The WHO classifies onchocerciasis as a neglected tropical disease. Onchocerciasis has long been known in some regions of Africa as "kru-kru" or "craw-craw". The microfilariae in the skin were discovered by Irish naval surgeon D. O'Neill when examining skin nodules in patients suffering from crowe's disease in Ghana in 1874. A few years later, in 1890, the adult worms were also discovered and identified by the famous Scottish scientist, the founder of clinical parasitology, Patrick Manson.

The role of microfilariae in skin lesions was discovered by J. Montpellier and A. Lacroix in 1920, and in river blindness by J. Hizette in the Belgian Congo in 1932. However, there is evidence that as early as 1915, Rodolfo Robles was the first to establish the link between the parasite and eye disease. The role of mosquitoes in the transmission of onchocerciasis was discovered by Scottish parasitologist B. Blacklock in Sierra Leone in the mid-1920s.

Relevance. About 17-25 million people are infected with onchocerciasis, and about 0.8 million suffer from varying degrees of vision loss. 99% of cases occur in 31 countries in sub-Saharan Africa, although onchocerciasis is also found in Yemen and parts of South America.

Etiology. Onchocerca volvulus, like other filarial helminths, has a filamentous body that becomes thinner towards the ends of the helminth. Females are 40-70 cm long and 0.13-0.4 mm wide. Males are much smaller, 2.5 cm long and 0.2 mm wide. Microfilariae are of two types: large, 0.29-0.37 mm long and 0.09 mm wide; and small, 0.15-0.29 mm long and 0.05-0.07 mm wide. Onchocerca volvulus, like most filariasis

nematodes, has an endosymbiotic relationship with bacteria of the genus Wolbachia. In the absence of Wolbachia, the development of Onchocerca volvulus microfilariae is impaired or even stopped.

Mechanism and factors of transmission. Onchocerciasis is characterised by a vector-borne transmission mechanism. Onchocerca volvulus is transmitted through the bites of midges of the genus Simulium. Midges bite mainly in the morning and evening, and usually do not enter homes. It usually takes many bites to cause infection. These midges breed in fast-flowing rivers and streams, mainly in remote villages located near fertile land where people are actively developing agriculture. Because midges are found near rivers, onchocerciasis is called river blindness. In the human body, adult worms give birth to microfilariae that migrate to the skin, eyes and other organs. When a female bites an infected person while feeding on blood, she also swallows microfilariae, which continue to develop in the midge's body and are subsequently transmitted to a healthy person during subsequent bites. The possibility of vertical transmission of onchocerciasis cannot be ruled out. Onchocerciasis has no racial differences in its course. For unknown reasons, the symptoms of onchocerciasis vary from region to region. In particular, skin lesions (onchodermatitis) are more common in forested areas, while blindness is more common in dry savannah areas. In the wet savannas, patients present with an intermediate type of clinical manifestation, i.e. both skin lesions and eye damage are noted. It is believed that up to 10 % of residents of endemic dry savannah areas affected by onchocerciasis may become blind due to the disease. No age-specific features of infection have been identified. There is evidence that as a result of immunological irritation by microfilarial antigens transmitted from an infected mother to her child, the latter later become slightly more ill than children born to healthy mothers. Immunity is not formed, and there are no known cases of self-recovery.

Pathogenesis. Pathology in onchocerciasis can be divided into two categories: the first is caused by microfilariae; the second by adults.

The most significant damage is caused by wandering microfilariae, with a load of up to 2,000 larvae/mg of skin. Microfilariae are found in all layers of the skin, but

are most concentrated in the dermal papillae. Microfilariae that parasitise human skin cause dramatic changes in the skin in the form of dermatitis. It progresses over time and leads to fibrosis, skin pigmentation, edema, dermal thickening and epidermal scarring. The skin's elastic fibres are gradually lost, resulting in wrinkled skin and sometimes ulcers. Adults that migrate through the body form nodules in the skin (onchocerciomas).

Microfilariae cause inflammation in the eyeballs, resulting in nodules on the conjunctiva, iris atrophy and other changes that cause severe visual impairment, up to blindness in one or both eyes.

A generalised inflammatory reaction of the lymph nodes occurs. General and local allergic reactions are of great importance in the pathogenesis of onchocerciasis, with the products of adult life playing a major role in their occurrence.

Clinic. According to the ICD-10, onchocerciasis is classified as a separate subclass B73 in the class of helminthiasis. Symptoms of onchocerciasis reflect the stages of parasite development and the degree of host immune response. Clinical manifestations are very diverse. Manifestations of onchocerciasis do not appear until the larvae mature into adults. On average, clinical signs appear between 9 months and 2 years after infection. The interval between infection and the onset of the first symptoms is sometimes referred to as the prelactic period or phase. Once the adults have fully formed, they cause inflammation in the skin around them in the form of subcutaneous nodules (onchocerciomas).

Skin lesions. Generalised itching can occur early in the disease and can be very severe. The following appear: papular rash (known as onchodermatitis), vesicles and pustules. Scratching due to itching can lead to the development of ulcers and secondary infection. Initially, the rash may be short-lived, but chronicity over several years can lead to lichenification, loss of skin elasticity, atrophy and/or pigmentation, skin scarring, and in some cases, ichthyosis. Due to the loss of elasticity of certain areas of the skin, it may sag. Focal symmetrical depigmentation of the skin on the lower legs creates a characteristic picture of "leopard skin". A clinical feature of onchocerciasis in Yemen is the predominantly single leg lesion (called "sowda"). Lymphadenitis is not

uncommon. Onchocerciomas are painless mobile subcutaneous nodules from several millimetres to several centimetres in diameter. They become visible 3-4 months after infection. Onchocerciomas are most often formed over bony prominences on the trunk and thighs (in the clinical course of onchocerciasis in Africa), or the head and shoulders (in South America). This is explained by the species specificity of midges, which have evolutionarily acquired differences in the nature of feeding and the place of bite attraction.

Eye lesions. Itchy eyes, redness or photophobia are early symptoms of ocular onchocerciasis. Over the years, corneal damage (keratitis) occurs as a result of an inflammatory reaction to the dead microfilariae, causing clouding and scarring of the cornea, which leads to loss of vision and complete blindness. Acute optic neuritis is less common, but it can also lead to blindness. It can also lead to iridocyclitis, glaucoma, and choroiditis.

Diagnosis. Traditionally, the diagnosis of onchocerciasis is confirmed by the detection of microfilariae under light microscopy in skin samples taken by biopsy. This method has a high specificity (100%) but low sensitivity (20%-50%) in the early stages of the disease. The diagnosis can also be made by direct microscopy of surgical specimens obtained during surgical removal of onchocerciomas. Microscopic examination of the excised onchocercioma makes it possible to detect characteristic filarial helminths with a cross-section, with eosinophils and lymphocytes in the periphery of the nodule.

Serological tests. The detection of antibodies does not establish whether they are the result of an acute illness or its chronic course.

The detection of antibodies against the antigen - Ov16 has a high sensitivity (approximately 80%) and specificity (approximately 85%). It can be used successfully at the beginning of the disease, when the results of skin microscopy are still negative. Capillary blood samples are collected after a finger puncture.

The immunochromatographic test is used to detect the presence of immunoglobulin G4 (IgG4) to recombinant Ov16 antigen.

Detection of antibodies to recombinant hybrid proteins (OvH2 and OvH3). This test is based on hybrid proteins from two separate proteins of Onchocerca volvulus (Ov20 and Ov33). The reaction is highly sensitive (> 95%) and specific (> 95%). It is performed by enzyme-linked immunosorbent assay (ELISA).

Detection of antibodies based on the use of a cocktail of 3 antigens (Ov7, Ov11, Ov16) in an ELISA. The sensitivity of the method reaches approximately 97 %, specificity - 100 %, which is better than in the test for detection of antibodies against the Ov16 antigen.

Antigen detection

Detection of the Oncho-27 antigen. The advantage of this test is that urine or tears are taken for testing. The sensitivity and specificity of the method reaches 100%.

Provocative test with diethylcarbamazine. Topical application of diethylcarbamazine in a cream base causes localised skin reactions (itching, papular rash, skin edema) in response to the death of microfilariae in the skin under the influence of the drug. The test has a varying degree of sensitivity (30%-92%). Today, the test is rarely used because of the possibility of serious skin reactions, which may require intensive care and hospitalisation. False-positive reactions can occur in patients with other filariasis diseases, such as loiasis.

Treatment. Since most of the pathogenesis of onchocerciasis is secondary to the circulation of microfilariae, the aim of therapy is to eliminate the circulation of the pathogen to reduce the manifestations, prevent the progression of eye lesions, and interrupt transmission. To date, there are no drugs known to be 100% effective against adult Onchocerca volvulus.

Ivermectin is considered to be the drug of choice, the main antimicrofilarial agent for onchocerciasis. Multiple courses of therapy reaching a dose of 150 mg/kg body weight with intervals of 3-12 months are recommended for at least 10-12 years. The drug is prescribed at shorter intervals for patients with frequent relapses. Ivermectin is generally well tolerated by patients. However, massive death of microfilariae can lead to rather severe skin reactions, itching, and increased lymphadenopathy. An increase in ocular lesions may also develop when using

Ivermectin. For this purpose, it is recommended to use a short course of prednisolone (2-3 g per course) together with ivermectin. Prescribing ivermectin every 3 months instead of every 12 months may lead to a reduction in inflammatory complications, as it prevents the number of microfilariae from increasing, thus reducing the number of microfilariae that die after treatment. Before prescribing ivermectin, the presence of concomitant loiasis should be excluded, as the administration of this drug may lead to a worsening of the toxic encephalopathy inherent in loiasis. Thus, by prescribing ivermectin, it is possible to kill microfilariae, but not adult parasites.

Studies of doxycycline therapy (100-200 mg/day for 6 weeks) have shown great promise. Doxycycline interrupts microfilarial embryogenesis, dramatically reducing or eliminating microfilariae within 18 months after treatment. Unlike ivermectin, the drug has moderate activity against adult Onchocerca volvulus, reducing their number by approximately 60%. The combination of doxycycline and ivermectin seems to be generally more effective than either drug alone. However, doxycycline has side effects and must be taken daily by the patient, limiting its usefulness for large-scale treatment programmes.

Moxidectin is a new antiparasitic drug currently being studied by WHO for use in the treatment of onchocerciasis. Moxidectin is related to ivermectin and causes a more sustained reduction in the number of microfilariae.

Surgical removal of onchocerci does not eliminate all adult Onchocerca volvulus.

Prevention. There is no vaccine against onchocerciasis. The disease can be prevented by preventing midges from biting. For this purpose, repellents and appropriate light-coloured clothing are used. In some areas, another way to prevent the disease is to reduce the midges population by spraying insecticides. Population-based prevention strategies in endemic areas of Africa and South America are based on the control of midges and the use of regular (every 6-12 months) mass treatment of patients with ivermectin.

LOAOS is a nematode filarial infection caused by Loa loa. Loaosis is restricted to the tropical rainforest belt in western and central Africa. Humans are the only definitive host of this parasite. The Loa loa microfilariae are transmitted by the blood-sucking of ticks (Chrysops [deer fly or tick]).

Microfilariae develop in adult worms in the subcutaneous tissues of humans; females reach a length of 40-70 mm, males 30-34 mm. Adults produce microfilariae. Adult worms migrate into subcutaneous tissues and under the conjunctiva, and microfilariae circulate in the bloodstream. The ticks become infected when they feed on the blood of an infected person during the day (the period of highest microfilarial infection).

Loa loa (eye worm) life cycle:

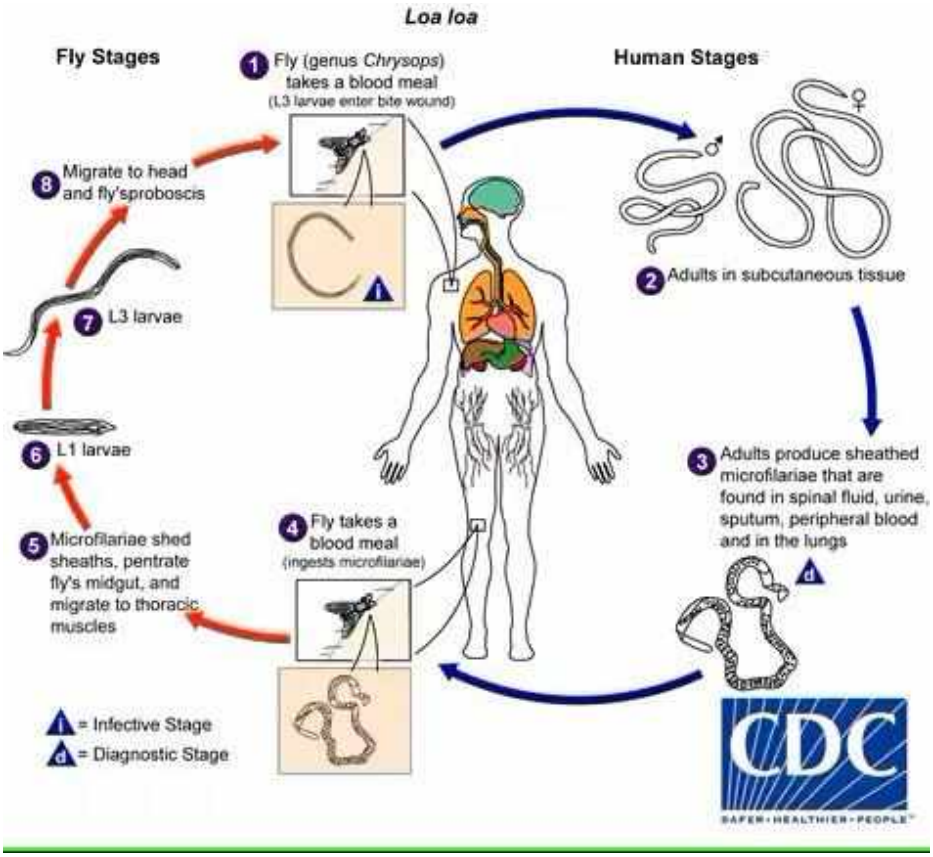

IMAGE COURTESY OF THE CENTERS FOR DISEASE CONTROL AND PREVENTION, WHICH FIGHTS PARASITIC DISEASES AND MALARIA WORLDWIDE.

Symptoms and signs of loaosis. Most people infected are asymptomatic. The infection can cause angioedema (Calabar swelling), which can develop anywhere on the body, mainly on the extremities; it is thought to be a hypersensitivity reaction to

allergens released by migrating adults. In local residents, edema usually lasts from 1 to 3 days, but is more common and severe in newcomers. Sometimes cardiomyopathy, nephropathy or encephalitis develop. Eosinophilia is common, but it is nonspecific. Nephropathy is usually manifested as proteinuria, moderate haematuria is possible, which is associated with the deposition of immune complexes on the basement membrane of the glomeruli. Sometimes moderate encephalopathy develops.

Diagnosis of loaosis

1. Visualisation of an adult worm crossing the eye in the subconjunctival space.
2. Identification of adult worms removed from the eye or skin
3. Identification and quantification of microfilariae in blood by microscopic examination or quantitative polymerase chain reaction (PCR)

Loiasis should be suspected in immigrants or tourists who have been exposed to endemic areas and present with eye worms, calabar edema, or unexplained peripheral eosinophilia. Sometimes the diagnosis of loiasis is confirmed by observation of the adult worm moving under the conjunctiva or by identification after removal from the eyes or skin.

The microscopic detection of microfilariae in peripheral blood confirms the diagnosis; the number of microfilariae per millilitre of blood should be quantified. Blood samples should be taken between 10 and 14 am, when microfilarial levels are highest.

Most serological antibody tests do not distinguish Loa loa from other diseases caused by filarial nematodes. Loa-specific antibody tests have been developed but are not widely available. In addition, a positive serological test does not distinguish between previous and current infection. A quantitative real-time PCR (RT-PCR) test to confirm the diagnosis and determine microfilarial load is available from the Parasitic Diseases Laboratory, National Institutes of Health.

People from endemic areas of Africa should be tested for loafer before starting treatment with diethylcarbamazine or ivermectin for other disorders, as these medicines can have significant adverse effects when used in people with loafer. If diethylcarbamazine or ivermectin is given to patients with a Loa loa microfilariae count

≥ 8000 per ml of blood, they are at risk of potentially fatal encephalopathy caused by the release of antigens from the dead microfilariae.

Treatment of loiasis. Loiasis is difficult to treat. Diethyl carbamazine is the only drug that kills the microfilariae and adult worms. In the United States of America, it is only available from the Centers for Disease Control and Prevention (CDC) after laboratory confirmation of loiasis; clinicians should seek expert advice before initiating treatment; the following should be done before starting diethylcarmabazine treatment

1. It is necessary to measure the number of microfilariae in the blood, as the use of diethylcarbamazine for the treatment of severe infection (≥ 8000 microfilariae/ml of blood) is associated with a risk of fatal encephalopathy.
2. Coinfection with onchocerciasis should be excluded, as diethylcarbamazine can cause severe hypersensitivity reactions and worsen eye and skin disease in patients with this infection.

Clinicians should get expert help in measuring the number of microfilariae and thus determining the severity of the infection.

Treatment of mild infection: patients with symptoms of loiasis and < 8000 microfilariae per ml of blood are prescribed diethylcarbamazine at a dose of 2.7 to 3.3 mg/kg 3 times daily for 21 days.

Treatment of severe infection: In patients with severe infection, filarial antigens (released by filarial cells when they die from diethylcarbamazine) can cause encephalopathy, leading to coma and death. Patients with a microfilarial count ≥ 8000 per ml of blood are at risk of this effect, and initial treatment with albendazole 200 mg orally twice daily for 21 days or plasmapheresis is recommended. The aim of this treatment is to reduce the microfilarial count to < 8000/ml per ml at the start of diethylcarbamazine treatment. Several courses of diethylcarbamazine may be necessary. For patients who have failed ≥ 2 courses of diethylcarbamazine treatment, albendazole 200 mg orally twice daily for 21 days may be prescribed.

Ivermectin is also used to reduce microfilariaemia, but albendazole is preferred because its onset is slow and the risk of encephalopathy is lower.

Prevention. Diethylcarbamazine (DEC) 300 mg orally once a week can be used to prevent loiasis in people travelling for long periods of time to endemic areas. Using insect repellent (including permethrin to impregnate clothing) and wearing long, long-sleeved clothing that covers the legs can reduce the number of bites. As the flies bite during the day, mosquito nets (for bed) are not relevant.

WUHERERIOSIS is found in tropical and subtropical areas of Africa, Asia, the Pacific, North and South America, including Haiti. Brugiasis is endemic in southern and southeastern Asia.

About 51 million people were infected as of 2018, and 40 million were disfigured by the disease. In 2000, the WHO launched its own Global Programme to Eliminate Lymphatic Filariasis. As a result, significant progress has been made in stopping the spread of the infection through large-scale annual treatment of people who are eligible. In 2020, more than 860 million people lived in areas where the infection was prevalent enough to require such annual treatment.

Lymphatic filariasis is an infection with any of the 3 species of Filarioidea. Lymphatic filariasis is transmitted by mosquitoes; the invasive larvae migrate to the lymphatic vessels where they develop into adult worms.

Adult worms inside the lymphatic vessels can cause inflammation, resulting in acute adenolymphangitis, or epididymitis, or chronic lymphatic obstruction, which in some patients leads to elephantiasis or hydrocytosis.

The symptoms of the acute period of the disease include fever, lymphadenitis, lymphangitis, epididymitis and funiculitis (inflammation of the spermatic cord). *The chronic process* is manifested by abscesses, hyperkeratosis, polyarthritis, hydrocele, lymphodema and elephantiasis. Another manifestation of the infection is tropical pulmonary eosinophilia with bronchospasm, fever and pulmonary infiltrates. The diagnosis is confirmed by the detection of microfilariae in blood or lymphatic tissue biopsy samples, ultrasound imaging of adult worms in lymphatic vessels, or serological testing. Treatment is performed with diethylcarbamazine. In case of bacterial cellulitis, antibiotics are used.

Lymphatic filariasis is caused by Wuchereria bancrofti (about 90% of cases), Brugia malayi or B. timori. It is transmitted by mosquitoes. The infective larvae migrate from the mosquito to the lymphatic vessels, where they develop into threadlike adult worms in 6-12 months. Females are 80-100 mm long; males are 40 mm long. Fertilised females produce microfilariae that circulate in the bloodstream.

Clinical manifestations. The disease may be accompanied by microfilariaemia without obvious clinical manifestations. Symptoms and signs of the disease are primarily associated with adult worms. Microfilariaemia gradually disappears after people leave the endemic area.

Acute inflammatory filariasis consists of recurrent episodes of fever lasting 4-7 days with lymph node inflammation with lymphangitis (acute adenolymphangitis) or acute epididymitis and inflammation of the spermatic cord; secondary bacterial infections are common. Limited involvement of the limb may cause an abscess that drains outward and leaves a scar. Episodes of acute adenolymphangitis usually precede the onset of the chronic stage of the disease, which occurs after ≥2 decades. Acute filariasis is more severe in newcomers to endemic regions compared to local residents.

Chronic filariasis develops slowly and takes many years to manifest. Most patients have asymptomatic lymphatic vasodilation, but chronic inflammation caused by adult worms and secondary bacterial infection can lead to chronic lymphedema in the affected area. Increased local susceptibility to bacterial and fungal infections further contributes to the progression of the disease. Chronic lymphedema of the lower extremity can progress to elephantiasis (chronic lymphatic obstruction). W. bancrofti can cause hydrocele and elephantiasis of the scrotum. Other forms of chronic filariasis are caused by destruction of lymphatic vessels or aberrant drainage of lymph fluid, leading to chyluria and lymphatic tumours.

Additional lymphatic symptoms include chronic microscopic haematuria and proteinuria and moderate polyarthritis, which are caused by the action of immune complexes.

Tropical pulmonary eosinophilia is a rare manifestation and is characterised by recurrent bronchospasm, transient pulmonary infiltrates, low-grade fever and severe

eosinophilia. It is caused by allergic reactions to microfilariae. Chronic lung damage can lead to pulmonary fibrosis.

Diagnosis is based on the microscopic detection of microfilariae in filtered or centrifuged blood concentrates, which should be obtained at the time when the level of microfilarial infection is highest (depending on the species).

Tests for antigens, antibodies and DNA of the parasite are an alternative to microscopic diagnosis.

Microscopic examination of blood samples or lymphatic tissue biopsy.

W. bancrofti antigen test (available internationally).

Antibody tests

The detection of microfilariae in the blood by microscopic examination confirms the diagnosis of lymphatic filariasis. Methods of blood enrichment, filtration, centrifugation are more sensitive than thick blood smears. Blood samples should be obtained at the time when the level of microfilarial infection is highest - at night in endemic areas, during the day on many islands of the sea. Viable adult worms can be seen in dilated lymphatic vessels on ultrasound; their movement is called the filarial dance.

Several types of blood tests are available:

1. Detection of antibodies: Enzyme-linked immunosorbent assays for antibodies to filarial IgG1 and IgG4
2. Antigen detection: immunochromatographic rapid test for W. Bancrofti antigens.

Patients with active filarial infection usually have elevated levels of IgG4 antibodies to filaria. However, there is considerable cross-sensitivity between filaria and other helminths, and a positive serological test result does not distinguish between past and present filarial infection.

Rapid test for the detection of W. bancrofti antigen is used worldwide in filariasis elimination programmes

Polymerase chain reaction (PCR) tests for W. bancrofti and B. malayi are available from research laboratories. Adults of both species can be identified in lymphatic tissue biopsies.

Treatment is diethylcarbamazine (DEC).

Diethylcarbamazine kills microfilariae and a few adult worms. Before starting treatment with diethylcarbamazine, patients are screened for co-infections caused by Loa loa and Onchocerca volvulus, as diethylcarbamazine can cause serious adverse reactions in patients with these infections.

Treatment of acute lymphatic filariasis.

Traditionally, DEC has been administered at 2 mg/kg orally 3 times daily for 12 days; alternatively, 6 mg/kg orally once daily is used.

Side effects of diethylcarbamazine are generally limited and depend on the number of microfilariae in the blood. The most common side effects are dizziness, nausea, fever, headache, muscle or joint pain, which are thought to be related to the release of filarial antigens.

Patients should be screened for co-infections with Loa loa or Onchocerca volvulus before starting diethylcarbamazine treatment, as diethylcarbamazine may cause serious adverse reactions in patients with these infections. A single dose of albendazole 400 mg orally plus ivermectin (200 μg/kg orally) may be used in areas where onchocerciasis is coexisting, but ivermectin alone does not kill the adult worms responsible for lymphatic filariasis. Diethylcarbamazine should not be used in patients with high levels of Loa loa microfilariae in the circulating blood because of the risk of life-threatening side effects, including encephalopathy.

