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MANUAL OF HELMINTHIASIS

FOR THE STUDENTS OF MEDICAL FACULTY

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Helminths are worms causing a wide variety of diseases globally called helminthiases. Helminthiases almost only occur in developing countries, particularly in areas where sanitation is low sanitation. This manual reviews current and recent information concerning pathogenic helminths, the characteristics of their eggs and sanitary strategies controlling them.

The offered manual of Helminthisms will help students to learning skills of inspection, diagnostic thought, planning medical and prophylactic measures at infectious pathology in people.

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INTRODUCTION

Helminths are worms causing a wide variety of diseases globally called helminthiases. Helminthiases almost only occur in developing countries, particularly in areas where sanitation is low sanitation. Although helminths are not microscopic animals, their eggs, which are the infective agents, are. Helminth eggs are discharged to the environment in faeces and the oral faecal route is the main dissemination pathway of the disease. The inadequate management and disposal of wastewater, sludge and faecal sludge1 pollutes crops, water and food that when ingested serve as vehicles for transmitting the disease. Unfortunately, there is a lack of knowledge about the sanitary control of helminths in specialized literature. This manual reviews current and recent information concerning pathogenic helminths, the characteristics of their eggs and sanitary strategies controlling them.

An integrated approach is required for the effective control of helminths which includes strategic and tactical use of anthelmintics which remains the corner stone to this end and careful management of grazing lands including control of stocking rates and appropriate rotation strategies.

The offered manual of Helminthisms will help students to learning skills of inspection, diagnostic thought, planning medical and prophylactic measures at infectious pathology in people.

COMMON PART

Helminthisms are illnesses, caused parasitic worms - helmints.

Etiology. Helminthisms are caused by the parasitic worms of three classes: round worms — nematods (Nemathelminthes), cestoids (Platyhelminthes