A number of drug combinations and regimens are used in mass treatment programmes.

Doxycycline was also used for a long time (e.g., 100 mg orally twice daily for 4-6 weeks). Doxycycline kills the Wolbachia endosymbiont bacteria that live in the parasite's intestines, which leads to the death of adult filarial worms. It can be used in combination with diethylcarbamazine or alone.

Attacks of acute adenolymphangitis usually resolve spontaneously, but antibiotics may be required to prevent secondary bacterial infections.

Treatment of chronic lymphedema.

Chronic lymphedema requires careful skin care, including the use of systemic antibiotics to treat secondary bacterial infections; these antibiotics can slow or prevent the progression of lymphedema. Conservative measures, such as elastic bandaging of the affected limb, shrink the tumour. Surgical decompression using nodal venous shunts to improve lymphatic drainage provides some long-term relief in extreme cases of elephantiasis. Large hydrocele can be treated surgically, but the disease often recurs.

Treatment of tropical pulmonary eosinophilia

Tropical pulmonary eosinophilia responds to diethylcarbamazine at a dose of 2 mg/kg orally 3 times daily for 14-21 days, but relapses occur in more than 25% of patients and require additional courses of therapy.

Prevention. Avoiding mosquito bites in endemic areas is the best protection for travellers (e.g. by applying diethyltoluamide [DEET] to exposed skin, permethrin-impregnated clothing, and bed nets).

In 2000, the WHO launched the Global Programme to Eliminate Lymphatic Filariasis to map endemic areas and treat entire at-risk populations with the following mass drug administration regimens:

1. Albendazole (400 mg) as monotherapy twice a year for the regions;

2. Ivermectin (200 μg/kg) and albendazole (400 mg) annually in onchocerciasis areas, and in some cases diethylcarbamazine (DEC) (6 mg/kg) and albendazole (400 mg) plus Ivermectin (200 μg/kg) in areas without onchocerciasis or loiasis. These treatment regimens result in a reduction in microfilarial infection and thus reduced mosquito transmission.

Presentation:

https://docs.google.com/presentation/d/1SrvvA1klEVI0cMZTOm6WqkH9F40mZwli/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for self-monitoring

1. Etiology of filariasis (onchocerciasis, loiasis, hookworm, brugiasis).
2. Epidemiology of filariasis (onchocerciasis, loiasis, hookworm, brugia

3. Pathogenesis of filariasis (onchocerciasis, loiasis, hookworm, brugia
4. Clinical course of filariasis (onchocerciasis, loiasis, hookworm, brugiasis).
5. Laboratory diagnostics of filariasis (onchocerciasis, loiasis, hookworm, brugiasis).
6. Differential diagnosis of filariasis (onchocerciasis, loiasis, hookworm, brugiasis).
7. Complications of filariasis (onchocerciasis, loiasis, hookworm, brugiasis).
8. Treatment of filariasis (hookworm, onchocerciasis, loiasis, dracunculiasis).
9. Prevention of filariasis (hookworm, onchocerciasis, loiasis, dracunculiasis).
10. The procedure for hospitalisation, rules for discharge of patients with filariasis (hookworm, onchocerciasis, loiasis, dracunculiasis).

Glossary

Filariasis is a group of helminth infections caused by filariae (threadworms) of the family Onchocercidae, order Filariidae, type Nemathelminthes. About 110 species of filaria are known, but only 8 of them are capable of causing infections in humans.

Brugiasis (filariasis Malayan and Timorian) is a combined name for two helminth infections that belong to the clinical subgroup of lymphatic filariasis, a group of filariasis. This infection is caused by very close to each other, but still separate species of Brugia malayi and Brugia timori, which is why brugiasis malayan and timorian are often distinguished, but still, due to very similar clinical signs, these diseases are often combined for consideration in one common section.

Elephantiasis, or elephantiasis, or elephantiasis is a chronic thickening of the skin and subcutaneous fatty tissue, accompanied by severe lymphatic stasis. It is a manifestation of various diseases where parts of the human body swell so much that they change the proportions of the body. Causes of elephantiasis: repeated or chronic inflammation of the skin and subcutaneous tissue: erysipelas, nonspecific lymphadenitis; cancer metastases to regional lymph nodes; filariasis, in particular lymphatic filariasis; inguinal granuloma or venereal granuloma, which leads to scrotal elephantiasis; congenital malformation of the lymphatic system, which consists in obstruction of lymphatic vessels or impaired lymphatic circulation;podoconiosis - the

appearance of elephantiasis due to immune damage to the lymphatic system; Wiedemann syndrome, or Proteus syndrome, or elephant man syndrome - a rare congenital disease that occurs with significant growths of the skin, bones, and swelling of the body.

The filarial life cycle - like all nematodes - consists of 5 stages of larval development in the vertebrate (primary) host and in the vector and intermediate host, the arthropod. Adult females give birth to thousands of first-stage larvae, or microfilariae, which enter the insect's body. The circulation of microfilariae in the bloodstream is called "microfilariaemia". Some microfilariae have a unique daily circadian rhythm of peripheral circulation. Mosquitoes and flies also have a circadian rhythm of feeding on animal blood. The highest concentration of microfilariae in the blood of the primary host usually occurs when the insects are actively feeding.

Test control of knowledge

>>Onchocerciasis is transmitted by repeated bites of infected persons

( ) ticks

( ) mosquitoes

( ) lice

(x) midges

( ) horseflies

>>In which helminthic disease allergic dermatitis is combined with damage to the organ of vision and ends in blindness in case of prolonged course <<

(x) onchocerciasis

( ) loaose

( ) wucheriosis

( ) bruchiosis

( ) dirofilariasis

>>The elephantine genitals are formed at <<

(x) wucheriosis

( ) loaose

( ) onchocerciasis

( ) brugiasis

( ) dirofilariasis

>>The drug of choice for the treatment of echinococcosis is: <<

( ) phenasal

( ) vermox

( ) male fern extract

(x) ditrazine

( ) that's right

>>The main method of diagnosing bruchiosis <<

( ) PCR

( ) coprogramme

( ) examination of a scraping from the perianal folds

( ) detection of eggs in the contents of the duodenum obtained during probing

(x) blood microscopy

>> Chemoprophylaxis of bruchiosis is carried out: <<

( ) phenasal

( ) vermox

(x) ditrazine

( ) with male fern extract

( ) albendazole

>>Loaoz is transferred <<

( ) ticks

( ) mosquitoes

( ) lice

(x) by damselflies

( ) midges

>>Which helminth is characterized by a transmissible mechanism of transmission: <<

( ) clonorchiasis

( ) diphyllobothriosis

( ) hymenolepidosis

( ) paragonimosis

(x) dirofilariasis

>>Which helminth is characterised by soft tissue swelling, genital lesions and conjunctivitis: <<

(x) loafer

( ) wuhereriosis

( ) onchocerciasis

( ) bruchiosis

( ) dirofilariasis

>> Infection with dirofilariasis occurs when bitten: <<

(x) mosquitoes

( ) ticks

( ) damselflies

( ) lice

( ) midges

Topic 6. Differential diagnosis of schistosomiasis.

Learning objectives: to acquire basic knowledge and skills on the topic under study and to be able to apply the acquired knowledge in practice.

Abstract. Schistosomiasis is a group of tropical helminthiasis, severe chronic diseases that occur with damage to the digestive or genitourinary system. The theoretical materials of the module introduce the basic concepts, terms and definitions of schistosomiasis.

List of skills:

1. Know how to take anamnesis;
2. Conduct an objective examination of the patient;
3. Justify the diagnosis;
4. Prepare medical documentation;
5. Evaluate the results of laboratory and instrumental research methods;
6. To master manipulations: intravenous injection of drugs, sampling of materials (blood, faeces, urine) for research;
7. Evaluate the results of bacteriological and serological methods of research;
8. Provide assistance in case of emergency.

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Schistosomiasis, natural focal point, definitive host, intermediate host, myracidium, sporocystis, cercaria, liver failure, observation, quarantine.

SCHISTOSOMIASIS is a group of tropical helminth infections, severe chronic diseases that affect the digestive or urogenital system.

The causative agents of schistosomiasis belong to the class of flukes. Among the many species of schistosomes that infect animals, five are of medical importance:

1. Schistosoma haematobium (the causative agent of genitourinary schistosomiasis),
2. S. mansoni (intestinal schistosomiasis pathogen),
3. S. japonicum (the causative agent of Japanese schistosomiasis),
4. S. mekongi (causative agent of Mekong schistosomiasis),
5. S. intercalatum (causative agent of intercalated schistosomiasis).

The schistosomes that infect humans are bisexual. The length of females ranges from 7 to 26 mm, width - 0.17-0.3 mm, males - from 4 to 20 mm, with a width of 0.5-1.2 mm. On the abdominal surface of the male there is a groove - a gyneciform canal, inside which there is a threadlike female. Depending on the morphological characteristics of the eggs, three groups of schistosomes are distinguished:

a) eggs with a lateral spine (S. mansoni);

b) eggs with a terminal spike (S. haematobium, S. intercalatum),

c) eggs in the form of a truncated oval with a rudimentary spine (S. japonicum, S. mekongi).

Life cycle. The development of isolated eggs occurs in water at a temperature of 10-30°C and sufficient insolation. Miracidia leave the eggshells and actively swim due to the presence of cilia. Possessing positive chemotaxis to the tissues of the intermediate host, a mollusk, they attach to the surface of its body and gradually penetrate it. In mollusks, the development and reproduction of larval forms of schistosomes occurs, which ends with the formation of cercariae - larvae with a tail. From 1-2 to several thousand cercariae can be released daily, which are invasive to humans. The cercariae are equipped with an elongated forked tail, which helps them to move actively in the water. At the head end of the larva there are oral and abdominal suckers, and glands secreting a secretion that helps cercariae to attach to and penetrate the body of the final host. Cercariae penetrate the human body most actively in the first hours after leaving the mollusk; after 8-12 hours, glycogen reserves are depleted and their activity drops sharply.

The average lifespan of adult parasites in the body of their final hosts is 3 to 8 years. Individuals can live up to 30 years. After completion of sexual maturation and mating, adults migrate to the veins of the bladder (S. haematobium) or mesenteric veins (S. mansoni, S. intercalatum, S. japonicum, S. mekongi), where they begin to produce eggs. The prelative period in schistosomiasis is 30-40 days. In some cases, eggs are detected in faeces or urine at 4-6 months of infection. Each pair of worms produces from 200 to 3500 eggs per day. S. japonicum and S. mekongi have the highest reproductive capacity.

Epidemiology. The group range of schistosomiasis extends along the equator between 38°N and 35°S. Schistosomiasis has been reported in 73 countries. The ranges of different forms of schistosomiasis are not the same and tend to expand continuously, which is associated with the continuous migration of the population and the creation of irrigation systems and artificial reservoirs. Schistosomiasis is second only to malaria in terms of its socio-economic importance for public health. Intestinal schistosomiasis is widespread in most countries of Africa and South America, in Yemen and some Caribbean islands; Japanese schistosomiasis - in the Philippines, Japan, China; Mekong schistosomiasis - in Kampuchea, Laos. A limited area of intercalated schistosomiasis has been reported in Gabon and Cameroon. Genitourinary schistosomiasis is widespread in most African countries in the Eastern Mediterranean region and in the European region (Morocco, Turkey).

The source of infection for genitourinary schistosomiasis is humans, and for intestinal schistosomiasis - humans, some rodents, and cattle. In addition to humans, Japanese schistosomiasis affects many species of domestic (pigs, dogs, cats, sheep, horses) and wild animals (foxes, various types of rodents). In most cases, freshwater mollusks are intermediate hosts of schistosomes. The most favourable for molluscs are stagnant or low-flowing water bodies with clean bottoms, well-developed vegetation and moderate shading, as well as reservoirs, primitively constructed or irrationally exploited irrigation canals, rice fields, etc. Mollusks usually live in fresh water, but some species (Oncomelania) can live in slightly salty water. The temperature optimum is 18-35°C. The intensity of schistosomiasis transmission is determined by the density

of the mollusc population, the rate of their reproduction and extinction, the age structure of the population and the frequency of human contact with their habitats. High infection of molluscs is facilitated by the continuous faecal contamination of water bodies by infected people.

People are usually infected by swimming, washing clothes, and working in irrigated fields. Infection with S. japonicum can occur through contact with grass on which infected semi-terrestrial molluscs have crawled. The susceptibility of humans to schistosome infection is general, with the most affected populations being those who are more likely to come into contact with mollusc habitats. In endemic areas, the highest rates of infection are observed in the age group of 10-20 years. Muslim men are usually affected much more often than women, whose contact with open water is less intense. Agricultural workers and personnel operating irrigation systems are at increased risk of infection. Schistosomiasis is most often imported to the CIS countries by residents of countries endemic for these helminths.

Human infection occurs through the skin or mucous membranes when in contact with or drinking water from open water bodies. The cercariae penetrate the stratum corneum and the mucous membrane, lose their tail appendage and develop into a hexosomula. Then the hexosomes penetrate the peripheral lymphatic or venous vessels, enter the right heart and lungs, and then move quickly to the liver vessels, where they develop, form adults, mature, and mate. The parasites migrate from the lungs to the portal vein system with the blood flow directly through the diaphragm to the liver. Female helminths lay their eggs in the lumen of venous vessels. Due to the action of a cytolytic enzyme secreted by the egg embryo, they penetrate the vessel wall and bladder or intestinal tissue, enter the bladder or intestinal lumen and are excreted in the urine or faeces of infected individuals into the environment. However, a large number of parasite eggs are not excreted from the human body, but are retained in various tissues. Most often, this process occurs in the bladder wall (in genitourinary schistosomiasis) or in the intestinal wall or liver. This feature of schistosome eggs is associated with the development of many complications of schistosome infections.

In the development of the pathological process in schistosomiasis, acute (migration) and chronic stages are distinguished. The acute stage of schistosomiasis is based on the sensitising effect of metabolic products of the larval stage and the traumatic effect of schistosomes during migration. During the period of cercariae penetration into the skin, a large number of passages are formed that facilitate the penetration of pathogenic microflora into the tissues, and micro haemorrhages appear. The products of schistosomal metabolism and decay cause the formation of eosinophilic inflammation. Schistosomal migration is accompanied by focal lesions of the pulmonary capillaries, manifested by edema, haemorrhage, inflammatory changes in the bronchioles, and sometimes the development of interstitial pneumonia. Inflammatory changes also develop in the liver. Parasitisation of adults in the vascular system is not accompanied by pronounced changes in the vascular wall. The main reason for the development of the pathological process in schistosomiasis is infiltration of various tissues by eggs. Eggs that do not find a way out through the bladder or intestines are carried by the bloodstream to various organs and tissues, most often to the liver and lungs. Inflammatory and allergic granulomas form around the eggs. The metabolic products of the embryos cause proliferation of the intima and connective tissue, which leads to the development of widespread obliterating endarteritis of the lung and liver vessels. The proliferation of fibrous tissue around the branches of the portal vein (tubular indurative fibrosis) leads to difficulty in blood flow, development of portal hypertension, accompanied by a significant enlargement of the liver and splenic compaction. Death of patients with such disorders often occurs as a result of bleeding from varicose veins of the esophagus. Obliterating endarteritis of the pulmonary vessels contributes to an increase in pressure in the small circle of blood circulation and the development of pulmonary heart.

Clinic. According to the nature of organ damage, there are genitourinary schistosomiasis and intestinal schistosomiasis. Clinical manifestations of the acute stage of schistosomiasis are similar. At the sites of cercariae penetration, cercariae dermatitis develops, more often in non-native populations. There is a tingling sensation, itching, local erythema develops, and papular rashes appear. The severity of

dermatitis depends on the number of cercariae penetrating the skin, the patient's age, the body's reactivity, and the frequency of infection (primary, repeated, etc.). After 5-6 days, the manifestations of dermatitis may regress and complaints of weakness, fatigue, and fever appear. During the period of schistosomal migration, their stay in the lungs, a cough appears, accompanied by the discharge of thick sputum or haemoptysis. Occasionally, obstructive bronchitis, myalgia, arthralgia, hepatosplenomegaly, and lymphadenopathy develop. The duration of the acute stage is 7-14 days, less often 5-6 weeks. During this period, blood tests reveal leukocytosis, eosinophilia, and an increased ESR. The process moves to the chronic stage when the parasite completes its development and egg production begins. From this period on, each species form of schistosomiasis proceeds differently. This is due to differences in the localisation of adults and the ways in which eggs migrate from the blood vessels to the environment.

The chronic stage of intestinal schistosomiasis caused by S. mansoni parasitisation is characterised by proliferative processes in the intestine and liver. Abdominal pain of indeterminate location and varying intensity occurs, stool becomes more frequent, tenesmus appears, and loss of appetite and weight is observed. Blood and mucus impurities appear in the faeces. With intensive invasion and progression of the disease, complications arise: mesenteric fibrosis, haemorrhoids, rectal prolapse, polyposis, and intestinal ulcers. Polyposis and fibrosis can in some cases lead to intestinal obstruction. Fragments of dead helminths and their eggs are introduced into the liver, which contributes to the development of necrotic and granulomatous changes. In the portal areas of the liver around the eggs, granulomas, chronic inflammation and severe fibrotic changes, in particular, tubular-indurative fibrosis, develop. The schistosomal nature of the process in the liver can be suspected in patients with an enlarged (due to the left lobe) dense liver, ascites, young age of patients, absence of jaundice, eosinophilia in the peripheral blood, and a sharp increase in the spleen.

In severe and prolonged infections, chronic membranous proliferative glomerulonephritis is detected. Circulating immune complexes of IgG and IgM with schistosome antigens are detected in the basement membranes. With the development

of portacavitary collaterals, schistosome eggs quickly reach the lungs, vascular obliteration develops and cardiopulmonary disorders appear.

In some cases, pseudotumours are formed in the descending colon, sigmoid colon and omentum, consisting of a large number of calcified schistosome eggs, granulomas and fibrous tissue. In some cases, schistosomiasis is asymptomatic for a long time.

Intercalated schistosomiasis is considered to be the most benign of the entire group of intestinal schistosomiases. The clinical manifestations and consequences of the disease have much in common with schistosomiasis caused by S. mansoni, but are mild and complications are very rare.

Schistosomiasis japonicum (Kataama disease) is one of the most severe forms of intestinal schistosomiasis. The severity of the course is associated with the fact that S. japonicum females produce a larger number of eggs than helminths of other species and they are laid in the form of massive clusters that stimulate a more intense tissue reaction, leading to significant intestinal damage.

Symptoms of the chronic stage appear 4-5 weeks after infection: fever, loss of appetite, weight, headaches, abdominal pain, anaemia, diarrhoea. Mucus and blood appear in the faeces, and the liver and spleen become enlarged.

The clinical course of Japanese schistosomiasis ranges from mild to fulminant forms, rapidly ending in death. This type of schistosomiasis most often develops complications: polyposis and intestinal ulcers, liver fibrosis with portal hypertension, a sharp increase in spleen and hypersplenism. When eggs are introduced into the central nervous system, paresis, paralysis, encephalitis, and meningoencephalitis develop.

Mekong schistosomiasis is more common in children under 10 years of age. The initial manifestations of the disease are similar to intestinal schistosomiasis caused by S. japonicum. In most patients, hepatomegaly is detected in the early stages. This form of schistosomiasis rapidly develops complications associated with severe portal hypertension, manifested by splenomegaly, and dilated esophageal veins. Mechanical damage to the varicose veins of the esophagus leads to bleeding, bloody vomiting, which in some cases can be fatal.

Genitourinary schistosomiasis. One of the most pathognomonic manifestations of genitourinary schistosomiasis is terminal haematuria. When the bladder walls contract, the helminth eggs pass through the mucous membrane, injuring it, so the bleeding is terminal. Permanent mechanical damage to the bladder mucosa leads to the development of an inflammatory process in the urinary system. Urination becomes more frequent and painful. Secondary inflammation causes severe destructive changes and ulcers on the mucosa. The inflammatory process can spread up to the kidneys or deep into the bladder muscle tissue. A pronounced fibroblastic reaction to schistosome eggs initiates papillomatous growths in the bladder wall, the development of neoplastic processes, fibrosis, and calcification. This is accompanied by a decrease or even cessation of egg excretion in the urine, which is not due to a decrease in the reproductive capacity of helminths, but also to difficulties in passing eggs through fibrous tissue. At this stage of the disease, complications develop, the ureters entering the bladder are deformed, and X-ray examination reveals narrowing or dilation of the ureters. Ureteral stenosis and fibrosis of the bladder neck, as well as papillomatous growths, can cause blockage of the ureters and, accordingly, ureteral dropsy and hydronephrosis. Urological changes are often detected - calcification of the bladder walls, deformation of the ureters, hydronephrosis, non-functioning kidney, and urinary calculi.

In endemic areas, bladder cancer is more common in patients affected by genitourinary schistosomiasis, histologically in the form of squamous cell carcinoma.

In addition to the urinary tract, the genitals are also involved in the pathological process. Infiltration of the urethra by eggs can lead to the development of pseudo-elephantiasis of the male genital organ. Sometimes the seminal vesicles are involved in the process, and prostatitis and epididymitis develop. In women, genital lesions are clinically manifested by the formation of papillomas of the vagina and external genitalia. Very often they bleed and this is the cause of heavy, bloody vaginal discharge. All these changes, as a rule, are formed without pronounced symptoms, so patients consult a doctor in the period of irreversible changes. In case of intensive infection, eggs or dead worms are carried into the lungs, liver and central nervous system. Diffuse

obliterating endarteritis contributes to the development of right ventricular decompensation of the heart. When eggs penetrate the brain or spinal cord, a brain tumour clinic develops, and granulomatous hepatitis develops in the liver.

Diagnosis. To establish the diagnosis of schistosomiasis, it is important to indicate the presence in an area endemic for this helminthic disease and contact with open water. For the diagnosis of infections caused by pathogens of the intestinal group of schistosomiasis, it is recommended to use the method of a thick smear of faeces with a cellophane coating (Cato method). A response can be obtained 30 minutes after the smear is prepared. It is better to take the material for the test from the faecal surface, where the largest number of eggs is located. Scraping the rectal mucosa helps to detect schistosome eggs. Rectoromanoscopy is the most widely used instrumental method. This examination reveals hyperaemia of the distal bowel, erosion, and ulcers in the early stage of the disease. In the chronic stage, papillomas, sandy spots (accumulation of schistosome eggs) are detected. Live and calcified schistosome eggs are found in biopsies of the rectal mucosa. To determine the cause of liver fibrosis, angiography and laparoscopy are performed.

Genitourinary schistosomiasis can be suspected in a patient with terminal haematuria, dysuria, eosinophilia, moderate leukaemia and an elevated ESR. To diagnose this form of schistosomiasis, a parasitological method of urine examination for the presence of eggs is used. Urine is collected during the period of the patient's greatest activity - from 10 to 14 pm. Cystoscopy is a reliable and fast method of instrumental diagnosis of genitourinary schistosomiasis. It allows you to recognise the invasion, identify complications, and monitor the dynamics of recovery after chemotherapy. X-ray examination of the urinary system and excretory urography in 100% of cases reveals a characteristic feature of chronic schistosomiasis - extremely clear contours of the urinary tract organs. This is due to the calcification of dead schistosome eggs that have infiltrated the walls of the bladder and ureters, and the development of fibrous tissue. In addition, this examination can detect strictures in the lower and dilatation in the upper ureters.

It is necessary to differentiate genitourinary schistosomiasis from bladder tuberculosis, haemorrhagic diathesis, renal neoplasms and urolithiasis of other etiologies.

It is necessary to differentiate the intestinal group of schistosomiasis from intestinal amebiasis, bacterial dysentery, balantidiasis, and liver cirrhosis.

Treatment. Two drugs are used for the treatment of schistosomiasis: praziquantel and oxamnidine. Praziquantel acts on all types of schistosomes. The drug is prescribed for adults and children:

- for infections with S. haematobium, S. mansoni, S. intercatum - 40 mg/kg per day in 2 doses on day 1.

- in case of S. japonicum, S. mekongi infections - 60 mg/kg per day in 3 doses on 1 day.

The efficacy of the drug against infections caused by S. haematobium is 80-95%. For infections caused by S.mansoni, S.intercalatum, S.japonicum, S.mekongi, the treatment effectiveness is usually more than 60%. Oxamnichine is active only in the case of infection caused by S. mansoni. The drug is prescribed for adults at a dose of 15 mg/kg in one dose; for children, 20 mg/kg per day in 2 doses - 1 day. Treatment efficacy ranges from 60 to 90%. The effectiveness of treatment is monitored after 4 months by examining faeces or urine.

Prognosis. Uncomplicated schistosomiasis can be successfully treated, even decompensated hepatoliver syndrome in schistosomiasis can be treated (50% of patients have complete regression of symptoms). In 90% of cases, a reduction in the size of polyps and, in some cases, complete regression is noted on the background of specific therapy.

Presentation:

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Glossary

Schistosomiasis is a group of tropical helminth infections that belong to the trematodes and are characterised by the predominantly genitourinary and digestive organs and a tendency to chronicity.

Natural focal point is a phenomenon when a pathogen, its specific vector and animal reservoirs of the pathogen exist in natural conditions for an unlimited period of time during the change of their generations, regardless of humans.

The final host is the organism in which the sexually mature stage of the parasite lives or reproduces sexually.

An intermediate host is an organism in which a parasite passes through an intermediate stage of its development, often associated with asexual reproduction (but not the final stage of sexual reproduction).

Miracidium is a trematode larva 0.03-0.3 mm long, its body is covered with cilia, it emerges from an egg in water, swims, then penetrates the body of an intermediate host (mollusk) and turns into a sporocyst.

The **sporocyst** is the first parthenogenetic generation in trematodes, 1-6 mm long. It develops from a larva - miracidia - in the body of an intermediate host (mollusk).

Cercaria is the last stage of a trematode larva that develops in and leaves an intermediate host (mollusk).

Quarantine is a system of anti-epidemic and regime measures aimed at complete isolation of the focus and elimination of the disease in it. Quarantine is imposed only in the event of widespread infection.

Test control of knowledge

>>Is schistosome transmission to humans possible?

( ) Transmission;

( ) When consuming poorly cooked fish;

(x) When swimming in a contaminated body of water;

( ) By airborne droplets;

( ) When swimming in the pool.

>>Indicate the place of parasitism of the schistosomiasis pathogen: <<

(x) Vessels;

( ) Small intestine;

( ) Biliary tract;

( ) Large intestine;

( ) Pancreatic duct.

>>A foreign citizen was diagnosed with intestinal schistosomiasis during an examination. Indicate the likely route of infection.

(x) When swimming in water;

( ) When eating dried fish;

( ) When using stroganoff;

( ) Because of dirty hands;

( ) For mosquito bites

>>Specify the intermediate host in the schistosome development cycle: <<

( ) Pisces;

(x) Shellfish;

( ) Ants;

( ) Cyclopes

>>Which of the following measures are relevant in the prevention of schistosomiasis?

( ) Wash your hands before eating;

( ) Use only boiled water;

( ) Do not eat raw fish;

(x) Do not swim in standing water;

( ) Eating sufficiently cooked meat

>>Indicate what material is examined in the laboratory diagnosis of urogenital schistosomiasis?

( ) Blood;

(x) Urine;

( ) Sputum;

( ) Faeces;

( ) CSF

>>A patient who came to Ukraine from Australia consulted a urologist with complaints of pain during urination. In the urine, taken for analysis in the daytime, eggs with a characteristic speck were found. What disease does this indicate?

( ) Opisthorchiasis;

( ) Intestinal schistosomiasis;

( ) Japanese schistosomiasis;

(x) Urogenital schistosomiasis;

( ) Dicroceliosis.

>> For the treatment of schistosomiasis is used: <<

( ) Pyrantel;

( ) Mebendazole;

( ) Piperazine;

(x) Prazikwantel;

( ) Albendazole.

>>Schistosomiasis is characterised by: <<

( ) Atrophy of the gastric mucosa;

( ) One of the main manifestations is tenesmus;

( ) Schistosomiasis is an HIV indicator disease;

(x) Infection occurs percutaneously;

( ) Myalgias are characteristic.

>>For schistosomiasis, the following is correct: <<

( ) Refers to geohelminths;

(x) Refers to biogeoliths;

( ) Facial swelling is characteristic;

( ) HIV indicator disease;

( ) All of the above.

Situational task

A 25-year-old patient A., who recently returned from South Africa, was admitted to the hospital on the 3rd day of illness with complaints of blood in the urine at the end of the urination act, a feeling of heaviness in the perineum during urination, colicky pain in the lumbar region, headache, Objectively: The patient is pale, the skin and visible mucous membranes are unchanged, the skin is dry. Hepatomegaly. Pulse is frequent, soft. Epidemiological history: 4 weeks ago, he returned from South Africa, where he swam in a stagnant body of water.

1. Indicate the preliminary diagnosis.
2. 2. Specify the diagnosis of the disease
3. 3. Specify the specific treatment
4. 4. Specify Preventive measures

Answers to the case study

1. Genitourinary schistosomiasis.

2. CBC (moderate leukocytosis, eosinophilia, increased ESR).

RBC (fresh and changed red blood cells). Urine test: schistosome eggs are present. RBC, RIF

3. Hospitalisation of the patient in an infectious disease hospital. Bed rest, high-calorie diet, sparing, taking into account organ pathology. Detoxification therapy. Etiotropic therapy includes Praziquantel at a daily dose of 0.04 g/kg in 2 doses per day.

4. Preventive measures include large-scale treatment of all risk groups, access to safe water, improved sanitation, and shellfish control.

Topic 7. Zoonoses: sap, melioidosis, sodoc, streptobacillosis, listeriosis, foot and mouth disease.

Learning objective: to gain basic knowledge and skills in zoonoses such as sap, melioidosis, sodoc, streptobacillosis, listeriosis and foot-and-mouth disease, which will allow to predict the course of diseases, the development of complications, and to form students' understanding of adequate and highly effective methods of diagnosis and treatment.

List of skills:

1. Follow the basic rules of working at the patient's bedside.
2. Take a medical history with an assessment of epidemiological data.
3. Examine the patient and detect zoonoses (sap, melioidosis, sodoka, streptobacillosis, listeriosis, foot and mouth disease).
4. Conduct a differential diagnosis with other infectious and especially non-infectious diseases with similar symptoms based on the leading zoonotic syndromes under study (sap, melioidosis, sodoc, streptobacillosis, listeriosis, foot-and-mouth disease).
5. To learn how to choose the best methods of laboratory and instrumental examination to confirm or cancel the diagnoses of the zoonoses under study (sap, melioidosis, sodoc, streptobacillosis, listeriosis, foot and mouth disease).
6. Prescribe treatment for patients with zoonoses (sap, melioidosis, sodoka, streptobacillosis, listeriosis, foot and mouth disease).
7. Draw up a plan of preventive measures for the elimination of zoonoses (sap, melioidosis, sodoc, streptobacillosis, listeriosis, foot-and-mouth disease).

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: zoonosis, sporadic morbidity, endemic regions, reservoir of the pathogen, latent infection, sepsis, septicaemia, cachexia, amyloidosis, virulence, latent infection, entry gate, primary effect, regional lymphadenitis, final disinfection, disinfection, quarantine.

SAP. An acute zoonotic bacterial infection that occurs in humans in acute septic and chronic forms with specific damage to the skin, mucous membranes, muscles, joints and internal organs. According to ICD-10 A24. Sap and myeloidosis

Etiology: The causative agent of scrapie is Burkholderia (Pseudomonas) mallei s. Halleomices mallei is a genus of Pseudomonas, family Pseudomonadaceae.

It is a gram-negative rod 2-4 μm long, 0.5-1 μm wide with rounded or slightly pointed ends, prone to polymorphism and easy transformation to L-forms. It does not form spores or capsules, has no flagella, and grows well on conventional nutrient media with the addition of glycerol.

They are not resistant in the external environment, but under susceptible conditions in water and moist soil they can remain viable for up to 1 month, in secretions and carcasses of sick animals - for several weeks. The pathogen is rapidly killed by heat and common disinfectants, but is resistant to low temperatures. In vitro, it is susceptible to streptomycin, tetracycline antibiotics and some sulfonamides (norsulfazole).

Epidemiology: The source and main reservoir in the wild are sick ungulates: horses, donkeys, mules, zebras, camels, as well as predators that become infected by eating the meat of sick animals. In horses, the disease is acute and fatal in 10% of cases. However, in most animals, the disease is chronic, affecting the skin, lungs and nasal mucous membranes. Animals are contagious throughout the disease.

Mechanism and routes of transmission. Transmission of the pathogen to humans occurs through close contact with sick animals as a result of their secretions getting on damaged skin and mucous membranes. Infection can also occur through re-contaminated objects (e.g. harness, straw). Contact with raw meat and the skin of dead animals is also dangerous. Occasionally, infection occurs through the dietary route

when drinking contaminated water. Aerogenous infection is possible in the laboratory. Very rarely, the pathogen is transmitted from a sick person.

In humans, the disease is more commonly recorded in the cold season (due to the stalling of animals).

The susceptibility of the population is very low. The incidence is mostly sporadic and occupational - veterinarians and animal caretakers. It is more common in some countries in Africa, South America and Asia. The causative agent of sapa is considered a factor in biological weapons.

Pathogenesis of scrapie: The incubation period lasts from 2-5 days to 3 weeks, rarely longer. The entrance gates of infection are damaged skin (microtraumas) or mucous membranes (nose, eyes, respiratory system, and occasionally the digestive tract). An inflammatory reaction occurs at the site of the pathogen's penetration, involving the regional lymph nodes, where an inflammatory process with purulent decay develops. The pathogen then spreads hematogenously throughout the body and causes secondary septic foci in the muscles and internal organs. A granulation-purulent process forms in the skin, respiratory tract and lungs. These foci can open up. The lungs are often affected with the formation of pneumosclerosis, abscesses, bronchiectasis. Purulent osteomyelitis and arthritis often develop. Purulent meningitis and brain abscesses may develop. In some cases, the generalisation of the process can occur without localised focal changes and is characterised by extremely severe sepsis.

Pathological anatomy: the picture corresponds to a typical septicaemia with multiple abscesses in various tissues and organs. Immunity to sap is short-lived or not developed at all.

Clinical picture. The disease begins acutely with a severe intoxication syndrome. The temperature suddenly rises to 38-40 °C with chills, headache, aching, arthralgia and myalgia, severe general weakness, vomiting. The temperature curve is of the hectic type. At the site of pathogen penetration, a primary lesion is formed - a dark red papule, which quickly turns into pustules with blood, later serous-purulent contents, and in 2-3 days it turns into an ulcer. Numerous new pustules surrounded by a wide bright red border form around individual pustules and ulcers. The ulcers have a

"greasy" or granulating bottom covered with pus or crusts, with undermined hard edges protruding above the surrounding tissue. Regional lymphangitis and lymphadenitis develop. In 5-7 days after a short-term decrease, the temperature rises again, which corresponds to the generalisation of the infection. The disease progresses rapidly. Multiple secondary nodules, papules, and pustules turn into pustules and ulcers appear. Later, the process involves internal organs, most often the lungs, as well as muscles, cartilage, and bones. Abscesses and deep infiltrates are formed, followed by purulent melting. The general condition of patients deteriorates sharply. Blood pressure drops, tachycardia, heart sounds become deaf, mucopurulent or bloody sputum appears, chest pain, shortness of breath, acrocyanosis. Radiological and clinical findings include pleuropneumonia and lung abscesses. Splenomegaly is often observed. Debilitating diarrhoea is possible.

The acute form of the disease lasts 1-2 weeks and almost all of them end in death due to acute cardiovascular failure and respiratory disorders. Without treatment, the mortality rate reaches 100%.

Chronic sap develops gradually, with exacerbations and remissions over several years in skin, pulmonary and nasal forms.

The most common form is cutaneous. Pustules and ulcers with regional lymphangitis develop slowly. The ulcers heal slowly, often recur and leave large hard scars. Numerous muscle abscesses with recurrent fistulas are formed. In the pulmonary form, there is a "creeping" pleuropneumonia with a predominant lesion of the lower lobes of the lungs. Later, numerous muscle abscesses are involved.

Nasal mucosa lesions are characterised by serous-purulent discharge, ulcers spreading to the pharynx, larynx, and trachea. In the chronic course, severe intoxication, prolonged fever of the wrong type, gradual development of cachexia, often secondary infections and general amyloidosis are characteristic. The mortality rate is about 50%.

Diagnosis of scrapie: Based on epidemiological data (contact with sick animals) and a characteristic clinical picture.

SARS is differentiated from sepsis, lung abscess, melioidosis, pulmonary tuberculosis and pulmonary forms of mycoses (aspergillosis, nocardiosis, histoplasmosis, etc.), lymph node tuberculosis, plague, smallpox, anthrax, furunculosis.

Laboratory methods used in diagnosis include isolation of the pathogen in Gram stained smears from ulcers, nasal mucosa, pustule contents, sputum, blood, and abscess puncture. Bacteriological inoculation of the material on glycerol or potato glycerol agar (growth of colonies in the form of amber-brown mucous plaque). Serological reactions (RSC, RA, RGGA) in the dynamics. An additional method is an allergic test with maleic acid. Maleine is administered intradermally or subcutaneously at a dose of 0.1 ml in a dilution of 1:10 or 1:100. The test becomes positive after 10-15 days of illness. However, this test is not specific enough and dangerous for humans.

Biological test on guinea pigs (males), hamsters and cats with the development of purulent orchitis in 2-3 days (Strauss phenomenon).

Treatment. Treatment of sap is not well developed.

1. Patients are treated in compliance with the anti-epidemic regime, as well as for particularly dangerous infections. In the acute course - for the entire period, in the chronic course - until the ulcers heal.

2. Etiotropic therapy with sulphonamide drugs and antibiotics (tetracycline, chloramphenicol, kanamycin, rifampicin, ciprofloxacin, ofloxacin). Currently, sulfathiazole at 5-6 g/day for 25-30 days or norsulfazole at 6-8 g/day for a month is recommended, in severe cases - in combination with antibiotics.

3. Pathogenetic and symptomatic therapy (detoxification, anti-shock with the use of GCS in the development of SCI, oxygen therapy, vitamins, blood substitutes, immunomodulators).

4. Surgical treatment of abscesses.

5. Topical remedies for chronic scabies - rubbing mercury ointment (2-3 g/day) with iodine into the skin, cauterising scabies nodes. ULTRAVIOLET LIGHT.

Prevention. Quarantine in case of a case of sap disease for 21 days. Emergency prophylaxis with antibiotics (doxycycline) or sulfonamide drugs for 5-10 days. With mandatory serum testing for the presence of antibodies at the end of quarantine.

No specific prophylaxis has been developed. Early detection and veterinary surveillance of sick animals (conjunctival maleic test) is important.

MELIOIDOSIS. Synonyms: pseudo-sap, East Indian sap, pneumoenteritis, morphine septicaemia - a tropical zoonotic bacteriosis that occurs with sepsis and the formation of abscesses in organs and tissues.

ICD-10 codes

A24. Melioidosis

A24.1 Acute melioidosis

A24.2. Subacute and chronic melioidosis

A24.3. Melioidosis, pulmonary, latent, relapsing form

A24.3. Other specified melioidosis

A24.4. Unspecified melioidosis

The causative agent of melioidosis, Burkholderia pseudomallei, belongs to the genus Pseudomonas of the family Pseudomonadaceae. It is a gram-negative, bipolarly coloured rod 2-6 μm long and 0.5-1 μm wide. Aerobic, flagellated, motile, grows well on nutrient media. It does not form capsules or spores. It has two thermolabile endotoxins that cause haemorrhagic-necrotic lesions or death of animals in the experiment. The pathogen persists for a long time in the environment. It survives up to 30 days in a humid environment, 24 days in decaying materials, and up to a month or more in water. It is killed by heat and disinfectants. The pathogen is susceptible to levomycetin, tetracycline, kanamycin, and some sulfonamide drugs.

Epidemiology. Melioidosis is endemic in Southeast Asia and Northern Australia, where it is observed in humans and animals. In Europe and the USA, cases of melioidosis are imported.

In regions endemic for melioidosis, the main reservoir of the pathogen in nature is soil and water contaminated with excretions of infected animals. Animals excrete the

pathogen in their urine and faeces, and become infected themselves when they consume feed and water. The infection can be observed in many species of animals: rats, mouse-like rodents, rabbits, cows, dogs, cats, kangaroos, etc. Arthropods are not involved in the transmission of the infection. In endemic areas, melioidosis is widespread, as evidenced by the fact that 7-10% of the adult population in these areas have antibodies to the melioidosis pathogen. Human infection can occur through the consumption of contaminated food or water, as well as through aerogenic transmission (airborne dust). Infection often occurs when small skin lesions are contaminated with soil. Human-to-human transmission is extremely rare. A case of sexual transmission of the infection to a patient with chronic prostatitis has been described (the pathogen of melioidosis was detected in the prostate secretion). Hospital-acquired infection of immunocompromised individuals is possible.

The highest incidence is observed during the rainy season in agricultural areas and in wartime. Up to 98% of cases are reported in men.

Pathogenesis of melioidosis. The entry point for the infection is minor injuries of the skin or mucous membranes of the digestive or respiratory tract. The pathogen reaches the regional lymph nodes by lymphatic route, where it sometimes multiplies with the formation of purulent foci. In septic forms of melioidosis, the pathogen enters the bloodstream and haematogenously spreads to various organs and systems, forming many secondary foci with caseous decay and abscesses, which can increase in size and merge. Most of the foci develop in the lungs, with single abscesses in all other organs and tissues, including bones. Secondary foci consist of a central zone of caseous necrosis surrounded by granulation tissue. Calcifications do not develop. The septic course of melioidosis is observed in severely debilitated individuals. Probably, melioidosis can also occur in the form of a latent infection, when the pathogen persists for a long time in the area of the entrance gate, causing generalisation of the process over a long period of time with a decrease in the body's immunobiological resistance. After melioidosis, antibodies appear in the blood. Cases of recurrent melioidosis have not been described.

Clinic. The incubation period of melioidosis lasts only 2-14 days (according to laboratory infections from the time of skin damage to the development of the disease).

The main clinical forms:

1) septic (lightning, acute, subacute, chronic);

2) pulmonary (infiltrative, abscessing);

3) latent;

4) recurrent.

The septic form is the most severe. The disease may develop gradually. An inflammatory infiltrate appears at the site of the pathogen's penetration (skin damage), regional lymphadenitis develops, body temperature rises, and soon the disease becomes septic. In the majority of patients, the septic form begins suddenly with chills, high fever, severe headache, and shortness of breath. In some cases, the disease is severe, with repeated vomiting and frequent liquid enteric stools, leading to excoriation, against the background of severe intoxication. An enlarged spleen and liver are often detected, and in some cases, jaundice. Symptoms of cardiovascular failure increase rapidly. This form resembles cholera and septic plague. The patient dies from infectious toxic shock in 2-4 days before secondary septic foci develop (lightning form). In other cases, the primary focus is inflammatory changes in the lungs, from which the infection then spreads haematogenously to various organs and systems. General weakness appears, the temperature with chills rises to 39 °C and above, the patient is disturbed by cough, pleural chest pain, dullness of the percussion sound, and moist wheezing is heard over the affected lungs. The process is more often localised in the upper lobes. Then the severity of the course increases. Multiple pustules appear on the skin and abscesses in the muscles and internal organs. Clinically, it is severe sepsis, which occurs as septicaemia. Only some patients have time to develop certain signs of septicaemia. The disease lasts 8-12 days. In untreated patients, this form is always fatal.

A very rapid development of septic infection is observed in weakened individuals (drug addicts, diabetics, alcoholics, etc.). In these cases, fever and signs of generalised intoxication increase rapidly. At the same time, signs of lung damage and multiple lesions of other organs appear. There is pharyngitis, pustular rash all over the

body, loose watery stools, severe shortness of breath, cyanosis. Some patients develop purulent arthritis, meningitis, and impaired consciousness. Lung radiography reveals nodular opacities with a diameter of about 10 mm, prone to coalescing into larger infiltrates. These forms of the disease usually do not respond to therapy.

In subacute and chronic forms of melioidosis, a longer course is noted with the formation of sluggish abscesses in various organs and tissues. A picture of septicaemia develops. Symptoms depend on the localisation of purulent foci. Against the background of a febrile condition, patients quickly lose body weight. These forms periodically give remissions, but without etiological therapy, patients also die within a month (subacute forms) or in a few months (chronic forms).

The pulmonary form of melioidosis can begin suddenly, but more often this disease develops gradually, sometimes even being detected by accident during X-ray examinations. The main symptoms of this form of melioidosis are fever, cough with purulent, sometimes bloody sputum, increasing weakness, weight loss, and chest pain. Fever is usually irregular or remitting with chills and sweating. A picture of severe pneumonia develops, followed by lung abscesses and purulent pleurisy. Rapid and significant weight loss is characteristic, with decreased appetite and weakness.

Haemogram: neutrophilic leukocytosis, elevated ESR. On X-ray examination, the changes are very similar to those of tuberculosis. The upper lobes are more often affected, in most patients with the formation of thin-walled cavities with a diameter of 1-4 cm. Some patients may have several cavities (2-3 or more). Sometimes the lung lesions occur in the form of infiltrates without caseous decay.

Recurrent melioidosis. The causative agent of melioidosis can persist in the body for a long time in the form of a latent infection, which is detected only during laboratory testing. In cases of immune suppression, the infection is activated in the form of acute septic or pulmonary disease or in the form of chronic localised purulent disease. Relapse develops a long time after the initial infection. A case of relapse 26 years after infection has been described. The possibility of distant recurrence is about 20%. In case of latent form, cases of self-healing are possible.

Complications: lymphadenitis, UTI, liver, spleen, kidney abscess, amyloidosis, fistula.

Diagnosis of melioidosis. The diagnosis is made in a comprehensive manner, taking into account epizootic data, clinical signs, pathological changes and the results of bacteriological tests. An accurate diagnosis is made using the bacteriological method by isolating the pathogen from blood, CSF, synovial fluid, urine, faeces, vomit, sputum, ulcer discharge and abscess contents.

Serological reactions are used (RBC with a specific antigen in a diagnostic titer of 1:8 and above. A negative RBC does not exclude the possibility of melioidosis; a hemagglutination reaction is more sensitive and early positive at titres of 1:16-1:64).

The bioassay is performed by injecting the material into guinea pigs under the skin (males can be injected into the abdominal cavity), and the animals die on the 10th-20th day (males develop orchitis and peritonitis, and subcutaneous injection causes ulcers at the injection site).

It is differentiated from sap, cholera, septicaemia, plague, malaria, typhoid, staphylococcal infection, pulmonary tuberculosis, systemic mycoses, nonspecific purulent diseases, amoebic liver abscesses.

Treatment: always carried out in a hospital setting under a strict anti-epidemic regime.

Long-term etiotropic therapy is used. The most effective is the use of intravenous ceftazidime or ceftriaxone in combination with tetracycline for 2-3 weeks, followed by oral doxycycline for 2-3 months. Good results have been obtained with parenteral levomycetin succinate for 30 days or more, imipenem, amoxiclav, azlocillin, ticarcillin, aztreonam.

They provide active detoxification and symptomatic therapy, weight loss, and surgical treatment of purulent foci.

Prognosis. Before the introduction of antibiotics, mortality in septic forms was close to 100%, with modern methods of treatment, about 50% or more die in septic forms. In other forms of melioidosis, the prognosis is more favourable. The probability of long-term relapse is about 20%.

Prevention. Specific prevention has not been developed. Patients with melioidosis are subject to isolation and hospitalisation. In endemic areas, measures are taken to destroy rodents and protect food from them. Drinking raw water and bathing in stagnant water bodies is prohibited.

If melioidosis is suspected, the affected animals are isolated and examined bacteriologically. Disinfection, deratisation and disinsection are carried out on the affected farm. Slaughter of sick and suspected animals for meat is prohibited. Patients with the disease require long-term medical supervision due to the possibility of late relapses.

FOOT-AND-MOUTH DISEASE (epidemic stomatitis) is an acute infectious disease from the group of zoonoses caused by the Dermaphilus pectoris virus, manifested by intoxication, aphthous lesions of the mucous membrane of the mouth, nose, skin between the fingers and near the nail bed.

ICD-10 code

B08.8. Other specified viral infections characterised by damage to the skin and mucous membranes.

Etiology. The causative agent belongs to the virus of the genus Rhinovirus, family Picornaviridae, which has high virulence, pronounced dermatotropism, and very high variability. It contains RNA, size 8-20 nm. The virus tolerates low temperatures and desiccation. High temperature, UV light, acidic and alkaline environments are detrimental to it.

The source of the disease is a foot-and-mouth disease animal, animal products derived from an animal or contaminated with its excretions. Humans become infected with foot-and-mouth disease by eating food contaminated with the virus or by poor hygiene in contact with a sick animal. Infection occurs through damaged mucous membranes of the mouth and nose. The infection is not transmitted from person to person. During epizootics, isolated cases are recorded in humans, more often in children and livestock workers. There is a possibility of intra-laboratory infection of people and aerogenic infection when working in a room where animals with FMD were

kept. The disease is observed in all countries, mainly in the form of epizootics. Immunity is type-specific and lasts up to 1-1.5 years.

Pathogenesis. The entry gate of the virus is the nasopharyngeal mucosa, less commonly the digestive tract and damaged skin, where it replicates and accumulates due to its pronounced epithelial tropism. The primary lesion develops in the area of primary localisation - specific vesicles. The virus then spreads lymphatically and haematogenously, and disseminates with the formation of numerous secondary lesions on the affected mucous membranes and skin. Virusemia is accompanied by fever and general intoxication. With the development of aphthae, the pathogen disappears from the blood. In severe disease, dystrophic changes occur in the cardiovascular system and central nervous system, kidneys, oesophagus, and stomach.

The formation of immunity is associated with cellular mechanisms (involving macrophages, T- and B-lymphocytes) and the production of virus-neutralising antibodies. The virus is eliminated in saliva, urine, and bile.

Clinical course. The incubation period is from 1 to 12 days, most often 3-8 days.

Classification:

Clinical forms: cutaneous, mucosal, cutaneous-mucosal.

Course: acute, protracted.

Severity: erased, mild, moderate, severe.

Complications: pneumonia, sepsis, myocarditis, and occasionally meningitis.

The onset of the disease is acute, accompanied by severe chills, headache, muscle pain, loss of appetite, and fever of up to 40 degrees. The fever peaks on day 1-2 and lasts for 5-6 days. After 1-2 days, symptoms of mucosal lesions occur: burning sensation in the mouth, excessive salivation, then small bubbles 1 to 3 mm in size appear on the mucous membrane of the mouth and nose, which subsequently increase, forming erosions. There is a sharp hyperaemia and swelling of the lips, gums, nasopharynx, photophobia, redness of the eyes, often pain when urinating, vomiting, and bowel dysfunction. Vesicles appear on the conjunctiva, around the mouth, nasal mucosa, urethra, genitals, skin in the interdigital spaces, phalanges and palms. If

treatment was carried out in a timely manner, the disease proceeds without complications, the fever lasts from 3 to 6 days, and all wounds heal. The total duration of the disease is 2 weeks. If the virus enters the body through the gastrointestinal tract, stomatitis may not appear. In this case, the disease proceeds as acute gastroenteritis. Typical symptoms are intoxication, fever, abdominal pain, nausea, vomiting, diarrhoea. Most often, this form manifests itself in children after infection through dairy products. In severe cases, spotted-papular and haemorrhagic rashes appear on the skin of the neck and trunk. Arterial hypotension and bradycardia are typical. Sometimes there is an enlargement of the liver and spleen. In uncomplicated foot-and-mouth disease, internal organs are not affected.

The prolonged course lasts for several months with relapses of vesicular exanthema against the background of a satisfactory general condition. The erased form presents with mild malaise, moderate headache and vesicles in the interdigital spaces. The inaparanthous form has no clinical manifestations and is diagnosed by the detection of specific antibodies in the blood. Virus carriage can last up to 5-6 months.

Differential diagnosis. Vesicular stomatitis, acute herpetic stomatitis, aphthous stomatitis, chickenpox, drug allergy, erythema exudatum multiforme, enterovirus infection.

Diagnosis:

- clinical symptoms
- epidemiological history
- Epizootological situation in the region
- laboratory tests:

1) Isolation of the virus from vesicles, afters, saliva, blood and faeces in animal kidney culture.

2) serological tests: RHC, RNGA in paired sera with an interval of 6-8 days. Antibody titre increase is observed up to 4 weeks of illness.

3) biological test on Guinea pigs, mice, rabbits.

Treatment. Patients are isolated in a hospital for at least 14 days until the acute manifestations disappear. The diet is sparing with semi-liquid food, fractionally 5-6

times a day with the use of large amounts of fluids. Before meals, 0.1 g of anaesthetic is given, and tube feeding is possible. The oropharynx is irrigated with 3% hydrogen peroxide solution, 0.1% potassium permanganate solution, chamomile or sage infusions, and 0.25% novocaine solution.

Etiotropic therapy: local antiviral therapy is used from the first days. Use 0.25-0.5% oxolinic, 0.25-0.5% tebrophenic, 4% heliomycin, 50% interferon ointments. Leukocyte IFN and 0.1-1% RNase solution are used.

Pathogenetic and symptomatic therapy: anti-inflammatory and analgesic drugs are prescribed. In case of eye lesions, sodium sulfacyl solution is used. Ultraviolet light and laser therapy are used to improve epithelialisation.

Detoxification therapy, cardiovascular drugs, vitamins, analgesics, antihistamines. In severe cases - GCS. Antibiotics and sulphonamides are used in case of secondary infection.

Prevention. Foot-and-mouth disease is one of the most dangerous quarantine veterinary infections. The measures regulated by the International Health Standards include early detection and elimination of foot-and-mouth disease epizootics, sanitary control and quarantine during animal transport.

Disinfection is mandatory. Sick animals are destroyed and burned. Healthy animals are vaccinated. A quarantine is imposed for 21 days after the last sick animal is recovered, killed or slaughtered and the final disinfection is carried out.

In endemic areas, it is forbidden to consume dairy products without heat treatment. Meat is subject to industrial processing. In endemic areas, pregnant women, adolescents and people with microtraumas of the hands are not allowed to work on farms.

The laboratories strictly adhere to the anti-epidemic regime.

Systematic sanitary and educational work is carried out among the population.

RAT BITE DISEASE (Synonyms: rat bite disease, rat bite fever, streptobacillosis, Haverhill fever, Sodocu, streptobacillosis) combines two diseases with similar clinical presentation caused by Spirochete (Spirillum minus) and

Streptobacillus (Streptobacillus moniliformis). The first of these is called sodoku, the second is called streptobacillosis, Haverhill fever. They are united by a history of rat bite and similar clinical symptoms.

Sodoku

ICD-10 codes

A25. Fever from a rat bite

A25.0. Spirillosis

A25.1. Streptobacillosis

A25.9 Rat bite fever is unspecified.

Etiology. The causative agent is Spirocheta minus (Spirillum minus). It is a short, motile rod (2 to 4 μm long, up to 0.5 μm wide) with 2-3 whorls. It is well stained by Romanowski-Gimza. It does not form spores or capsules. It grows poorly on nutrient media. It is sensitive to penicillin, tetracycline, macrolides. Pathogenic for rats, white mice, guinea pigs, monkeys.

Epidemiology. The reservoir and source of infection are rats and, less frequently, other animals (weasels, squirrels, rabbits, dogs, gerbils). Among animals, the pathogen is transmitted by bites, eating infected corpses, transplacental transmission and milk. Human infection occurs through contact with rat bites, rarely with other animals, and sometimes through contaminated milk. The disease is not transmitted from person to person. The susceptibility to infection is high, and people who have contact with animals (hunters, veterinarians, etc.) are most often affected. After the disease, type-specific immunity remains. The incidence is sporadic.

Pathogenesis. The pathogen penetrates through damaged skin. At the site of inoculation, the pathogen accumulates and forms a primary lesion, from which spirochetes spread lymphogenically with the formation of lymphangitis and lymphadenitis. Then the pathogen enters the bloodstream, haematogenously enters the organs of the reticuloendothelial system, where it is fixed and causes the re-generalisation of the infectious process (repeated attacks of the disease). Recovery occurs due to the formation of specific immunity and phagocytosis of the pathogen.

Clinic. The incubation period lasts from 3 days to 2 months. During incubation, the wound at the bite site can heal.

The disease begins acutely with a fever of 39-40°C, which persists for 5-7 days, then critically decreases with profuse sweating. The attack recurs in 3-7 days. The number of attacks (in the absence of etiological therapy) is from 6 to 20 or more, and the disease can last for several months. At the onset of the disease, simultaneously with the onset of symptoms of intoxication, a primary affect occurs at the bite site, which is first a dense painful infiltrate, on which a blister and then an ulcer form. Lymphangitis and regional lymphadenitis develop. There are chills, fever, adynamia, and muscle pain. Very often, starting from the 2nd or 3rd attack, a spotted-papular, urticular, rarely vesicular, petechial rash appears, affecting the whole body, but with predominant thickening in the area of the primary affect. The rash lasts 3-5 days and disappears without pigmentation. During attacks, patients suffer from severe muscle and joint pain, neuralgia, and sometimes paresthesia. Successive attacks occur in the absence of primary affect. The prolonged course of the disease exhausts patients, and bacterial infections often occur, which can lead to death.

An abortive course with recovery after the first fever attack is possible. Mortality without antibiotic therapy reaches 10-12%.

Complications: brain abscesses, soft tissue abscesses, polyarthritis, myocarditis, endocarditis, pericarditis, nephritis, bronchopneumonia, sepsis, lesions of the NS.

Differential diagnosis: felinosis, malaria, sepsis, plague, tularemia, rickettsiosis, Lyme disease, meningococcal infection, measles, rubella, infectious mononucleosis, brucellosis, cutaneous anthrax, secondary syphilis, rheumatoid polyarthritis.

Diagnosis.

Epidemiological history

Clinical picture

Detection of spirochetes in a blood smear, from the primary affect discharge, in a smear and a "thick drop" stained by Romanowsky-Giemsa. Blood sampling is

recommended at the height of fever. Lysis and agglutination reactions with spirochete and patient's serum can be used starting from the 6th-8th day of the disease. Specific antibodies in maximum titres are detected 1-3 months after the onset of the first clinical symptoms, persist for 1.5-2 years, then gradually decrease.

Biological method: intraperitoneal infection of white mice or guinea pigs.

Treatment. Treatment in a hospital with bed rest.

Etiotropic therapy: benzylpenicillin by mouth in a daily dose of 6-12 million units, doxycycline 0.1 g orally 2 p/day. Macrolides or cephalosporins are also used in general therapeutic doses. The duration of antibiotic therapy is 7-14 days. Fatalities may occur in the absence of timely treatment. Pathogenetic and symptomatic therapy depending on the course of the disease. Follow-up in case of disease complications.

Prevention. No specific prevention has been developed.

Nonspecific includes rat control and compliance with safety rules for contact with rodents.

Emergency prophylaxis for rat bites - doxycycline for 5 days.

STREPTOBACILLOSIS A25.1.

Etiology. The causative agent of Streptobacillus moniliformis is a Gram (+) polymorphic bacillus, unstable in the environment. Requires protein media for growth.

Epidemiology. The mechanisms of infection and clinical presentation are similar to sodoc. However, there are significant differences. Sporadic cases are reported in many countries. Infection occurs through the bite of rodents, including laboratory white rats. Infection is possible when rat blood gets into microtraumas of the skin or mucous membranes. Infection is also possible through water and food (if contaminated with rodent excretions). A sick person does not pose an epidemiological risk.

Pathogenesis. It is poorly studied. It resembles a septic condition with damage to various organs. There are lymphadenitis, septic foci in various organs, streptobacillary pneumonia, abscesses, infarctions of the spleen, kidneys, and brain.

Clinic. The incubation period lasts 3-10 days, sometimes longer. The disease begins acutely, with chills, headache and muscle pain. After 2-3 days, a rash (macules,

papules, haemorrhages, sometimes vesicles) appears and quickly disappears on the skin of the extremities, sometimes all over the body. There is often no primary affect. Rashes appear simultaneously, often in the area of individual joints, can spread to the palms and soles, and are accompanied by severe itching. The rash disappears in 3-4 days. Inflammation of the joints, more often of the knees and elbows, is characteristic. The skin over the joints is hyperaemic. Clinical symptoms last for 10 days. In some cases, meningitis, endocarditis, and abscesses occur. The mortality rate is 7-10%.

Complications. Arthritis, myocarditis, endocarditis, pericarditis, soft tissue abscesses, pneumonia, purulent meningitis, persistent diarrhoea, sepsis. Nowadays, they are rare and mainly occur in severe disease, in weakened individuals, and in the elderly.

Diagnostics. Bacteriological examination of blood and synovial fluid,

Serological methods (RA, RCA): maximum antibody titres are observed in 1-3 months; CBC: possible hyperleukocytosis.

Differential diagnosis. Sodocu, yersinia, typhoid fever, malaria, chronic brucellosis, sepsis, leptospirosis, Marseille fever.

Treatment

Etiotropic therapy with penicillin and tetracycline drugs, possibly levomycetin, for at least 7 days. Without treatment, the mortality rate reaches 7-10%.

Infusion and detoxification therapy.

Vitamin therapy.

Medical examination in case of complications of the disease.

Prevention: compliance with sanitary and hygienic rules, disinfection of drinking water, compliance with the rules of cooking, selling and storing products. Comprehensive preventive measures and deratization are carried out. No immunoprophylaxis measures have been developed.

LISTERIOSIS. This is an acute infectious disease, mainly of people with reduced resistance, with various ways of transmission and various clinical

manifestations, most often in the form of tonsillitis, polyadenitis, septicaemia, meningoencephalitis.

ICD-10 codes:

A 32. Listeriosis

A 32.0. Cutaneous listeriosis

A 32.1. Listeriosis meningitis and meningoencephalitis.

A 32.7. Listeriosis septicaemia.

A 32.8. Other forms of listeriosis.

A 32.9. Listeriosis is not specified.

Etiology. The causative agent is Listeria monocytogenes - a Gram (+) bacillus 0.5-2.0 μm long and 0.3-0.5 μm wide. It moves slowly due to 1-4 flagella. Does not form spores, may have a capsule. It belongs to facultative anaerobes, is well cultivated on neutral or slightly alkaline meat-peptone media at 37°C. On dense media, it forms small greyish-white colonies (S-form), which lose virulence during cultivation and turn into rough colonies (R-form). Hemolysin, a lipolytic factor that dissolves macrophages, is released. During decay, endotoxin is released.

The cells contain somatic (O) and flagellar (H) antigens. There are 16 serovars, of which three (1/2b, 1/2a, 4b) cause up to 90% of all cases of listeriosis in humans. There are 9 phagotypes of listeria. Under adverse conditions, listeria form the L-form, which is less virulent. They penetrate macrophages and endothelial cells with the help of the membrane protein internalin. The bacterial haemolysin, listeriolysin O, destroys phagolysosome membranes, which is the main factor of virulence. Bacterial phospholipases help to counteract the antibodies formed. Listeria are quite resistant in the environment, especially to low temperatures and desiccation. They remain in soil and water for years and can multiply. They also multiply in milk and meat at a temperature of 4-6 °C. When boiled, they die in 3-5 minutes. Disinfectant solutions are fatal.

Epidemiology. It belongs to the typical zoonotic infections with natural foci. It has been detected in 42 species of mammals and 22 species of birds. The reservoir in nature is mainly wild rodents (mice, rats, gerbils, squirrels, hares), other mammals

(foxes, wolves, wild boars, deer, gazelles) and birds (partridges, grouse, etc.). Household rodents, farm animals (often sheep), domestic animals and poultry are infected from wild animals.

In mammals and birds, listeriosis is predominantly latent or asymptomatic. Infected animals excrete the pathogen in urine, faeces, milk, nasal mucus, amniotic fluid, and contaminating the environment. Therefore, the main route of transmission is alimentary. Ixodes and gamma ticks, lice, and fleas can spread the infection, but the vector-borne route plays a secondary role.

In healthy people, Listeria has been isolated from the vagina, cervix, nose, ear, blood, urine, and faeces. However, the role of humans in the spread of the infection remains poorly understood.

Humans become infected from animals mainly by eating raw milk, insufficiently thermally processed meat products, eggs, and water. When caring for sick animals, slaughtering them, processing their skin and meat, the contact route of infection can be implemented. Aerogenic infection cannot be ruled out when cleaning the premises where animals are kept, processing fur, or agricultural work. Infection through blood-sucking insects can also be assumed (this route has not been proven in humans). Vertical transmission of listeria through the placenta or during childbirth by ingestion of amniotic fluid, blood contamination, and vaginal discharge is well known. A case of transmission of Listeria to a child through mother's milk has been described. Sexual transmission is also possible.

Of all the population groups, housewives, livestock farmers, veterinarians, meat-packing plant workers, and people with immunodeficiency are most likely to be infected. People are infected all year round. The incidence is sporadic. Minor outbreaks are possible. The incidence increases in spring and summer.

Pathogenesis. The pathogen enters the body through mucous membranes and damaged skin. Then, listeria penetrate the regional lymph nodes by lymphatic means, affecting them. After breaking through the lymphatic barrier, the bacteria are haematogenously distributed throughout the body, settling in the parenchymal organs. The occurrence of listeriosis and the severity of the course depends on the resistance

of the macroorganism and the infectious dose, as the pathogen has low virulence. Individuals with reduced T-cell function are more likely to be affected. The ability of listeria to stay and multiply intracellularly, in particular in macrophages and granulocytes, plays an important role. It often occurs as a septic infection. Listeria often show a pronounced affinity for nervous tissue, causing the development of meningoencephalitis.

Numerous small foci of inflammation in the form of granulomas (listeriosis) are formed in the tonsils, liver, spleen, adrenal glands, and brain, some of which may become necrotic with the formation of abscesses. General and local changes are largely due to the action of bacterial endotoxins. Allergic changes occur and immunity is formed. In addition to cellular reactions, specific agglutinins, precipitins and complement-binding antibodies are detected in the blood. However, post-infectious immunity is weak, and relapses and reinfection are possible. The patient remains a bacterial carrier.

Clinic. The incubation period lasts 2-4 days. There are angio-septic, nervous, ocular glandular, tuff-like and isolated (rare) forms of listeriosis. In addition, there is listeriosis in pregnant women and newborns.

The angio-septic form occurs more often in adults after nutritional infection.

General intoxication, febrile reactions, tonsillitis (catarrhal, follicular or lacunar, less commonly ulcerative filmy tonsillitis, peritonsillar abscess). On day 5-6 of the disease, a finely spotted or polymorphic rash without a typical localisation may appear. It resembles infectious mononucleosis. The course is severe. In case of sepsis or meningitis, the disease lasts up to 1-2 months.

The nervous form is more common in children, due to the greater permeability of the BBB and reduced immunological resistance. Meningitis, meningoencephalitis, and less commonly, encephalitis with lesions of individual pons (pairs VII, VIII), psychosis, and arachnoiditis develop.

Purulent meningitis. Generalised intoxication, fever, paresthesia and hyperesthesia are pronounced. In encephalitis, hallucinations, aggressiveness, and

mental automatism are noted. Severe fatal stem encephalitis has been described. Convalescents may have paresis and mental disability.

The ocular glandular form is characterised by the development of unilateral or bilateral conjunctivitis.

The eyelids are swollen. Several superficial follicles with granulomas in the centre (3-5 mm in diameter) appear on the hyperemic conjunctiva. Choroiditis may be involved. Regional lymph nodes are enlarged, dense, painful. Fever, chills, headache, dyspeptic symptoms.

Typhoid-like form

Prolonged fever, enlarged liver and spleen, rosaceous-papular rash and haemorrhage. Fever is accompanied by intense sweating. Fever (remitting, constant, wave-like) lasts 2-3 weeks. Generalised lymphadenopathy is common. Septic foci occur in various organs (endocarditis, specific abscess pneumonia, purulent pleurisy, polyserositis, hepatitis with jaundice).

In pregnant women, it occurs latently or in the form of acute respiratory infections, tonsillitis, polyadenitis, pyelonephritis, cystitis, enteritis, meningitis or meningoencephalitis.

Since listeria have a tropism for the uterus and placenta, there is often a high incidence of preeclampsia, miscarriages, abortions, and premature births with the birth of a dead fetus. The amniotic fluid is turbid. Vaginal carriage of listeriosis may persist after listeriosis.

In newborns, it is more common in the form of granulomatous sepsis or meningitis.

At the onset of the disease in the first week of life, whitish granulomas appear on the mucous membranes, and papules and petechiae on the skin. Severe intoxication (refusal to breastfeed, repeated vomiting, convulsions), inflammatory infiltrates in the lungs, myocarditis, hepatomegaly with jaundice may occur. Complications: hydrocephalus, mental retardation. If the disease occurs at a later stage, the child initially appears healthy, but after 1 month septicaemia or purulent meningitis develops. The mortality rate is high.

Isolated forms (liquid): listeria pneumonia, endocarditis, hepatitis, urethritis.

Classification:

Clinical forms: angina-septic, nervous (meningitis, meningoencephalitis, polyradiculoneuritis, psychosis), ocular-glandular, glandular, typhoid, endocarditis, septicaemia, listeriosis of pregnant women, fetus and newborn, skin.

Course: acute, subacute, chronic.

Severity: mild, moderate, severe.

Asymptomatic carrier.

Complications: SCI, hydrocephalus, mental retardation.

Diagnosis:

Bacteriological examination (nasal and oropharyngeal mucus, eye discharge, blood, CSF, lymph node puncture; in parturients - pieces of placenta, amniotic fluid, vaginal discharge; in newborns - blood from the umbilical cord, meconium; sectional material - brain, liver, spleen) - sowing on agar and nutrient broth (glucose-glycerol-serum or glucose-liver-meat). It is rarely possible to isolate Listeria by the direct method.

Biological study on white mice. Animals die in 2-6 days.

Conjunctival test on guinea pigs. When a daily broth culture is administered, purulent conjunctivitis develops after 2-3 days.

Serological reactions. In the second week of the disease, antibodies are detected in RA (diagnostic titer 1:400-1:800) and RNGA (1:80), and later in RBC (1:5-1:10). With recovery, the titres decrease rapidly.

PCR.

In the clinical picture of encephalitis, CT and MRI are used (to detect focal changes, including abscesses).

Differential diagnosis:

Angina-septic form: tonsillitis, diphtheria, infectious mononucleosis, staphylococcal sepsis.

Ocular glandular form: tularemia, blennorrhoeic conjunctivitis.

Nervous form: tuberculosis and other bacterial meningitis and meningoencephalitis.

Typhoid form: typhoid fever, paratyphoid fever, sepsis.

In pregnant women: brucellosis, toxoplasmosis, CMV infection.

Viral hepatitis, epidemic mumps, ornithosis, chickenpox, syphilis.

Treatment. Patients with severe disease and pregnant women are hospitalised. Bed rest, good nutrition, physical and mental rest are indicated.

Etiotropic therapy is prescribed as early as possible - the antibiotics of choice are ampicillin and benzylpenicillin. Trimethoprim-sulfomethoxazole, vancomycin, carbopenem may be prescribed; in case of endocarditis, ampicillin with aminoglycosides is prescribed. High doses are prescribed (except for pregnant women). The duration of therapy for meningoencephalitis is 21 days, for brainstem lesions, brain abscess and endocarditis - 6 weeks. Cephalosporins and chloramphenicol are not effective.

Pathogenetic and symptomatic therapy: detoxification, cardiovascular, antihistamines, analgesics. In severe cases, prednisolone 20-40 mg per day for 5-7 consecutive days is prescribed. Topical therapy (rinsing the oropharynx with antiseptics, in the ocular-glandular form - 30% sodium sulfacyl, 0.5% hydrocortisone solution). Aloe, cyclopherone, donor Ig, multivitamins are used to stimulate defences, regeneration and metabolic processes. The overall mortality rate is 20-30%, in immunodeficiency states it reaches 30-50%.

Prevention. Prevention and elimination of listeriosis in farm animals (quarantine of animals for a month, avoid contact with wild animals). Deratization, catching stray dogs and cats, disinfestation. Carrying out anti-epizootic and anti-epidemic measures in listeriosis-affected farms.

It is important to exclude the alimentary route of infection - personal hygiene, processing of vegetables and fruits, pasteurisation of milk.

Persons from risk groups in the epidemic focus are indicated to eradicate Listeria colonisation with ampicillin or trimethoprim-sulfamethoxazole for 5-7 days.

Women with a complicated obstetric history should be screened for listeriosis. No specific prophylaxis has been developed.

Presentation:

https://docs.google.com/presentation/d/1tIrNAPHDnjLfrkijE6swCxayVmaGgd4o/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for students' self-control

1. Etiology and epidemiology of sap.
2. Etiology and epidemiology of melioidosis.
3. Etiology and epidemiology of foot and mouth disease.
4. Etiology and epidemiology of rat bite disease (sodoc, streptobacillosis).
5. Etiology and epidemiology of listeriosis.
6. Pathogenesis and clinic of sap.
7. Pathogenesis and clinic of melioidosis.
8. Pathogenesis and clinic of foot-and-mouth disease.
9. Pathogenesis and clinic of rat bite disease (sodoc, streptobacillosis).
10. Pathogenesis and clinic of listeriosis.
11. Specific diagnostics of the sap.
12. Specific diagnosis of melioidosis.
13. Specific diagnosis of foot-and-mouth disease.
14. Specific diagnosis of diseases caused by rat bites (sodoc, streptobacillosis).
15. Specific diagnosis of listeriosis.
16. The basic principles of treatment and prevention of sap.
17. Basic principles of treatment and prevention of melioidosis.
18. Basic principles of foot and mouth disease treatment and prevention.
19. The basic principles of treatment and prevention of rat bite disease (sodoc, streptobacillosis).
20. Basic principles of treatment and prevention of listeriosis.

Glossary

Zoonotic infections (from the ancient Greek ζῷον "animal, living being" + Greek νόσος "disease") are a group of infectious and parasitic diseases whose pathogens parasitise certain animal species and for which animals are a natural reservoir. The source of infection (or invasion) for humans is a sick animal or an animal carrier of pathogens. Under certain sanitary and environmental conditions that favour a particular mechanism of pathogen transmission, it is possible to transmit zoonoses to humans. However, zoonotic pathogens cannot circulate in groups of people, since humans are a biological dead end for them, are not included in the epizootic process and do not participate in the evolution of the pathogen as a species. Only in some zoonoses, for example, plague, yellow fever, under certain conditions, a sick person can be a source of infectious agents.

Sporadic **morbidity -** (Greek: sporadikos scattered, separate) a low, usual for a given area and time level of morbidity of a disease, manifested in the form of separate, unrelated cases.

Endemic morbidity (endemicity) is the level of morbidity that is constantly recorded in a certain territory and is characteristic of this territory due to the presence of a reservoir of infection. If the reservoir is animals, then there is an enzootic, which in turn can determine the level of endemic incidence of zoonoses among humans.

Reservoir of the pathogen - a set of the main sources of infection - is a biological species (human, animal) or the environment that ensures the existence of the pathogen in nature as a biological species.

Latent (inapparent, asymptomatic) infection - (from the Latin latentis - hidden) - a limited process with a long and cyclic circulation of the pathogen, similar to that observed in overt forms of the infectious process, without external cellular manifestations. The pathogen enters the body, multiplies in it, but does not cause appropriate immune responses from the macroorganism. A latent infection may result in recovery and the macroorganism's elimination of the infectious agent.

Sepsis (*σῆψις - putrefaction*) is a pathological process of organ dysfunction based on the body's response in the form of a systemic inflammatory response

syndrome (SIRS) caused by various infectious factors. In fact, sepsis is a special form of the body's response to various pathogens. Organ dysfunction is assessed using the SOFA (Sequential Organ Failure Assessment) scale.

Septicaemia is the formation of purulent foci in various tissues and organs in sepsis.

Cachexia - (wasting syndrome, general atrophy) (Latin *cachexia*, from the ancient Greek *κακός* - bad and the ancient Greek ἕξις - condition) - a clinical syndrome, a condition accompanied by a large loss of body weight due to the disappearance of fatty tissue, muscle atrophy.

Amyloidosis (amyloid dystrophy) is a disease of protein metabolism disorders accompanied by the formation and deposition of a specific glycoprotein, amyloid, in tissues.

Virulence - (from Latin virulentus - "poisonous"), the degree of pathogenicity of a given infectious agent. Virulence depends on both the properties of the infectious agent and the susceptibility of the infected organism. Virulence is judged by the severity of diseases caused by viruses, bacteria or other pathogens, and in animal experiments by the lethal dose of the infectious agent.

Entrance gates are tissues of the body through which a microorganism enters the macroorganism.

Primary affect (also called primary complex) is a clinical symptom that indicates the presence of a limited inflammatory process at the site of primary penetration of an infectious disease agent. If such an inflammatory process occurs on the skin, it is traditionally referred to as primary affect, while the term "primary complex" is often used for inflammatory primary processes in other organs or tissues.

Regional lymphadenitis is an inflammation of the lymph nodes caused by various pathogens that spreads through the lymphatic system from nearby inflammation sites.

Final disinfection - is carried out once in the centre of infection after isolation, departure, death of a patient or bacillus carrier. Its purpose is to completely disinfect objects that could be contaminated with an infectious agent. The final disinfection is

carried out in the foci of those infections whose pathogens are resistant in the environment.

Disinfection (from the French word des - negation and Latin infectio - infection) is the destruction of pathogenic and opportunistic microorganisms (bacteria, viruses, rickettsia, protozoa, fungi, toxins) in the environment surrounding a person.

Quarantine (from the Italian quaranta - forty) is an administrative and healthcare measure to restrict contacts of an infected or suspected infected person(s), animal, cargo, goods, vehicle, settlement, at the country level or between states, used to prevent the spread of certain dangerous infectious diseases.

Test control of knowledge

>>What is the cause of the development of sap?

( ) virus

(x) Gram-negative bacillus

( ) Gram-positive bacillus

( ) staphylococcus aureus

( ) mushrooms

>>What is used for etiotropic treatment of sap?

( ) co-trimoxazole

( ) ceftriaxone

(x) sulfathiazole

( ) valavir

( ) metronidazole

>>What is the main reservoir of the causative agent of melioidosis in endemic regions?

( ) cloven-hoofed animals

( ) mouse-like rodents

( ) milk

(x) soil and water

( ) person

>>What is the most severe form of melioidosis?

( ) pulmonary

(x) septic

( ) recurrent

( ) complicated

( ) anginal

>>What is the etiological factor of foot-and-mouth disease?

(x) RNA virus

( ) DNA-containing virus

( ) bacterium

( ) mushrooms

( ) worm

>>What is the typical clinical manifestation of foot-and-mouth disease?

( ) purulent brain damage

( ) purulent lesions of the skin and mucous membranes

(x) aphthous lesions of the skin and mucous membranes

( ) heart disease

( ) development of acute renal failure

>> There is an etiological factor in sodoku: <<

( ) staphylococcus aureus

(x) spirochete

( ) streptobacillus

( ) giardia

( ) candidate.

>>The main tank and the source of soda: <<

( ) birds

( ) sheep

(x) rats

( ) mites

( ) is correct.

>>There is an etiological factor in streptobacillosis: <<

( ) staphylococcus aureus

(x) Streptobacillus

( ) spirochete

( ) giardia

( ) candidate.

>>Emergency prophylaxis for a rat bite: <<

( ) benzylpenicillin

( ) ceftriaxone

( ) oseltamivir

(x) doxycycline

( ) is not carried out.

>>The most common clinical form of listeriosis in adults: <<

( ) cutaneous

( ) oculoglandular

( ) intestinal

( ) pneumatic

(x) angina-septic.

>>The drug of choice for the etiotropic therapy of angio-septic listeriosis: <<

(x) a penicillin group drug

( ) cephalosporins

( ) aminoglycosides

( ) macroliths

( ) is correct.

MODULE 2. CLINICAL PARASITOLOGY AND TROPICAL MEDICINE. DISEASES CAUSED BY VIRUSES AND THOSE CONTROLLED BY THE WHO.

Topic 8: Arboviral infections. Features of the clinical course. Hemorrhagic fevers: Ebola, Lassa, Marburg, Dengue, Chikungunya, South American haemorrhagic fevers. Phlebotomous fever. Encephalitis: California, Venezuelan, American equine, Japanese, Rift Valley. The concept of rotary typhus.

Learning objectives: To get acquainted with the concept of arboviral infections and features of their clinical course. To get acquainted with the classification of haemorrhagic fevers. Be able to diagnose encephalitis in infectious diseases. Learn the classification of encephalitis. To consider the concept of falciparum.

List of skills:

1. Follow the basic rules of working at the patient's bedside.
2. Take a medical history with an assessment of epidemiological data
3. Examine the patient and identify the main symptoms and syndromes of haemorrhagic fevers, relapsing fever and typhoid fever and justify the clinical diagnosis.
4. Draw up a plan for laboratory and instrumental examination of the patient.
5. Interpret laboratory test results
6. Correctly assess the results of specific diagnostic methods.
7. Draw up an individual treatment plan taking into account epidemiological data, stage of the disease, complications, severity of the condition, allergic history, concomitant pathology; provide emergency care
8. Provide recommendations on the regime, diet, examination, and supervision during the period of recuperation.
9. Prepare medical documentation.
10. Search for up-to-date literature in the field
11. Participate in the dissemination of medical knowledge among the population

12. Conduct educational work (talks, lectures) among the public, patients and healthcare facilities

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Arboviral diseases; haemorrhagic fever; encephalitis

ARBOVIRAL INFECTIONS - (an acronym from the Latin arthropoda - arthropod and the English word borne - born) - is a group of viruses that are transmitted to vertebrates (including humans) through the bite of blood-sucking insects (mosquitoes, ticks, fleas, lice, bedbugs)

Classification

1. Family Togaviridae
2. Family Flavivivridae
3. Family Bunyaviridae
4. Family Rhabdoviridae
5. Family Arenaviridae
6. Family Reoviridae

Clinically, arboviral infections manifest as fever of unknown genesis, CNS lesions (encephalitis), and haemorrhagic fevers.

DENGUE FEVER is a viral disease transmitted by mosquitoes that has been spreading rapidly in all WHO regions in recent years. Dengue is widespread in the tropics, with local differences in risk largely dependent on rainfall, temperature and rapid urbanisation.

Severe Dengue was first recognised in the 1950s during the Dengue epidemics in the Philippines and Thailand. There are 4 distinct but closely related serotypes of the virus that cause Dengue (DEN-1, DEN-2, DEN-3 and DEN-4). After recovering from

an infection caused by one of these serotypes, lifelong immunity to that particular serotype is established. However, cross-immunity to other serotypes after recovery is only partial and temporary. Subsequent infections with other serotypes increase the risk of developing severe dengue.

Epidemiology. According to a recent estimate, 390 million people are infected with Dengue annually (95% confidence interval 284-528 million), of whom 96 million (67-136 million) have clinical manifestations (of any severity). Another study on the prevalence of Dengue estimated that 3.9 billion people in 128 countries are at risk of Dengue virus infection. It is estimated that 500,000 people with severe Dengue require hospitalisation each year, of whom 2.5% die. Between 2010 and 2016, a 28% reduction in the mortality rate was recorded worldwide due to significant improvements in patient management by strengthening the capacity of countries.

Mechanism and route of transmission. The main vectors are Aedes aegypti mosquitoes. The virus is transmitted to humans through the bites of infected female mosquitoes. After an incubation period of 4-10 days, an infected mosquito is able to transmit the virus throughout its remaining life.

Infected humans are the main carriers of the viruses and contribute to their reproduction by being a source of viruses for uninfected mosquitoes. Patients already infected with Dengue virus can transmit the infection (within 4-5 days; maximum 12 days) via Aedes mosquitoes after the first symptoms of the disease. Aedes albopictus, the second most important vector of Dengue in Asia, has spread to North America and more than 25 countries in the European region. Ae. Albopictus are easily adaptable to new environments and can therefore survive in colder areas of Europe.

Clinic. High fever (40°C/104°F) is accompanied by two of the following symptoms:

- severe headache,
- pain in the area behind the eyes,
- muscle and joint pain,
- nausea, vomiting,
- swollen lymph nodes or rash.

Severe Dengue is a potentially fatal complication associated with plasma leakage, fluid accumulation, respiratory failure, severe bleeding, or organ damage.

Dangerous signs appear 3-7 days after the first symptoms, along with a fever (below 38°C/100°F) and include severe abdominal pain, uncontrollable vomiting, rapid breathing, bleeding gums, fatigue, agitation and the presence of blood in the vomit.

The next 24-48 hours of this critical stage can be fatal; proper medical care is needed to prevent complications and death.

Diagnosis. Methods are used to confirm the diagnosis:

1. Virus isolation and serological identification; test material: blood, serum, tissue.
2. PCR; test material - blood, plasma, serum, tissue
3. Antigen detection; test material is serum or tissue.
4. ELISA for the detection of IgM; test material - blood, plasma, serum

Test turnaround time: from 30 minutes (quick orientation tests) to 7-14 days (final confirmation).

Treatment. There is no specific treatment for Dengue fever. In cases of severe Dengue, medical care by doctors and nurses with expertise in the manifestations and development of the disease can help save lives and reduce mortality rates from more than 20% to less than 1%. In the treatment of severe Dengue, it is critical to maintain the patient's body fluids at the proper level.

Vaccination. The first dengue vaccine, Dengvaxia® (CYD-TDV), was licensed in December 2015 and has now been approved by regulatory authorities in 20 countries for use in people aged 9-45 years in endemic areas. The analysis demonstrated that a subset of study participants who were likely to be seronegative at the time of their first vaccination were at increased risk of more severe Dengue and hospitalisation as a result of the disease compared to unvaccinated participants.

Clinical trials have shown that the live attenuated dengue vaccine CYD-TDV is effective and safe for people who have previously been infected with Dengue virus (seropositive patients), but is associated with an increased risk of severe dengue in those who are exposed to natural dengue infection for the first time after vaccination (seronegative patients).

Prevention and control. Currently, the only way to control or prevent the transmission of Dengue virus is to control mosquito vectors through the following measures:

- preventing mosquitoes from accessing places for laying eggs;
- proper disposal of solid waste and destruction of artificial habitats made by humans;
- keeping home water supplies in closed containers and emptying and washing them weekly;
- use of appropriate insecticides for water tanks stored outdoors;
- use of personal protective equipment, mosquito nets, long-sleeved clothing, insecticide-treated materials;
- Improving the participation and mobilisation of individual communities for sustainable vector control;
- During outbreaks, emergency vector control measures may also include the use of spray insecticides;
- Active monitoring and surveillance of vectors is necessary to determine the effectiveness of vector control measures.

EBOLA FEVER. The Ebola virus causes an acute severe illness that is often fatal if left untreated. Ebola Virus Disease (EVD) first emerged in 1976 in 2 simultaneous outbreaks in Nzara (now South Sudan) and Yambuku, Democratic Republic of Congo. The second outbreak occurred in a village next to the Ebola River, from which the disease takes its name.

The 2014-2016 outbreak in West Africa is the largest and most complex Ebola outbreak since the virus was discovered in 1976. More people have become ill and died in this outbreak than in all other outbreaks combined. It is also spreading between countries, having started in Guinea and spread across land borders in Sierra Leone and Liberia.

Five types of Ebola have been identified: Zaire, Bundibugio, Sudan, Reston and Tai Forrest. The first three of them - Ebola viruses Bundibugio, Zaire and Sudan -

are associated with major outbreaks in Africa. The virus that caused the outbreak in West Africa in 2014-2016 belongs to the Zaire species.

Mechanism of transmission. The natural hosts of the Ebola virus are fruit bats of the Pteropodidae family. Ebola enters the human population through close contact with the blood, secretions, organs or other body fluids of infected animals, such as chimpanzees, gorillas, fruit bats, monkeys, forest antelopes and porcupines found dead or sick in humid forests.

Ebola is then spread by person-to-person transmission through close contact (through damaged skin or mucous membranes) with the blood, secretions, organs or other body fluids of infected people, as well as with surfaces and materials (e.g. bedding, clothing) contaminated with such fluids. Healthcare workers often become infected while providing care to patients with suspected or confirmed EVD, as a result of close contact with patients and insufficiently strict infection control practices. Funeral rites, which include direct contact with the body of the deceased, can also contribute to the transmission of the Ebola virus. People remain contagious as long as the virus is in their bodies.

Clinic. The incubation period is from 2 to 21 days. People are not contagious before symptoms appear. The first symptoms are:

- sudden onset of fever
- muscle pain
- headache
- sore throat.

This is followed by vomiting, diarrhoea, rash, kidney and liver dysfunction and, in some cases, both internal and external bleeding (e.g., blood from the gums, blood in the stool).

Laboratory tests reveal low levels of white blood cells and platelets along with elevated liver enzymes.

Virus carriage in people after EVD: Ebola virus is known to persist in immunocompromised parts of the body of some people who have had Ebola virus disease. These parts of the body include the testes, the inside of the eyes and the central

nervous system. In women who are infected during pregnancy, the virus persists in the placenta, amniotic fluid and embryo. In women infected while breastfeeding, the virus can persist in breast milk. The recurrence of symptoms in any person who has had CWD due to increased viral replication in a particular part of the body has been documented, although it is a rare phenomenon. The causes of this phenomenon are not fully understood.

Diagnosis. It can be difficult to distinguish EVD from other infectious diseases, such as malaria, typhoid fever and meningitis. The following tests are performed to confirm that symptoms are caused by the Ebola virus:

- Enzyme-linked immunosorbent assay with antibody capture (ELISA);
- antigen detection tests;
- Serum neutralisation reaction;
- reverse transcriptase polymerase chain reaction (RT-PCR);
- electron microscopy;
- Isolation of the virus in cell cultures.

When selecting diagnostic tests, technical specifications, disease incidence and prevalence rates, and the social and health consequences of test results should be taken into account. Diagnostic tests that have been independently and internationally evaluated are strongly recommended for use.

The tests currently recommended by the WHO include the following:

- Automated and semi-automated nucleic acid amplification tests (NAT) for routine diagnostics.
- Rapid antigen detection tests for use in remote areas without access to NAT. These tests are recommended for screening purposes as a component of surveillance, but reactive tests should be confirmed by NAT.

Specimens taken from patients pose an extremely high biological hazard; laboratory testing of uninactivated specimens should be carried out under conditions of maximum biological isolation. During national and international transport, all biological specimens should be placed in triple-packaging systems.

Treatment. Supportive care with oral or intravenous fluids and treatment of specific symptoms improves survival. There is no proven treatment for CJD yet. However, a number of potential treatments are currently being evaluated, including blood products, immune and drug therapies**.**

Vaccination. An experimental vaccine against the Ebola virus has demonstrated a high preventive effect against this deadly virus in a large-scale trial in Guinea. The vaccine, called rVSV-ZEBOV, was tested in 2015 in a trial involving 11,841 people. Among the 5,837 people who received the vaccine, no cases of Ebola were reported 10 days or more after vaccination. At the same time, 23 cases were reported among those who did not receive the vaccine 10 days or more after vaccination.

Prevention. Reducing the transmission of CWD is achieved by:

- reduce the risk of transmission of infection from wild animals to humans. Animals should be handled with gloves and other appropriate protective clothing. Their products (blood and meat) should be subjected to thorough heat treatment before being eaten.
- reducing the risk of person-to-person transmission of infection through direct or close contact with people with symptoms of CWD, especially with their body fluids. When caring for patients at home, gloves and appropriate personal protective equipment should be worn. Hands should be washed regularly after visiting patients in hospitals and caring for patients at home.
- Reducing the risk of possible sexual transmission - as this risk cannot be ruled out, men and women who have recovered from Ebola should abstain for at least three months after the onset of symptoms.

Infection control in healthcare facilities. Healthcare workers should always follow standard precautions when caring for patients, regardless of the intended diagnosis. These include basic hand hygiene, respiratory hygiene, the use of personal protective equipment (to protect themselves from splashing or otherwise coming into contact with infected materials), safe injections, and safe burial of the dead.

Healthcare workers caring for patients with suspected or confirmed Ebola virus infection should take additional infection control measures to prevent contact with the patient's blood and body fluids, as well as contaminated surfaces or materials such as clothing and bedding. When in close contact (within one metre) with a CEVD patient, healthcare workers should protect their face (with a face shield or medical mask and goggles) and wear a clean, non-sterile, long-sleeved gown and gloves (sterile gloves for some procedures).

Laboratory workers are also at risk. Specimens taken from humans and animals for the diagnosis of Ebola infection should be handled by trained personnel in properly equipped laboratories.

LASSA HAEMORRHAGIC FEVER

Epidemiology. Humans usually become infected with Lassa virus through contact with the urine or faeces of infected Mastomys rats. Lassa virus can also be transmitted from person to person through direct contact with the blood, urine, faeces or other secretions of a person infected with Lassa fever. There is no epidemiological evidence of airborne transmission of Lassa virus from person to person.

Person-to-person transmission occurs both in communities and in healthcare facilities through the use of contaminated medical equipment, in particular through the reuse of needles. There is evidence of sexual transmission of Lassa virus.

People living in rural areas, where Mastomys rats are commonly found, are most at risk. Healthcare workers who provide care to patients with Lassa fever in the absence of appropriate barrier care and infection control practices are at risk.

Clinic. The incubation period is from 6 to 21 days. Early symptoms of the disease:

- high temperature,
- general weakness and malaise.

After a few days, you may experience headache, sore throat, muscle pain, chest pain, nausea, vomiting, diarrhoea, cough and abdominal pain.

In severe forms of the disease, swelling of the face, fluid in the lung sinuses, bleeding from the mouth, nose, vagina or gastrointestinal tract may develop. Later, shock, convulsions, tremors, disorientation and coma may occur.

In 25% of patients who are cured, deafness develops. In half of these patients, partial hearing recovery occurs in 1-3 months. During the recovery period, temporary hair loss and gait disturbances may occur.

In fatal cases, death usually occurs within 14 days of the onset of symptoms. The disease is particularly severe in the later stages of pregnancy, with maternal mortality and/or fetal loss rates exceeding 80% during the last trimester.

Diagnosis. Due to the diverse and non-specific symptoms of Lassa fever, clinical diagnosis, especially in the early stages of the disease, is often difficult. Lassa fever is difficult to distinguish from other viral haemorrhagic fevers.

The final diagnosis of an infection caused by Lassa virus can only be made in the laboratory with the following tests:

1. Enzyme-linked immunosorbent assay (ELISA)
2. Antigen detection tests
3. Reverse transcription polymerase chain reaction (RT-PCR) method
4. Isolation of the virus by cell culture.

Treatment and vaccination. In the early stages of the disease, which manifests itself clinically, the antiviral drug ribavirin is considered to be an effective treatment for Lassa fever. There is no information on the efficacy of ribavirin as a post-exposure prophylactic against Lassa fever.

To date, there is no vaccine against Lassa fever.

Prevention and control of the disease. Prevention of Lassa fever is based on good hygiene at the community level to prevent rodents from entering homes, storing grain and other food in containers that are inaccessible to rodents, removing waste disposal sites from buildings, keeping homes clean and keeping cats. Since the population of Mastomys in endemic areas is very large, their complete extermination is not possible. Family members should always take precautions to prevent contact with blood and bodily fluids when caring for sick people.

Healthcare workers who provide care to patients with suspected or confirmed Lassa fever should take additional infection control measures to prevent contact with patients' blood and bodily fluids, as well as with infected surfaces or materials. When in close contact with patients with Lassa fever (within one metre), healthcare workers need face protection (face shield or medical mask and goggles), a clean, non-sterile long-sleeved gown and gloves.

Laboratory workers are also at risk. Specimens taken from humans and animals for the purpose of investigating Lassa virus infection should be handled by trained personnel, and the research should be carried out in laboratories with maximum biosecurity.

CHIKUNGUNGA FEVER. The mosquito-borne viral disease chikungunya was first described during an outbreak in southern Tanzania in 1952. The causative agent is an RNA virus belonging to the genus alphavirus of the togavirus family. The name "chikungunya" comes from a Kimakonde verb meaning "to become contorted", which corresponds to the appearance of hunched people suffering from joint pain.

Epidemiology. Chikungunya is distributed in Africa, Asia and the Indian subcontinent. For a number of years, the level of human infection in Africa has remained relatively low, but a major outbreak occurred in the Democratic Republic of the Congo in 1999-2000 and in Gabon in 2007.

Since 2016, there have been a total of 349,936 presumptive and 146,914 laboratory-confirmed cases. The highest number of cases was reported in Brazil (265,000 presumptive cases), Bolivia and Colombia (19,000 presumptive cases each). Since 2016, local transmission of chikungunya has been reported for the first time in Argentina following an outbreak with more than 1,000 suspected cases. "

In the Africa region, an outbreak of chikungunya was reported in Kenya, with more than 700 suspected cases. In 2017, Pakistan continued to take measures to respond to the outbreak that began in 2016.

Transmission mechanism. The virus is transmitted from person to person through the bites of infected female mosquitoes. The disease is mainly transmitted by

two species of mosquitoes, Aedes aegypti and Aedes albopictus, which can also transmit other viruses, including Dengue. These mosquitoes can bite people throughout the daylight hours, but are most active in the early morning and late afternoon. Both mosquito species bite people outdoors, while Ae. Aegypti - indoors. Symptoms usually appear 4-8 days after an infected mosquito bites a person, but this period can be from 2 to 12 days.

Clinical signs. Chikungunya is characterised by sudden fever, often accompanied by joint pain. Other common signs and symptoms include muscle and headache, nausea, fatigue, and rash. The joint pain is often very severe, but usually goes away after a few days or weeks.

In most cases, patients recover completely, but in some cases, joint pain can last for several months or even years. Some cases of ocular, neurological and cardiac complications, as well as gastrointestinal disorders have been reported. Serious complications are rare, but the disease can be fatal in the elderly. The disease often presents with mild symptoms and the infection may not be detected or misdiagnosed in areas where dengue is prevalent.

Diagnosis. Serological tests, such as enzyme-linked immunosorbent assay (ELISA), can confirm the presence of IgM and IgG antibodies to chikungunya virus. The highest levels of IgM antibodies are reached 3-5 weeks after the onset of the disease and persist for approximately two months.

Specimens collected within the first week of symptom onset should be tested by both serological and virological methods (PCR). Different reverse transcriptase polymerase chain reaction (RT-PCR) methods are available, but the sensitivity of these methods varies. Some of them are suitable for clinical diagnosis.

The PCR products obtained from clinical samples can also be used for virus genotyping, which allows comparison of virus samples from different geographical areas.

Treatment. There are no specific antiviral drugs for the treatment of chikungunya. Treatment is mainly aimed at relieving symptoms, including joint pain, with antipyretics (except aspirin), optimal painkillers and fluids.

Prevention. Prevention and control of the disease is based on reducing the number of natural and artificial water-filled containers that provide habitat and breeding grounds for mosquitoes. During outbreaks of the disease, insecticides can be sprayed to kill flying mosquitoes, surfaces inside and around containers where mosquitoes land can be treated, and water in containers can be treated to kill immature larvae.

To protect yourself during chikungunya outbreaks, it is recommended to wear clothing that covers your skin as much as possible against mosquito bites during the daytime. Exposed skin or clothing can be sprayed with repellents in strict accordance with the instructions contained in the annotations. Repellents should contain DEET, IR3535 or icaridin. Mosquito spirals and other insecticide sprays can also help protect against mosquito bites indoors.

RIFT VALLEY FEVER (RVF) is a viral zoonosis that primarily affects animals but can also infect humans. Infection can lead to severe disease in both animals and humans. The disease also results in significant economic losses due to deaths and abortions amongst RVF-infected livestock.

The RVF virus is a member of the phlebovirus genus. It was first discovered in 1931 during an investigation of an epidemic among sheep on a farm in the Rift Valley, Kenya.

Epidemiology. Most human infections occur as a result of direct or indirect contact with the blood or organs of infected animals.

There is some evidence of the possibility of human infection with BSE when consuming unpasteurised or raw milk from infected animals.

Humans can also be infected by the bites of infected mosquitoes, most commonly Aedes and Culex mosquitoes; transmission of the RVF virus by haematophages (blood-feeding flies) is also possible.

To date, no cases of human-to-human transmission of RVF have been reported. There have also been no reports of transmission of RVF to healthcare workers under standard infection control measures.

Clinic.

Mild form of HRV in humans

- The incubation period of RVF lasts from 2 to 6 days.
- Infected people either do not have any detectable symptoms or develop a mild form of the disease, characterised by a febrile syndrome with sudden onset of flu-like fever, muscle aches, joint pain and headache. Some patients develop neck stiffness (immobility), photosensitivity, loss of appetite and vomiting; in such patients, the disease in its early stages can be mistaken for meningitis.
- Usually, the symptoms of RVF last from four to seven days, after which they can be detected by the body's immune response, which is manifested in the appearance of antibodies and the disappearance of the virus from the blood.

Severe form of RVF in humans

While most human cases are relatively mild, a small proportion of patients develop a much more severe form of the disease. It is usually accompanied by one or more of three distinct syndromes: eye disease (0.5-2% of patients), meningoencephalitis (less than 1%) or haemorrhagic fever (less than 1%).

Ocular form: In this form of the disease, the usual symptoms characteristic of the mild form of the disease are accompanied by damage to the retina. Typically, eye damage occurs one to three weeks after the first symptoms appear. Patients usually complain of blurred or blurred vision. After 10-12 weeks, the disease can go away on its own without any long-term consequences. However, with macular lesions, 50% of patients experience permanent vision loss. Death among patients with the ocular form of the disease alone is rare.

Meningoencephalitis: The onset of meningoencephalitis usually occurs one to four weeks after the first symptoms of RVF. Clinical symptoms include severe headache, memory loss, hallucinations, confusion, disorientation, dizziness, seizures, lethargy and coma. Later (more than 60 days), neurological complications may occur. The mortality rate among patients with this form of the disease alone is low, but residual neurological deficits, which can be severe, are common.

Hemorrhagic form: Symptoms of this form of the disease appear two to four days after the onset of the disease. First, there are signs of severe liver damage, such as jaundice, followed by signs of haemorrhage, such as vomiting blood, blood in the faeces, a red rash or bruising (caused by skin haemorrhage), nose and gum bleeding, menorrhagia and bleeding from venipuncture sites. Mortality among patients who develop the haemorrhagic form of the disease reaches approximately 50%. Death usually occurs three to six days after the onset of symptoms. The virus can be detected in the blood of patients with HRV in the form of haemorrhagic jaundice within 10 days.

Diagnosis. Rift Valley fever can be difficult to distinguish from other viral haemorrhagic fevers, as well as from many other diseases that cause fever, including malaria, shigellosis, typhoid fever and yellow fever.

A definitive diagnosis of an infection caused by Rift Valley fever virus can only be made in the laboratory with the following tests:

- reverse transcription polymerase chain reaction (RT-PCR);
- immunosorbent assay using fixed enzymes to detect IgG and IgM antibodies;
- Isolation of the virus in cell cultures.

Treatment. Given that most cases of people with RVF disease are relatively mild and short-lived, no special treatment is required for these patients. In more severe cases, general supportive care is the preferred treatment.

Vaccination. An inactivated vaccine has been developed for use in humans. However, this vaccine is not licensed and is not commercially available. It is being used for experimental purposes to protect veterinarians and laboratory workers at high risk of infection with PRRS. Other candidate vaccines are being tested.

Prevention. Outbreaks of PRRS in animals can be prevented by sustainable animal vaccination programmes. To prevent epizootics, animals should be immunised before an outbreak occurs. If an outbreak has already occurred, vaccination CANNOT be carried out, as there is a high risk of an outbreak intensifying. Restricting or prohibiting the movement of livestock can be effective in slowing the spread of the virus from infected to uninfected areas. Good hand hygiene, gloves and other appropriate protective clothing should be worn and safety precautions should be taken

when handling sick animals or their tissues and when slaughtering animals. In areas affected by epizootics, all products of animal origin (blood, meat and milk) must be thoroughly cooked before consumption. The importance of using mosquito bite protection at the individual and community level, which should include the use of insecticide-impregnated mosquito nets and, if available, individual repellents, wearing light-coloured clothing (long-sleeved shirts and trousers), and avoiding outdoor activities during the peak vector activity season.

Although no cases of human-to-human transmission of EVD have been identified, there is still a theoretical risk of transmission from infected patients to healthcare workers through contact with infected blood or tissue. Healthcare workers caring for patients with suspected or confirmed EVD should follow standard precautions when handling specimens from these patients.

Standard precautions are recommended for the care and treatment of all patients. They apply to the handling of blood (including dried blood) and all other body fluids, secretions and excretions (except sweat).

MARBURG HAEMORRHAGIC FEVER (MHF) was discovered in 1967 after outbreaks in Marburg and Frankfurt (Germany). The mortality rate is 88%. The EVD virus belongs to the Filoviridae family, just like the Ebola virus.

Epidemiology. Countries in which MHF has been reported:

- Angola
- DR Congo
- Germany
- Kenya
- Serbia
- South Africa
- Uganda

Primary human infection occurs after prolonged exposure to mines or caves inhabited by colonies of Rousettus aegyptiacus bats. The second carrier of the virus is African green monkeys (Cercopithecus aethiops). Pigs have also been shown to be

susceptible to the virus, and are therefore considered as potential host animals during outbreaks. Transmission occurs primarily from person to person through close contact with blood, secretions, organs or other bodily fluids of infected people. Transmission of infection to healthcare workers in the treatment of MHF patients has been reported as a result of close contact in the absence of appropriate infection control measures.

Clinic. The incubation period is from 2 to 21 days.

Initial symptoms:

1. High temperature
2. Severe headache
3. Ailments
4. Crusted rash

On the 3rd day - diarrhoea, abdominal pain and colic, nausea, vomiting.

On the 5th-7th day, there is a haemorrhagic syndrome, which is manifested by bleeding from the nose, intestines, and uterus. The central nervous system is affected - confusion, aggressiveness. In severe cases, death occurs on the 8-9th day.

Diagnosis. Differential diagnosis is carried out with malaria, typhoid fever, shigellosis, cholera, plague.

The final diagnosis can only be determined by the results of laboratory tests, namely:

- enzyme-linked immunosorbent assay (ELISA);
- antigen detection tests;
- Serum neutralisation reaction;
- reverse transcriptase polymerase chain reaction (RT-PCR);
- Isolation of the virus in cell cultures.

Treatment and vaccination. In severe cases of the disease, symptomatic therapy is necessary due to the fact that patients often need intravenous infusions or oral rehydration with solutions containing electrolytes.

To date, there is no specific treatment or vaccine for AML. A number of candidate vaccines are being tested, but they may not become available for several

years. Laboratory studies of new drug therapies are showing encouraging results and are currently being evaluated.

Prevention. Reducing the risk of transmission of infection from bats to humans as a result of prolonged exposure to mines or caves where colonies of fruit-eating bats live.

Reducing the risk of human-to-human transmission in local communities through direct or close contact with infected patients. Close physical contact with patients with AML should be avoided. When caring for sick people at home, gloves and personal protective equipment should be worn. Wash your hands regularly after visiting sick relatives in hospitals and after caring for sick people at home. Inform the local population about the dangers of WNV. People who die of Marburg fever should be buried quickly and safely.

CALIFORNIA ENCEPHALITIS. The California encephalitis virus (CEV) is a member of the Bunyaviridae family. Genetic information is stored on single-stranded RNA. The size of the virus is 90-100 nm in diameter.

Epidemiology. Natural circulation of CEV occurs between *Aedes triseriatus* mosquitoes and vertebrates (chipmunks and squirrels). A person can get this virus after a mosquito bite. This can lead to clinical manifestations or viral carriage.

Clinic. The incubation period of CEV is 5-15 days.

Initial symptoms:

1. Increase in temperature
2. Headache
3. Nausea and vomiting
4. Ailments
5. Depression

Epilepsy occurs during the disease, but not often. In most cases, there is a complete recovery. Less commonly, after recovery, epileptic seizures, hemiparesis, and cognitive impairment are observed.

Diagnosis. To confirm the diagnosis, cerebrospinal fluid and serum are tested for the presence of specific IgM to EBV and neutralising antibodies. In fatal cases, nucleic acid amplification, immunohistochemistry, and viral culture on autopsy tissue may be used to confirm the diagnosis. All tests are performed by specialised and accredited laboratories. Results are available within 4-14 days.

Treatment. No specific treatment for CEV has been developed. Patients are admitted to hospital for symptomatic treatment. No specific vaccine has been developed.

Prevention. The most effective way to prevent an CEV infection is to prevent mosquito bites. Use insect repellent, wear long-sleeved shirts and trousers, treat clothing and equipment, and take steps to control mosquitoes indoors and outdoors.

VENEZUELAN ENCEPHALITIS. Venezuelan encephalitis virus (VEE) is a member of *the Togaviridae* family. Genetic information is stored on single-stranded RNA. The size of the virus is 60-65 nm in diameter. Endemic areas: North America, Caribbean (group I); Central and Southern Africa (groups IIa, IIb, III).

Epidemiology. Natural circulation of VEE occurs between *Culiseta melanura* mosquitoes and birds in deciduous tree swamps. Transmission to humans requires mosquito species that can create a "bridge" between infected birds and uninfected mammals, such as some species of *Aedes*, *Coquillettidia* and *Culex*. Horses are just as susceptible to the virus as humans. However, they are not a source of infection for humans.

Clinic. The incubation period is 4-10 days. There are 3 clinical forms of the disease: systemic infection, carriage or encephalitis. The systemic infection has a sudden onset and is characterised by chills, fever, malaise, arthralgia and myalgia. The disease lasts from 1 to 2 weeks and ends with recovery when there is no CNS damage. In young children, the encephalitis form of the disease is characterised by an abrupt onset.

In older children and adults, encephalitis manifests itself after several days of systemic illness. Signs and symptoms in patients with encephalitis include fever,

headache, irritability, anxiety, drowsiness, anorexia, vomiting, diarrhoea, cyanosis, convulsions, and coma.

Diagnosis. To confirm the diagnosis, cerebrospinal fluid and serum are tested for the presence of specific IgM to VEE and neutralising antibodies. In fatal cases, nucleic acid amplification, immunohistochemistry, and viral culture on autopsy tissue may be used to confirm the diagnosis. All tests are performed by specialised and accredited laboratories. Results are available within 4-14 days.

Treatment. There is no specific treatment for EBV. Patients are admitted to hospital for symptomatic treatment. No specific vaccine has been developed.

Prevention. The most effective way to prevent an VEE infection is to prevent mosquito bites. Use insect repellent, wear long-sleeved shirts and trousers, treat clothing and equipment, and take steps to control mosquitoes indoors and outdoors.

AMERICAN ENCEPHALITIS. The American encephalitis virus (AEV) belongs to the family *Flaviviridae*. Genetic information is stored on single-stranded RNA. The virus is 40 nm in diameter.

Epidemiology. Natural circulation of AEV occurs between *Culex* mosquitoes and wild birds. Occasionally, AEV infects domestic birds. Transmission to humans requires mosquito species that can create a "bridge" between infected birds and uninfected mammals, such as some species of *Cx pipiens*, *Cx quinquefasciatus* and *Cx nigripalpus*.

Clinic. The incubation period is 5-15 days. The onset of the disease is usually abrupt, with fever, headache, dizziness, nausea and malaise. Signs and symptoms increase over several days to a week. Some patients recover spontaneously after this period; others develop signs of a CNS infection, including neck stiffness, confusion, disorientation, dizziness, tremors and unsteadiness. Coma can develop in severe cases.

The disease is generally milder in children than in adults. Around 40% of children and young adults with AEV develop only fever and headache or aseptic meningitis; almost 90% of older people with AEV develop encephalitis.

The overall mortality rate is between 5 and 15%. The risk of fatal disease also increases with age.

Diagnosis. To confirm the diagnosis, cerebrospinal fluid and serum are used for the presence of specific IgM to AEV and neutralising antibodies. In fatal cases, nucleic acid amplification, immunohistochemistry, and viral culture on autopsy tissue may be used for confirmation. All tests are performed by specialised and accredited laboratories. Results are available within 4-14 days.

Treatment. No specific treatment for AEV has been developed. Patients are admitted to hospital for symptomatic treatment. No specific vaccine has been developed.

Prevention. The most effective way to prevent AEV infection is to prevent mosquito bites. Use insect repellent, wear long-sleeved shirts and trousers, treat clothing and equipment, and take steps to control mosquitoes indoors and outdoors.

JAPANESE ENCEPHALITIS. Japanese encephalitis virus (JEV) belongs to the family *Flaviviridae*. Genetic information is stored on single-stranded RNA. The size of the virus is 15-22 nm in diameter.

Epidemiology. Natural circulation of JEV occurs between *Culex tritaeniorhynchus* mosquitoes and pigs and birds. Transmission of JEV occurs primarily in rural agricultural areas, often associated with rice production and flooding. In some parts of Asia, these conditions can occur near urban centres. In temperate areas of Asia, the transmission of JEV is seasonal. Human disease usually peaks in summer and autumn. In the subtropics and tropics, transmission can occur year-round, often with a peak during the rainy season.

Clinic. The incubation period is 5-15 days. Less than 1% of people infected with JEV develop clinical illness. Initial symptoms often include fever, headache, and vomiting. Changes in mental status, neurological symptoms, weakness and movement disorders may develop over the next few days. Epileptic seizures are common, especially among children.

Diagnosis. To confirm the diagnosis, cerebrospinal fluid and serum are used for the presence of specific IgM to JEV and neutralising antibodies. In fatal cases, nucleic acid amplification, immunohistochemistry, and viral culture on autopsy tissue may be used to confirm the diagnosis. All tests are performed by specialised and accredited laboratories. Results are available within 4-14 days.

Treatment. There is no specific treatment for JEV. Patients are admitted to hospital for symptomatic treatment.

Vaccination. Licensed inactivated vaccine against JEV. It can be used from the age of 2 years. Vaccination is carried out in 2 doses according to the schedule: 0-28 days. The last dose is given at least 1 week before travelling. Revaccination is performed in 1 year. People planning to travel to an endemic area for more than 1 month should be vaccinated. People travelling to an endemic area for less than 1 month can be vaccinated. Vaccination is not recommended for travellers with a short stay in an endemic area.

Prevention. The most effective way to prevent an JEV infection is to prevent mosquito bites. Use insect repellent, wear long-sleeved shirts and trousers, treat clothing and equipment, and take steps to control mosquitoes indoors and outdoors.

Presentations:

https://docs.google.com/presentation/d/1XRIncf1j4awFftGw53twrSIQVZMbsZbp/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

https://docs.google.com/presentation/d/1dHz-gGAbu9lpHh0guE20B-iwhgpAIwnS/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

https://docs.google.com/presentation/d/1QDVG9gvXfnxnUkfw2K4E-dsHr6t-on8i/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for students' self-control

1. Name the rickettsiae pathogenic to humans.
2. The main factors of rickettsial pathogenicity.

3. Name the vector of Provachek's rickettsia.

4. Describe the source of infection and vectors in Brill-Zinsser disease.

5. Principles of treatment of relapsing fever

6. Principles of typhoid prevention

7. Epidemic situation with arbovirus infections in the world and Ukraine?

8. What are the latest advances in the diagnosis, treatment and prevention of arboviral infections?

9. Prospects for the development of a prophylactic vaccine and chemoprophylaxis for arbovirus infections?

10. What are the rules for filling out medical records, the algorithm of actions for patients and doctors?

Test control of knowledge

>>What does the term "Arboviral infections" mean?

( ) Infections transmitted only by air

( ) Infections transmitted by animal bites

( ) Waterborne infections

(x) Acronym for arthropoda and borne

( ) Acronym for arthemida and boris

>>What are the clinical forms of arboviral infections: <<

( ) Fevers of unknown origin

( ) Hemorrhagic fevers

( ) Encephalitis

( ) Correct 2, 3

(x) Correct 1, 2, 3

>>How many Dengue serotypes are there?

( ) One

( ) Two

( ) Three

(x) Four

( ) Five

>>Who is the carrier of Dengue fever?

(x) Mosquitoes

( ) Lice

( ) Fleas

( ) Pets

( ) Wild animals

>>What type of Ebola virus caused the largest epidemic outbreak in the world in 2014-2016?

(x) Zaire

( ) Bundibuggio

( ) Sudan

( ) Reston

( ) Tai Forest

>>Who is the natural source of Ebola?

( ) Gophers

( ) Chimpanzees

( ) Rodents

( ) Birds

(x) Fruit bats

>>Ebola virus incubation period: <<

( ) 1-2 days

(x) 2-21 days

( ) 1 month

( ) 60 days

( ) 1 year

>>Who is the source of Lassa haemorrhagic fever in nature?

( ) Gophers

( ) Chimpanzees

(x) Rodents

( ) Birds

( ) Fruit bats

>>Incubation period of Lassa virus: <<

( ) 1-2 days

(x) 6-21 days

( ) 1 month

( ) 60 days

( ) 1 year

>>What specific therapy is used for haemorrhagic fevers (Dengue, Lassa, Ebola)?

( ) Ribavirin

( ) Interferons

( ) Acyclovir

( ) Antibiotics

(x) There is no specific treatment.

Topic 9: Peculiarities of rickettsiosis: Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia. Q fever. Bartonellosis. Ehrlichiosis.

Learning objective: to acquire basic knowledge and skills in clinical manifestations, differential diagnosis, specific examination methods, patient management tactics and basic preventive measures for rickettsiosis: Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, North Asian tick-borne typhus; Q fever, Bartonellosis, Ehrlichiosis.

List of skills:

1. Follow the basic rules of working at the patient's bedside
2. Take a medical history with an assessment of epidemiological data
3. Know the forms and methods of health education work
4. Search for modern literature in the field of study
5. Summarise scientific and practical literature
6. Participate in the dissemination of medical knowledge among the population
7. Draw up a plan for laboratory and additional examination
8. Choose research methods that are adequate to achieve the goal and objectives
9. Interpret the results of different research methods
10. Use modern research methods
11. Determine the medical tactics of patient management
12. Determine the tactics of hospitalisation and isolation of patients
13. Participate in the organisation of basic preventive measures

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Rickettsiosis, Rickettsial Infection, Rocky Mountain spotted fever, Tsutsugamushi fever, Ehrlichiosis, Q fever, North Asian tick-borne typhus, Bartonellosis

ROCKY MOUNTAIN SPOTTED FEVER is an acute endemic rickettsial disease characterised by a generalised intoxication syndrome, fever, exanthem and damage to the nervous and cardiovascular systems.

Etiology. The causative agent of the infection - **Rickettsia rickettsii - is** an intracellular parasite characterised by polymorphism, often having a rod-shaped or lanceolate form. The pathogen is cultivated on the yolk sacs of chicken embryos and by infecting laboratory animals.

Epidemiology. Rocky Mountain spotted fever is a vector-borne zoonosis with natural focal point. The main reservoir is *wild rodents*, rarely *domestic animals* (dogs, cattle). The carrier of infection is Ixodes ticks, transovarial transmission. Mechanism of transmission - transmissible through the bite of infected ticks. Seasonality - *spring and summer* (maximum vector activity).

Pathogenesis. The entrance gate of infection is the bite site, where rickettsiae are initially accumulated, but there is no primary effect. Then, the pathogen enters the bloodstream through the lymphatic system, and parasitemia develops with damage to target cells: vascular endothelium and muscle fibre mesothelium.

In the affected endothelium, swelling and proliferation, cellular infiltration and necrosis with the formation of parietal thrombi are observed. There is perivascular proliferation with the formation of specific cellular infiltrates consisting of macrophages, lymphatic and plasma cells with the occurrence of destructive or destructive-proliferative thrombovasculitis. A thromb-haemorrhagic syndrome develops.

The most pronounced endothelial changes are observed in the myocardium, brain, lungs, adrenal glands, and skin. In severe cases, obturative forms of necrotizing panarteritis are formed, which lead to ischaemic lesions of organs and systems, primarily the brain and myocardium.

Clinical picture. The incubation period is 3-14 days. The disease is characterised by: sudden onset, less often prodromal phenomena for 1-2 days in the form of symptoms of general intoxication; fever from febrile figures to hyperthermia for 2-3 weeks; severe intoxication syndrome with symptoms of toxic CNS damage; 3-4 days (less often 5-6 days), appearance of a spotted rash, which changes to maculopapular rash, localised on the extremities, face, scalp, palms and soles, and to a lesser extent on the abdomen and face. After regression of the rash, a bran-like peeling remains; thrombhemorrhagic syndrome - bleeding, haemorrhagic rash - petechiae throughout the body, which merge to form necrotic areas (fingertips, earlobes, scrotum). After regression, pigmentation remains; enanthema on the conjunctiva and soft palate, tongue; arterial hypertension, bradycardia (tachycardia - severe); hepatosplenomegaly; constipation; kidney damage with proteinuria.

Complications. Pneumonia, phlebitis, nephritis, myocarditis, CNS lesions in the form of hemiplegia, neuritis, visual impairment, deafness, obliterative endarteritis.

Diagnosis. *Serological methods:* RNIF (recommended by WHO); RA with OH_{19} and OH_2 proteins; RHC with a specific antigen (from 2 weeks of the disease); PH. *Biological method* **-** infection of laboratory animals (males) with the development of scrotal necrosis.

Treatment. *Etiotropic therapy:* tetracycline 1.2-2.0 g/day up to 3 days of normal temperature, levomycetin 2.0 g/day up to 3 days of normal temperature

NORTH ASIAN TICK-BORNE TYPHUS is an acute rickettsial disease characterised by a benign course with primary affect, regional lymphadenitis and polymorphic rash.

Etiology. The causative agent of infection **is Rickettsia sibirica,** an intracellular parasite that lives in the cytoplasm and nucleus of affected cells

Epidemiology. Tick-borne typhus of North Asia is a vector-borne zoonosis with natural focal point. The main reservoir is wild rodents. The carrier of infection is ixodid ticks, transovarial transmission. Mechanism of transmission - transmissible

through the bite of infected ticks. Seasonality - spring-summer, September (maximum vector activity).

Pathogenesis. The entry point of the infection is the bite site, where rickettsiae initially accumulate with the development of primary affect. Then, the pathogen enters the bloodstream through the lymphatic system, causing lymphangitis and regional lymphadenitis, and parasitemia develops with damage to target cells: the vascular endothelium. Massive proliferation of rickettsiae occurs in the vascular endothelium, which leads to intoxication, decreased vascular tone, and reactive allergic reactions.

Clinical picture: incubation period - 2-7 days with the formation of primary affect (skin infiltration and compaction with necrosis or ulceration in the centre, covered with a brown crust) and regional lymphadenitis. It is characterized by sudden onset, persistent fever from febrile to hyperthermia for 1-2 weeks, hyperaemia, puffiness of the face, injection of sclerae and conjunctiva, and on days 2-5 (rarely on day 6), abundant polymorphic (rosacea, papules, spots) rash localised on the lateral surface of the trunk, flexural surfaces of the arms, inner thighs, face, neck and buttocks - not draining, on unchanged skin. After regression, pigmentation or bran-like peeling of the skin, symptoms of toxic CNS damage: headache, insomnia, rarely agitation, arterial hypotension, bradycardia (tachycardia - severe), hepatosplenomegaly.

Complications. They are rare and are caused by concomitant pathology or secondary bacterial infection.

Diagnosis. *Serological methods:* ELISA; RPC with specific antigen (from the 2nd week of the disease) 1:40-1:160, RNGA 1:200. *Biological method* - infection of laboratory animals.

Treatment. *Etiotropic therapy:* tetracycline 0.3-0.4 g 4 times/day up to 2 days of normal temperature, doxycycline 0.2 g/day up to 2 days of normal temperature, levomycetin 0.5-0.75 g 4 times/day 2 days of normal temperature

TSUTSUGAMUSHI FEVER is an acute rickettsial disease characterised by the presence of primary affect, lymphadenopathy and maculopapular rash.

Etiology. The causative agent of the infection is Rickettsia tsutsugamushi *(R. orientalis)*, an intracellular parasite of polymorphic form (cocci, rods, diplococci), which parasitises only in the cytoplasm of affected cells.

Epidemiology. vector-borne zoonosis with natural foci.

The main reservoir is *ticks of the genus Trombidiidae,* an additional reservoir is *small rodents, marsupials, insectivores*. Transmission agent - *ticks of the genus Trombidiidae*, transovarian transmission. Mechanism of transmission - transmissible through the bite of infected ticks. Seasonality - *spring-summer, September* (maximum vector activity).

Pathogenesis. The entry gate of infection is the bite site, where rickettsiae initially accumulate with the development of primary affect. Then, the pathogen enters the bloodstream through the lymphatic system, causing lymphangitis, regional or generalised lymphadenitis, and parasitemia develops with damage to target cells: the vascular endothelium. Massive proliferation of rickettsiae occurs in the vascular endothelium, which causes endothelial desquamation with the formation of wall thrombi, intoxication, and severe allergic reorganisation of the body.

Clinical picture. Incubation period **-** 7-20 days, formation of primary affect (skin infiltration and induration with necrosis or ulceration in the centre, covered with a scab, painless) and regional lymphadenitis. It is characterised by sudden onset with chills, persistent or remitting fever from febrile figures to hyperthermia for 3-4 weeks, severe intoxication, pharyngitis, tracheobronchitis, pneumonia, and hyperaemia, puffiness of the face, neck, catarrhal syndrome in the mouth and pharynx, injection of sclerae and conjunctiva with haemorrhages, haemorrhagic conjunctival enanthema of the Chiari Avtsin type, regional lymphadenitis with the development of generalised lymphadenopathy, on the 4th-7th day, a profuse polymorphic rash (spots, rosacea, papules) localised on the chest and abdomen, spreading to the trunk and extremities, possible skin peeling and pigmentation in the presence of a haemorrhagic exanthema, symptoms of toxic CNS damage (severe forms), development of encephalitis, delirium tremens, arterial hypotension, bradycardia (tachycardia - severe), hepatosplenomegaly.

Complications. Encephalitis, pneumonia, hepatitis, nephritis, myocarditis, vascular thrombosis.

Diagnosis. *Serological methods:* RNIF, RA (Weil-Felix) 1:80-1:160 (from the 2nd week of the disease), RPC (from the 2nd week of the disease) 1:20-1:80. *Biological method* **-** infection of laboratory animals.

Treatment. *Etiotropic therapy:* tetracycline 0.3-0.4 g 4 times/day up to 2-3 days of normal temperature, levomycetin 0.5-0.75 g 4 times/day for 2 days of normal temperature.

CU FEVER is an acute natural-focal disease characterised by damage to the mononuclear phagocyte system, polymorphism of the clinical picture with a subacute or chronic course.

Etiology. The causative agent of **Coxiella burneti** infection is an intracellular parasite of polymorphic form (cocci, bacilli), parasitises in target cells with the formation of specific vacuoles, and forms a spore-like form outside the cells. The pathogen is cultivated on cell cultures, chicken embryos, and ticks.

Epidemiology. The source and main reservoir are *small wild mammals, small and cattle, and cargo*. Additional reservoirs are *birds* and *ticks.*

Tick-borne infection, transovarial transmission. Mechanisms of transmission:

- Transmissible through the bite of infected ticks, less commonly ticks, fleas;
- aerosol (in places where infected animals are kept, processing of contaminated raw materials);
- contact (slaughter of cattle, during calving or lambing);
- faecal-oral (waterway, nutritional (milk and dairy products that are not heat-treated, meat), contact and household).

Pathogenesis. The pathogen enters the body by various routes, penetrates the lymphatic system and then enters the systemic bloodstream with the development of parasitemia. Some coxsackieviruses parasitise the cells of the mononuclear phagocyte system (reticulocytes and macrophages), where they actively multiply, while others affect parenchymal organs.

Clinical picture. The incubation period is 3-32 days. It is characterized by sudden onset with severe symptoms of general intoxication, fever from febrile figures to hyperthermia with chills and profuse sweating for 2 weeks, facial hyperaemia, scleral vascular injection, catarrhal syndrome in the oropharynx, tongue covered with grey-white plaque, myocarditis, bronchitis, pneumonia (scarcity of physical findings, involvement of mediastinal lymph nodes and sometimes pleura), abdominal pain of various localisations, paroxysmal, caused by impaired autonomic innervation of the intestine, arterial hypotension, bradycardia, hepatosplenomegaly, very rarely exanthema (rosacea) in combination with arthralgia.

Complications. Secondary bacterial infection - pneumonia, pleurisy, lung abscess, pyelonephritis, thrombophlebitis, pancreatitis, orchitis.

Diagnosis. *Serological methods:* WBC (from the 2nd week of the disease) 1:32-1:256, ELISA - IgM (from the 7th to the 30th day of the disease), IgG, RNIF, RA. *Biological method* **-** infection of laboratory animals.

Treatment. *Etiotropic therapy:* tetracycline 2 g/day for up to 7-10 days, or 0.1 g 3 times/day parenterally for 2-3 days followed by oral administration; fluoroquinolones (ofloxacin, ciprofloxacin); rifampicin.

EHRLICHIOSES are a group of acute vector-borne diseases manifested by fever and generalised intoxication, exanthem and specific blood cell damage.

Etiology. Pathogens belong to the class Rickettsiales, family Anaplasmataceae. They are gram-negative, non-motile intracellular parasites, do not form spores. Among the diversity of Ehrlichiae, at least 4 are currently pathogenic for humans: E. chaffeensis and E. muris - causative agents of monocytic ehrlichiosis; Anaplasma phagocytophilum - causative agent of granulocytic anaplasmosis; E. sennetsu - causative agent of sennets fever.

Ehrlichiae infect mainly leukocyte cells and are found in the cytoplasmic vacuoles of affected cells. The pathogen is cultivated in cell cultures and in white mice.

Epidemiology. The source and main reservoir for monocytic and granulocytic ehrlichiosis are *small wild mammals, small and cattle, cargo*, and an additional

reservoir is *birds* and *ticks.* The infection is transmitted by *ticks*, transovarial transmission. Transmission mechanisms: vector-borne through the bite of infected ticks, less commonly ticks, fleas; aerosol (in places where infected animals are kept, handling of contaminated raw materials); contact (slaughter of livestock, during calving or lambing); faecal-oral (water, nutritional (milk and dairy products that are not heat-treated, meat), household contact).

Pathogenesis. The pathogen enters the body by various routes, penetrates the lymphatic system and then enters the systemic bloodstream with the development of parasitemia. Part of the pathogen parasitises the cells of the mononuclear phagocyte system (reticulocytes and macrophages), where it actively multiplies, while another part affects parenchymal organs. The vascular endothelium is damaged, followed by impaired vascular permeability with the development of haemorrhagic syndrome in the parenchymal organs and skin. The causative agent of monocytic ehrlichiosis E. Chaffeensis can penetrate the blood-brain barrier, causing meningitis.

Clinical picture. The incubation period is 1-21 days. It is characterized by sudden onset with severe symptoms of general intoxication, fever from febrile figures to hyperthermia with chills and profuse sweating for more than 2 weeks, facial flushing, scleral vascular injection, catarrhal syndrome in the oropharynx, tongue covered with grey-white plaque, myocarditis, bronchitis, pneumonia (scarcity of physical findings, involvement of mediastinal lymph nodes and sometimes pleura), abdominal pain of various localisations, paroxysmal, caused by impaired autonomic bowel inertia, arterial hypotension, bradycardia, hepatosplenomegaly, very rarely exanthema (rosacea) in combination with arthralgia.

Complications. Secondary bacterial infection - pneumonia, pleurisy, lung abscess, pyelonephritis, thrombophlebitis, pancreatitis, orchitis. Internal combustion engine syndrome. Acute renal failure.

Diagnosis. *Parasitoscopy (acute phase):* thick blood drop, Romanowski-Gimza stain, detection of pathogens in macrophage and monocyte vacuoles. *Serological methods:* WBC (from the 2nd week of the disease) 1:32-1:256, ELISA - IgM (from the 7th to the 30th day of the disease), IgG, RNIF 1:64-1:80 (fever period

up to 1 year), immunoblotting, PCR. *Biological method* - infection of laboratory animals.

Treatment. *Etiotropic therapy:* tetracycline 2 g/day for up to 7-10 days; doxycycline 0.1 g twice/day for the first day, then 0.1 g/day for 7-10 days; levomycetin succinate; fluoroquinolones (ofloxacin, ciprofloxacin); rifampicin.

BARTONELLOSIS is a group of infectious diseases of humans and animals characterised by polymorphism of clinical manifestations, development of acute and chronic forms.

Bartenellosis includes:

- Disease from cat scratches
- Systemic bartonellosis
- Cutaneous and mucocutaneous bartonellosis
- Other bartonellosis
- Bartonellosis unspecified
- Trench fever

Etiology. Bartonellosis pathogens belong to the genus Bartonella. They are aerobic haematophages, look like short pleomorphic rods. They do not form spores or capsules. Some species have flagella or pili. They have a membrane with surface proteins on its surface and a three-layered shell.

Stained by Romanowski-Gimza, in pathological examination of tissue biopsies - by silvering. The culture medium is chocolate agar.

Epidemiology. The main reservoir for B. bacilliformis is humans. The mechanism of transmission is vector-borne. The vector is mosquitoes of the genus Lutzomia.

For V. quintana, the main reservoir is humans. The transmission mechanism is transmissible. The vector is a clothes louse. For B. Henselae, B. clarridgeiae the main reservoir is cats. The mechanism of transmission is contact. For other Bartonellae, the main reservoir can be cats, dogs, rodents, ticks. The most common mechanism of transmission is vector-borne through flea and tick bites.

Pathogenesis.

Disease from cat scratches

Immunocompetent patients: the pathogen penetrates the skin or mucous membranes with subsequent multiplication and accumulation, resulting in a primary effect in the form of a local reaction. The bacteria then enter the lymphatic vessels, where regional lymphadenitis develops.

Immunocompromised patients: the pathogen penetrates the skin or mucous membranes with subsequent multiplication and accumulation, resulting in a primary effect in the form of a local reaction. Then the bacteria penetrate the lymphatic and blood vessels, hematogenous and lymphogenous dissemination occurs, resulting in polyadenopathy and damage to parenchymal organs.

Oroya fever and trench fever

There is no primary affect. The primary accumulation and multiplication of the pathogen occurs in the skin. Subsequently, the bacteria penetrate the lymphatic and blood vessels, and haematogenous and lymphogenous dissemination occurs. Bartonellae have tropism for the endothelium of microcirculatory vessels, damaging it, resulting in circulatory disorders and hypoxia, which are clinically manifested by generalised intoxication syndrome. Re-entry of Bartonella into the bloodstream leads to red blood cell damage and inhibition of leukocyte apoptosis.

Chronic forms of bartonellosis

It develops in immunocompromised patients. Due to the intracellular localisation of most pathogens, prolonged bacteremia is observed

Bartonellosis caused by V. Henselae and V. quintana

The pathogen damages the endothelium of microcirculatory vessels, causing endothelial cell proliferation and angioproliferation (in immunocompromised individuals), resulting in the growth of granulation tissue.

At the same time, V. Henselae affects mainly the skin, lymphatic system of the liver and spleen, while V. quintana affects the skin, subcutaneous tissue and bone tissue.

Carrion's disease is caused by B. bacilliformis and manifests itself as an acute form (Oroya fever) and chronic (Peruvian wart).

Acute form (Oroya fever): incubation period - 10-21 days, gradual onset with fever from subfebrile to febrile figures, which subsequently rises to hyperthermia, fever is accompanied by symptoms of intoxication, chills, profuse sweating, haemorrhagic syndrome on the skin, polyadenopathy, hepatosplenomegaly, anaemic syndrome (pale skin, weakness, shortness of breath), significant decrease in haemoglobin and erythrocytes by 20-30%.

Chronic form (Peruvian wart) - develops 1-6 months after the acute form, rash: red-purple macules - papules - nodules on the skin, subcutaneous tissue, conjunctiva, mucous membranes, recurrent fever, haemorrhagic syndrome, anaemic syndrome in the blood.

Trench (Volyn) fever is caused by V. quintana.

The incubation period is 10-30 days. It is characterised by an acute onset with fever from febrile to hyperthermia, which lasts up to 5 days and is accompanied by chills and symptoms of general intoxication. Fever of two waves, conjunctival injection, rosaceous or macular rash, hepatosplenomegaly, anaemic syndrome.

Cat scratch disease (benign lymphoreticulosis)

It is caused by B. Henselae and B. clarridgeiae. The incubation period is from 3 to 60 days. *Typical variant:* formation of a primary lesion at the scratch or bite site in the form of an ulcer or pustule, regional lymphadenitis, fever and symptoms of intoxication. *Atypical oculoglandular variant (Parino syndrome):* infection occurs through the conjunctiva, unilateral conjunctivitis, parotid lymphadenitis, severe fever and symptoms of intoxication, development of red-yellow nodules on the conjunctiva of the eyelids.

Neurological variant: most often develops after the typical and atypical variants. It occurs in the form of meningitis, meningoencephalitis, encephalomyelitis, encephalopathy, mental status changes, severe cephalgic syndrome, fever, positive meningeal signs, and less commonly, seizures and focal symptoms. *Systemic variant:* lung damage (pneumonia, bronchitis, abscess), liver damage (granulomatous hepatitis), spleen damage (abscess), kidney damage (glomerulonephritis), joint damage (arthritis), bone disorders (osteomyelitis, osteitis, periostitis), prolonged fever,

lymphadenopathy, hepatosplenomegaly, haemolytic anaemia, thrombocytopenic or nonthrombocytopenic purpura, weight loss.

Bacillary angiomatosis is a rapidly progressive disease characterised by proliferative vascular lesions involving many organs and systems. *The cutaneous variant is characterised by* fever, generalised intoxication syndrome, exanthema in the form of a red-violet papule that grows to a node or rises on a pedicle above the skin surface. Over time, erosion with a haemorrhagic crust or necrosis forms on the surface; the nodes can also be located subcutaneously, single or grouped, and can eat away at the skin and become infected. *Bone variant* - fever, generalised intoxication syndrome, primarily long tubular bones are affected, but any bone tissue can be involved in the pathological process, as well as bone marrow, lesions are painful, hepatosplenomegaly and thrombocytopenia in bone marrow involvement.

Gastrointestinal variant - fever, generalised intoxication, gastric, intestinal, oral or anal mucosal lesions, regional lymphadenopathy. *Respiratory variant* - fever, generalised intoxication syndrome, tracheal and bronchial lesions, regional lymphadenitis or generalised lymphadenopathy. *Neurological variant* - fever, generalised intoxication syndrome, brain, cranial nerves or peripheral nerve damage.

Bacillary peliosis. The pathogen leads to the formation of multiple cysts filled with blood. It develops mainly in the system of macrophage phagocytes in the liver, spleen, abdominal lymph nodes, bone marrow, kidneys, adrenal glands, pancreas, lungs, and gastrointestinal tract with the development of dysfunction of the respective organs.

Chronic bacteremia. Most often it is asymptomatic with a negative bacteriological blood test. In some patients, it manifests itself as fever and symptoms of intoxication with exanthema, lymphadenopathy, hepatosplenomegaly, changes in the haemogram in the form of leukocytosis, thrombocytopenia.

Septic endocarditis. It is most often asymptomatic. In some patients, it develops gradually and is characterised by typical clinical and echocardiographic changes that are typical of heart valve disease of other bacterial etiologies.

Diagnosis. Biopsy with histological examination of the affected organs and systems. Serological methods: ELISA (detection of antibodies to Bartonella), ELISA (detection of antibodies in blood serum or cerebrospinal fluid). Culture method. The optimal method is the isolation of Bartonella from blood, rarely from other infected tissues, using a nutrient medium - chocolate agar. PCR with detection of Bartonella DNA.

Treatment. Etiotropic therapy: azithromycin (in case of significant lymphadenopathy), doxycycline, gentamicin, rimfapicin.

Presentation:

https://docs.google.com/presentation/d/13aL2o_wn2JkHnxGWcPgjo7B8PBgpD-DC/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Questions for self-monitoring

1. Etiology of rickettsioses (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
2. Epidemiology of rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
3. Pathogenesis of rickettsioses (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
4. Clinical course of rickettsioses (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
5. Laboratory diagnostics of rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.

6. Differential diagnosis of rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
7. Complications of rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
8. Treatment of rickettsioses (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Cu fever, Bartonellosis, Ehrlichiosis.
9. Prevention of rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, Bartonellosis, Ehrlichiosis.
10. The procedure for hospitalisation, rules for discharge of patients with rickettsiosis (Rocky Mountain spotted fever, tsutsugamushi, endemic typhus, tick-borne typhus of North Asia), Q fever, bartonellosis, ehrlichiosis.

Glossary

Rickettsiosis, Rickettsial Infection) is a group of vector-borne infectious diseases caused by intracellular parasites - rickettsiae - and characterised by a number of common pathogenetic, clinical and immunological properties.

Rocky Mountain spotted fever is an acute rickettsial disease characterised by symptoms of generalised intoxication and the appearance of a profuse maculopapular exanthema, which becomes haemorrhagic in severe disease.

Marseille fever is an acute rickettsial disease characterised by a benign course, primary affect, widespread maculopapular rash, fever and joint pain

Tsutsugamushi fever is an acute rickettsial disease characterised by the presence of primary affect, lymphadenopathy and maculopapular rash.

Ehrlichiosis is a group of acute vector-borne diseases manifested by fever and generalised intoxication, exanthema and specific blood cell damage.

Q fever is an acute natural-focal disease characterised by damage to the mononuclear phagocyte system, polymorphism of the clinical picture with a subacute or chronic course.

North Asian tick-borne typhus is an acute rickettsial disease characterised by a benign course with primary affect, regional lymphadenitis and polymorphic rash.

Bartonellosis is a group of infectious diseases of humans and animals characterised by polymorphism of clinical manifestations, development of acute and chronic forms. Bartonellosis includes: Cat scratching disease, Systemic bartonellosis, Cutaneous and mucocutaneous bartonellosis, Other bartonellosis, Bartonellosis unspecified, Trench fever.

Test control of knowledge

>>Rocky Mountain spotted fever is transmitted by bites from infected people

( ) midges

( ) mosquitoes

( ) lice

(x) mites

( ) horseflies

>>In which rickettsiosis is the primary affect combined with a maculopapular rash and lymphadenopathy?

( ) Rocky Mountain fever

( ) Ku fever

( ) endemic typhus fever

( ) epidemic typhus fever

(x) tsutsugamushi fever

>>Transient deafness develops when

( ) Rocky Mountain fever

(x) endemic typhus fever

( ) Ku fever

( ) tsutsugamushi fever

( ) Brill's disease

>> Antibacterial drugs of which group are the choice for the treatment of tick-borne typhus of North Asia are: <<

(x) tetracycline

( ) cephalosporin

( ) macrolides

( ) fluoroquinolones

( ) penicillin

>>The main method of diagnosing Q fever <<

(x) serological

( ) PCR

( ) microscopic

( ) bacteriological

( ) biological

>> Emergency antibiotic prophylaxis of Q fever is carried out: <<

( ) cefazolin

( ) penicillin

( ) with male fern extract

(x) doxycycline

( ) albendazole

>> Hot tsutsugamushi is transferred <<

( ) damselflies

(x) mites

( ) mosquitoes

( ) lice

( ) midges

>>The main method of diagnosing bartonellosis

( ) PCR

(x) biopsy with histological examination

( ) serological

( ) bacteriological

( ) biological

>>Which disease is characterised by fever, intoxication, cephalgia, myalgia, arthralgia, rash and specific blood cell damage: <<

( ) wucheriosis

( ) onchocerciasis

( ) bruchiosis

( ) Lyme disease

(x) ehrlichiosis

>> Infection with ehrlichiosis occurs: <<

( ) airborne droplets

( ) haemocontact

(x) by transmission

( ) faecal-oral

( ) transplacental

Topic 10. Infectious diseases included in the list of events that may constitute a public health emergency on an international scale and fall under the International Health Regulations 2005. Pneumonic plague, yellow fever, West Nile fever.

Learning objective: to gain basic knowledge of what to do when detecting especially dangerous (quarantine) infections.

List of skills

1. Be able to justify a preliminary clinical diagnosis of the most common tropical infectious diseases and parasitic infestations.
2. Be able to recognise complications and emergencies in patients with the most common tropical infectious diseases and parasitic infestations.
3. Be able to prescribe a plan for the examination of patients with the most common tropical infectious diseases and parasitic infestations.
4. Perform clinical and laboratory differential diagnosis of tropical infectious diseases and parasitic infestations.
5. Prescribe rational treatment for patients with tropical infectious diseases and parasitic infestations at different stages of medical care.
6. Be able to provide emergency care to patients with tropical infectious diseases and parasitic infestations.

Hardware and software:

1. A personal computer, laptop, tablet, smartphone or similar device,
2. Operating system Microsoft Windows 8/8.1/10/11, Linux, MacOS or similar.
3. Browser Google Chrome, Microsoft EDGE, Opera, FireFox, Safari or similar.
4. Access to the global network, local network,
5. Access to the electronic library of online courses of ZSMFU.

Glossary of terms: Pneumonic plague, yellow fever, West Nile fever, international health regulations

The relevance of studying this topic lies in the fact that plague and contagious haemorrhagic fevers occur in areas where there are natural foci of these infections. These are mainly the African continent, the Americas and Asia. Having gained independence, Ukraine has expanded its ties with many countries around the world, and the threat of importing highly dangerous infections (HDI) has increased significantly. Unfortunately, the factor of the possible use of bacteriological weapons, which mainly include EIDs and quarantine agents, has not yet been removed.

Particularly dangerous (quarantine) infections are a group of diseases that are subject to quarantine measures in accordance with international health regulations. This group includes plague, cholera, and contagious viral haemorrhagic fevers. The importance of studying these diseases is due to the presence of some quarantine infectious diseases in Ukraine (Congo-Crimean fever), as well as the possibility of importing OVD into Ukraine.

PLAGUE is a particularly dangerous, acute, naturally occurring, zoonotic bacterial infection with multiple routes of transmission, characterised by a febrile intoxication syndrome with predominantly affected lymph nodes, lungs and other organs.

Etiology. The causative agent of the plague is Yersinia pestis, a gram-negative bacillus, does not form spores, is stationary, bipolar in colour, grows well but slowly on simple nutrient media; its optimum growth temperature is 28°C. It has 3 biotypes: antigua, orientalis, mediaevalis. The pathogen is a facultative intracellular parasite. The high virulence of the plague pathogen is determined by V and W antigens, which provide resistance to intracellular phagocytic destruction. Pathogenicity factors include endo- and exotoxins, a number of enzymes: hyaluronidase, coagulase, haemolysin, fibrinolysin, and others.

Epidemiology. Plague is a naturally occurring focal zoonotic anthropogenic disease. There are natural ("wild plague") and anthropurgic foci of plague. Natural foci occupy about 8% of the world's land area. Active enzootic foci remain in Southeast Asia. The main sources of "wild plague" are rodents, gophers, voles, marmots, gerbils,

tarbans, etc. In addition to natural foci of plague, anthropogenic (urban, port) plague foci can form. The main source of infection in anthropic foci is black and grey rats, cats, camels and other animals. Humans can be an additional source of infection.

The infection can be transmitted to a healthy person in various ways: vector-borne, contact, nutritional, and airborne.

Human susceptibility to plague is very high. The incidence rate is close to one.

Pathogenesis. The ability of the pathogen to suppress the phagocytic activity of neutrophils and macrophages with the development of the phenomenon of incomplete phagocytosis is important, which complicates the launch of the immune response. The disease is accompanied by severe toxemia. The toxins affect the central nervous system, causing severe neurotoxicosis, and the cardiovascular system with the development of acute cardiovascular failure. They cause disturbances in the haemostatic system with the development of thrombo-haemorrhagic syndrome.

Clinical manifestations. Clinical manifestations include localised and generalised forms. Localised forms include: cutaneous, bubonic and cutaneous-bubonic. Generalised forms include: primary and secondary septic forms; primary and secondary pulmonary forms.

The incubation period lasts from several hours to 6 days. In vaccinated individuals, the incubation period is extended to 10 days. In all forms, the disease begins acutely. Patients can indicate not only the day but also the hour of the disease onset. Body temperature with severe chills, rising to 39.5 - 40 °C or more. Headache, dizziness, musculoskeletal pain, weakness, and sometimes vomiting are typical. Appearance of the patient: the face is flushed and swollen. In the most severe cases, cyanosis of the face occurs, facial features become sharpened, and an expression of fear, hopelessness, and horror ("plague mask") appears. The conjunctiva and sclera are injected, and there are often pinpoint haemorrhages on the transitional fold. The tongue is dry, thickened, trembling, covered with a thick white coating ("chalky"), the mucous membranes of the oropharynx are hyperaemic. From the first days of the disease, signs of central nervous system and cardiovascular system damage appear.

Some patients develop agitation, frightening hallucinations, impaired coordination of movements ("drunken gait"), and muscle tremors. Others have lethargy, deafness. Shortness of breath. The boundaries of the heart are dilated, heart sounds are deaf. Tachycardia up to 120 - 140 per minute, pulse arrhythmia, progressive drop in blood pressure.

In the cutaneous form of plague, a spot first appears at the site of the pathogen's penetration, followed by a papule, vesicle, and pustule filled with serous and haemorrhagic contents. The pustule is located on a solid red-purple base and is characterised by significant pain. When the pustule is opened, an ulcer is formed, the bottom of which is covered with a dark scab. Ulcers heal slowly, forming a scar.

In the tambourine form, there are no local skin lesions. The most common are inguinal buboes. The first sign of a tubercle is the appearance of severe pain and local tenderness in the area where the tubercle forms. Because of the pain, patients take a forced position (bent leg, arm outstretched). In 2-3 days, the bubo is formed. The tubercle is painful with indistinct contours, a dense conglomerate of several nodes fused to the subcutaneous tissue, and inactive. The skin over the tubercle is purple-red, cyanotic. The size of the tubercle can reach 10 cm. Lymphangitis is not observed. At the end of the tubercle formation stage, the phase of its resolution begins. On the 6-8th day, the bubo is opened, and thick pus containing plague pathogens is discharged. Large ulcers may form at the site of the buboes. Healing is slow. After opening the buboes, the general condition of patients improves. Antibiotics are prescribed to prevent suppuration. The tubercle either resolves or scleroses. Especially dangerous are inguinal buboes, as in these cases secondary pulmonary plague often develops.

The initially septic form of plague is extremely severe. The incubation period can be reduced to 2 days. In most patients, on the first day of illness, signs of cardiovascular failure begin to progress, oliguria, anuria, and thrombhemorrhagic syndrome rapidly develop. Against the background of severe toxicosis, large, confluent crimson-black haemorrhages ("black death") appear on the skin. Hemorrhages on the mucous membranes. Nosebleeds. Cases of recovery are

extremely rare. The clinic of the secondary-septic form is similar, but develops against the background of the bubonic form.

The primary pulmonary form of plague is also extremely severe. Against the background of neurotoxicosis, on the first day of illness, there is a stabbing pain in the chest, cough, and severe shortness of breath. At the beginning, the sputum is clear, viscous, glassy, on the 2nd day it becomes liquid, foamy, bloody and, finally, bloody. The act of breathing involves the work of the auxiliary muscles, the wings of the nose. Breathing becomes harsh. The physical findings are very poor and do not correspond to the serious condition of the patients. There is a slight shortening of the sound over the affected lobe, and rales. Interstitial and alveolar pulmonary edema develops, which is based on toxic damage to the lung capillaries with a sharp increase in their permeability. The sputum contains a huge number of plague bacilli. In the absence of adequate etiological therapy, patients die within 3-4 days from cardiovascular and respiratory failure. The clinical picture of secondary pulmonary plague, which complicates the course of the bubonic form of the disease, is similar, but a sudden deterioration occurs several days after the onset of the disease. The course of the disease is more favourable compared to that of the primary pulmonary form.

Plague in vaccinated individuals is characterised by a prolonged incubation period of up to 10 days. Fever is subfebrile, symptoms of intoxication are not pronounced. The tambourine is small, very painful, but without pronounced manifestations of periadenitis. In the absence of timely adequate therapy in the first 3-4 days, further development of the disease will not differ from that of unvaccinated patients.

Laboratory diagnostics. The most important are bacteriological and bacterioscopic methods of examination. The causative agent can be isolated from the bubonic point, skin elements, blood, sputum, as well as from cadaveric material (pieces of internal organs, blood, bone marrow, etc.). Laboratories should operate in accordance with the instructions on the mode of operation of anti-plague institutions. The detection of typical bipolar bacteria during bacterioscopy within 1 hour allows

for a preliminary diagnosis of plague. It takes 5-7 days to obtain a pure culture and identify it.

The biological test method increases the likelihood of isolating the plague microbe. Guinea pigs and white mice are used. Among the serological methods used are RPC, RIGA, and REMA.

Differential diagnosis. The pulmonary form of plague is differentiated from croup pneumonia. Particularly difficult to diagnose are cases of croup pneumonia when, instead of rusty sputum, little changed blood is produced during coughing. In croup pneumonia, there is no epidemiological history characteristic of plague (staying in plague foci), no pronounced percussion and auscultatory objective findings. Signs of neurotoxicosis in plague pneumonia are more pronounced and occur much earlier.

Complications. Infectious toxic shock, acute cardiovascular failure, acute respiratory failure.

Treatment. At the pre-hospital stage, patients with severe intoxication should receive emergency care. All drugs should be administered against the background of infusion therapy (lactosol, rheopolyglucin, etc.). Corticosteroids are administered at the rate of prednisolone of at least 5 mg/kg of patient weight.

Etiotropic treatment includes the administration of antibiotics. The drugs of choice are aminoglycosides (streptomycin, gentamicin), levomycetin (chloramphenicol), tetracycline drugs and fluoroquinolones (ciprofloxacin, ofloxacin). Depending on the clinical form of plague, streptomycin is prescribed at a dose of 2 to 4.5 g per day (in localised forms, 1 g every 12 hours, in generalised forms, 1.5 g every 8 hours). The administration of large doses of bacteriological drugs can lead to massive bacteriolysis and the development of endotoxic shock. Etiotropic therapy should be started with intravenous administration of a bacteriostatic antibiotic - chloramphenicol. Levomycetin is prescribed in a daily dose of 6 g with a dose reduction after temperature normalisation. Tetracycline is prescribed in a daily dose of 4-6 g per day. Ciprofloxacin in the bubonic form - 0.5 g orally every 12 hours, in generalised forms - 0.4 g every 12 hours intravenously. The course of antibiotic therapy is 7-10 days.

Along with antibacterial treatment, detoxification therapy is carried out.

Crystalloid and colloidal solutions are used in a ratio of 3:1. The daily volume of fluid should be at least 40 mg/kg body weight. Glucocorticosteroids. According to indications, anti-shock therapy, correction of respiratory failure, DIC syndrome, control of pulmonary edema, cerebral edema and swelling are performed.

Prevention. Preventive measures are aimed at preventing the importation of the infectious agent from abroad, reducing the epizootic activity of natural foci and preventing human disease in these foci. According to epidemiological indications, preventive vaccination is carried out in plague foci, primarily for people at high risk of infection (geologists, shepherds, employees of anti-plague institutions). It is necessary to constantly conduct sanitary and educational work among the population. These measures are stipulated by the special International Sanitary Regulations and the Rules for the Sanitary Protection of the Territory. If plague patients are detected, an emergency anti-epidemic commission is created, which imposes quarantine and draws up a comprehensive plan for the elimination of the outbreak.

Measures in the epidemic centre

I. Measures for patients. Patients with plague are subject to mandatory hospitalisation in a plague hospital. Convalescents are discharged after full clinical recovery. Patients with the bubonic form of plague are discharged no earlier than 4 weeks from the date of clinical recovery after a 2-fold negative result of bacteriological examination of the bubonic puncture. Patients with the generalised form are discharged no earlier than 6 weeks from the date of clinical recovery after multiple negative results of sputum bacteriological examination.

After discharge, medical monitoring is mandatory for 3 months.

II. Measures for contact persons. A healthcare worker who has detected a plague patient or a patient with suspected plague, without leaving the premises (before the arrival of the evacuation team) where the patient was detected, by telephone or through a person who has not been in contact with the patient, notifies the chief physician of the institution and the chief physician of the relevant Sanitary and Epidemiological Station about the detected patient. Medical personnel and other persons who have been in direct contact with the patients are isolated in a hospital for

6 days. For the purpose of early detection of patients, contact persons are closely monitored with twice-daily thermometry. For emergency prophylaxis, one of the following antibiotics is used: vibromycin 1 capsule per day, doxycycline 0.1 g twice daily, streptomycin 0.5 g twice daily intramuscularly. After the patient is hospitalised, final disinfection is carried out in the area.

III. Measures against the focal point. Disinfection in plague foci is carried out by disinfection teams.

YELLOW FEVER (Febris flava) (synonyms: Yellow fever - English; Gelbfieber - German; Fievre jaune - French; Fiebre amarilla, Vomito negro - Spanish) is an acute arboviral disease transmitted by mosquitoes, characterised by fever, severe intoxication, thromb-haemorrhagic syndrome, kidney and liver damage.

Etiology. The causative agent is Viscerophilus tropicus virus, belongs to the family Togoviridae, genus Flavivirus, contains RNA, is an arbovirus of antigenic group B. It has antigenic similarity to Japanese encephalitis, Dengue and St Louis encephalitis viruses.

Epidemiology. Yellow fever is a quarantine disease. The endemic foci are large areas of South America (Bolivia, Brazil, Colombia, Ecuador, Peru, etc.) and equatorial Africa. The source and reservoir of infection are wild animals (monkeys, opossums, and occasionally other species), as well as sick people. The carriers are mosquitoes. There are 2 types of yellow fever: 1) urban (anthropogenic) and 2) rural (jungle yellow fever). In the anthropogenic type, infection by a mosquito (Aedes aegypti) occurs during a bite of a sick person at the end of the incubation period or in the first 3 days of the disease. In the rural type of yellow fever, the source of infection is monkeys, and the mosquito vector is Aedes aficanus, Aedes simpsoni.

Pathogenesis. The virus enters the human body when bitten by an infected mosquito. There are cases of laboratory infections by aerogenous means. From the site of invasion, the pathogen spreads through the lymphatic system and reaches regional lymph nodes, where it multiplies and accumulates. A few days later, the virus enters the bloodstream, where it can be found for 3-5 days. By haematogenous route, the virus

penetrates various organs (liver, spleen, kidneys, bone marrow, lymph nodes), causing damage to them. A thromb-haemorrhagic syndrome develops, which manifests itself in the form of multiple haemorrhages in various organs. The liver is enlarged, necrotic liver cells are located in small foci. These focal homogeneous eosinophilic bodies found in the perisinusoidal spaces of the liver are called Crownsilver bodies. Fatty degeneration of hepatocytes is noted in the central areas of the liver lobules. Liver damage leads to severe jaundice. Changes are detected in the kidneys (edema, haemorrhage, necrosis of the renal tubules), spleen, myocardium, and lymph nodes. After the disease, the patient develops a strong immunity that lasts for 6-8 years.

Symptoms and course. The incubation period ranges from 3 to 6 days. There are 3 periods in the clinical course of yellow fever:

- initial febrile period (hyperaemia stage);
- a period of remission;
- reactive period (stasis stage).

In severe forms of the disease, there may be no remission period.

The disease begins suddenly with the onset of severe headache, severe pain in the lower back, back, and extremities. By the end of the 1st day, the body temperature reaches 39-40 °C and higher. There is hyperaemia and puffiness of the face, swelling of the eyelids, injection of scleral and conjunctival vessels. The pulse rate increases to 100-130 per 1 min. On the 2nd day, the patient's condition worsens, painful thirst, nausea, repeated vomiting of mucus and then bile join the symptoms described above. The mucous membrane of the oral cavity is hyperaemic, the tongue is dry, and the edges of the tongue are reddened. By the end of the first period (3-4th day of illness), cyanosis, jaundice, and minor blood impurities in the vomit may appear. On the 4th-5th day of the disease, the patient's health improves, the body temperature decreases to subfebrile (remission stage). However, after a few hours, the temperature rises again, the patient's condition progressively deteriorates - the reactive period begins. A thromb-haemorrhagic syndrome develops in the form of bloody vomiting, bleeding from the nose, intestines, uterus, petechiae and large haemorrhages appear on the skin. The patient's face becomes pale. The pulse is rare (up to 50-40 beats per minute), does not

correspond to fever (Faget's symptom), blood pressure decreases, the amount of urine decreases, and sometimes anuria develops. The urine contains a large amount of protein, cylinders. Weakness increases, delirium appears. In severe cases, death occurs from renal failure or infectious collapse (infectious toxic shock). With a positive result, the patient's condition gradually improves from day 7-9. In mild cases, the symptoms of the disease are mild, jaundice and thrombohemorrhagic syndrome may not occur. In the presence of very severe forms, patients may die on the 2-3rd day of the disease even before the development of jaundice (lightning forms).

Complications - pneumonia, myocarditis, gangrene of soft tissues or extremities, sepsis as a result of the layering of secondary bacterial microflora.

Diagnosis and differential diagnosis. Recognition of yellow fever is based on epidemiological prerequisites (stay in an endemic area, incidence of yellow fever, etc.) and clinical data. The following laboratory tests are diagnostic: leukaemia, neutropenia, detection of protein and cylinders in the urine, as well as an increase in serum bilirubin, residual nitrogen and a significant increase in serum aminotransferase activity. Characteristic changes are detected in histological examination of liver biopsies. Serological methods used are the PCR, the neutralisation reaction and the RGGA, but the latter often gives positive reactions with other viruses. The test is performed with paired sera.

Currently, **virus isolation** from blood is used by inoculating material into mosquito cell culture or by injecting material into the thoracic cavity of mosquitoes. The likelihood of virus isolation is higher if the material is taken in the first 3 days of the disease. Serological methods include the plaque suppression reaction with paired sera and the detection of IgM antibodies to yellow fever virus and virus antigens by enzyme-linked immunosorbent assay. The latter method allows confirming the diagnosis within 3 hours.

It is necessary to differentiate yellow fever from dengue, pappatachi fever, leptospirosis, other haemorrhagic fevers and viral hepatitis.

WEST NILE FEVER is an acute infectious disease of viral etiology that presents with polyadenitis, skin rash, and sometimes with symptoms of serous meningoencephalitis.

Etiology and Epidemiology. West Nile Virus belongs to the genus Flavivirus, and its antigenic structure is similar to the Japanese encephalitis virus, Dengue.

In Egypt, Israel and France, the incidence has a pronounced summer-autumn seasonality. Depending on climatic differences, cases are recorded from May-June to August-September. In tropical areas, the seasonality factor is less important in the epidemiology of this disease.

West Nile virus is transmitted by mosquitoes. Birds, representatives of the aquatic and near-water ecological complex serve as a reservoir of the virus.

Pathogenesis. The virus enters the bloodstream through the skin when a mosquito bites. This is followed by haematogenous dissemination of the pathogen with virulence, with systemic lymphatic tissue damage, manifested by the development of lymphadenopathy. In some cases, the virus penetrates the blood-brain barrier and infects the meninges and brain matter.

Clinic. The incubation period lasts 2-8 days. The disease begins acutely, without prodrome. High fever, generalised weakness, chills, and pain in the eyeballs are manifested. The duration of the temperature period ranges from 3 to 12 days, and the temperature decreases according to the type of accelerated lysis. In some patients, there is a repeated increase in temperature. In some cases, the disease occurs only with the listed general infectious symptoms and is difficult to differentiate from various commonplace, including respiratory, diseases. The similarity with the latter is enhanced by scleritis, conjunctivitis, bright hyperaemia of the pharyngeal rims, and sometimes minor catarrhal phenomena (cough, sore throat). However, the disease can also occur in other clinical forms with nausea, vomiting, abdominal pain, loose stools, enlarged liver and spleen. K.Marberg et al. (1956) noted the following frequency of symptoms: conjunctivitis - 60%; hyperaemia of the posterior pharyngeal wall and pharyngeal rims - 40%; splenic enlargement - 20%; liver enlargement - 10%; nausea - 25%; vomiting - 10%; abdominal pain - 20%; loose stools - 30%.

Typical symptoms of West Nile fever also include skin rashes and polyadenitis. The rash has a diverse character - roseola-like, polymorphic-spotted, scarlet fever-like, etc. It appears on the 2nd-4th day of the disease, mainly on the upper half of the body, and disappears in a few days, leaving no pigmentation. In some outbreaks, the rash occurs in 50% of patients. Polyadenitis is one of the most common symptoms of the disease and is manifested by an increase in all groups of lymph nodes, including the cervical and occipital. Sometimes they reach the size of a walnut and are painful to palpate. Polyadenitis is observed for a long time - from 2 weeks to 1 - 1 ½ months. Some patients have serous meningitis or meningoencephalitis. The development of serous meningitis or meningoencephalitis is accompanied by a sharp headache, vomiting, hyperesthesia, and lethargy. Meningeal symptoms may be very mild or even absent.

In meningoencephalitis, these symptoms are accompanied by signs of focal nervous system damage (anisoreflexia, pyramidal and extrapyramidal signs, horizontal nystagmus), which are subject to rapid reversal during the period of recuperation. severe forms of encephalitis with paresis, paralysis and death are occasionally observed. In surviving patients, paresis and paralysis reverse. Changes in peripheral blood are quite variable, with leukaemia with a left shift in the formula being more common.

The cerebrospinal fluid in serous meningitis and meningoencephalitis is clear, flows under increased pressure and contains an increased number of cells, mainly lymphocytes. The protein content is normal or slightly reduced. The cerebrospinal fluid returns to normal within 2-3 weeks.

Diagnosis and differential diagnosis. The combination of fever, polyadenitis, and skin exanthem is of the greatest diagnostic value. The question of the presence or absence of an inflammatory process in the meninges can only be resolved by examining the cerebrospinal fluid. Indications for a spinal tap are intense headache and repeated vomiting even in the absence of meningeal symptoms.

The methods of virological and serological examination of patients with West Nile fever are similar to those used for the specific laboratory diagnosis of Japanese encephalitis (infection of newborn white mice, RGGA, RBC, neutralisation reaction).

The differential diagnosis is made with enterovirus diseases, leptospirosis, and the classic form of Dengue fever. The latter is the most difficult to distinguish from West Nile fever. The diagnosis is facilitated by the occurrence of serous meningitis and meningoencephalitis, which are not typical for Dengue fever.

Treatment is symptomatic. Diuretics and dehydrating agents (diacarb, furosemide or lasix) are prescribed for symptoms of nervous system damage. Relief from severe headache and vomiting is provided by spinal puncture due to reduction of intracranial pressure.

The prognosis is favourable. The exception is the rare cases of severe meningoencephalitis, which are similar to Japanese encephalitis and St Louis encephalitis and usually occur in elderly people with a low immune response.

Prevention. There is no specific vaccine or seroprophylaxis against West Nile fever. Protection against mosquito bites is recommended.

Presentation:

https://docs.google.com/presentation/d/1iOyzIe3UYl9e1YbjOB-BdzDnGX8wXZnK/edit?usp=sharing&ouid=112343790550469793652&rtpof=true&sd=true

Test control of knowledge

>>A 26-year-old patient with a history of meningococcal meningitis suddenly deteriorated upon admission to the hospital: he fainted. Objectively: pale grey skin, cold, damp, rapid breathing, blood pressure 60/20 mmHg, tachycardia. What is the reason for the deterioration of the condition?

( ) Collapse

( ) Swelling of the brain

(x) Infectious and toxic shock

( ) Acute adrenal insufficiency

( ) Thromb haemorrhagic syndrome

>>Patient D., 30 years old, with manifestations of ITP has cyanosis, low venous pressure, low blood pressure. What is the appropriate way to start detoxification therapy?

( ) Vasodilators

( ) Glucose

( ) Albumin

( ) Rheosorbilactin

(x) Crystalloids

>>Patient K., 24 years old, who was admitted to the clinic with an acute intestinal infection, has low blood pressure, tachycardia, slight thirst, periodic convulsions, decreased diuresis. What is the cause of this disease?

(x) Hypovolaemia

( ) Acute renal failure

( ) Acute heart failure

( ) Hyperkalaemia

( ) Acidosis

>> For hypovolemic shock, changes in which biochemical and laboratory parameters are most characteristic?

( ) Increase in relative plasma density

( ) Hypokalaemia

( ) Metabolic acidosis

(x) Increase in haematocrit

( ) All of the above

>>Patient D., 27 years old, with high fever, pain in the calf muscles and lower back, sclera and skin characteristic, has leukocytosis with a neutrophilic shift. The urine contains protein, leukocytes, and cylinders. The amount of urine produced has decreased to 550 ml per day. How to identify this complication?

( ) Acute urinary retention

( ) Infection and toxic shock

(x) Acute renal failure

( ) Urolithiasis

( ) Prostatitis

>>Patient L, 22 years old, with viral hepatitis, has an increase in jaundice, tachycardia. He answers the questions correctly, has impaired concentration, euphoria, mild hand tremor, hyperreflexia. How to assess this condition? <<

(x) Precompany

( ) Multiplicity

( ) Spacer

( ) Neurasthenia

( ) Character trait

>>Patient U., 34 years old, with a severe course of meningococcal meningitis, on admission to the hospital, has confusion, psychomotor agitation, convulsions and meningeal signs. What complication can be expected in such a clinic?

( ) Infection and toxic shock

( ) Acute renal failure

( ) Hypovolaemia

( ) Epilepsy

(x) Cerebral oedema

>> Patient D., 32 years old, who returned from India 3 days ago, has high fever, weakness in the legs, pain in the right inguinal region, where a conglomerate of painful, interconnected lymph nodes is palpated. The skin over the conglomerate is purple-cyanotic in colour. What is your diagnosis?

( ) Tularemia

(x) Plague

( ) Banal lymphadenitis

( ) AIDS

( ) Anthrax

>>Patient M., 40 years old, returned from the Congo. The disease began acutely with chills and fever. A drop in temperature was accompanied by sweating. Microscopy of a blood smear revealed Pl. falciparum was detected. The drug of choice in this case is?

(x) Artemisin

( ) Mefloquin

( ) Quinine

( ) Primakhin

( ) Delahil

>>A patient with severe HBV on the second day of hospitalisation developed lethargy and disorientation in space. He answers questions with a delay. The size of the liver decreased. Neurologically - nystagmus, decreased tendon reflexes, misses the finger-nose test. Diagnosis?

( ) HBV severe

( ) Hepatic encephalopathy, precoma I

( ) Comma I

( ) Comma II

(x) Hepatic encephalopathy, pre-coma II

Recommended literature

Basic

1. Infectious diseases / edited by O. A. Holubovska. 3rd edition, revised and supplemented - K.: VSV "Medicine", 2020. - 686 p.
2. Infectious and parasitic diseases in pregnant women and women in labour / edited by Malyi V.P., Kopcha V.S. - Lviv - Publisher Marchenko T.V., 2024 - T 1 - p.336.
3. Infectious and parasitic diseases in pregnant women and women in labour / edited by Malyi V.P., Kopcha V.S. - Lviv - Publisher Marchenko T.V., 2024 - T 2 - p.331.
4. Infectious and parasitic diseases in pregnant women and women in labour / edited by Malyi V.P., Kopcha V.S. - Lviv - Publisher Marchenko T.V., 2024 - T 3 - p.280.
5. Immunoprophylaxis of infectious diseases: a textbook / L.I. Chernysheva, F.I. Lapiy, A.P. Volokha et al. 3rd edition, revised and supplemented - K.: VSV "Medicine", 2022. 334 p.
6. Socially significant and especially dangerous infectious diseases / Yurko K.V., Solomennyk H.O. - K.: VSV "Medicine", 2023. 255 p.

Additionally

1. Infectious Diseases: Textbook: in 2 volumes / edited by V.P. Malyi, M.A. Andreychyn. - Lviv: Magnolia 2006, 2018. - Vol. 1. - 652 p.
2. Infectious Diseases: Textbook: in 2 volumes / edited by V.P. Malyi, M.A. Andreychyn. - Lviv: Magnolia 2006, 2018. - Vol. 2. - 726 p.
3. Vinohrad N.O. Special epidemiology: a textbook for students of higher medical schools, institutes and academies / N.O. Vinohrad, Z.P. Vasylyshyn, L.P. Kozak - 2nd edition, revised and supplemented - Kyiv: Medicine, 2018. 368 p.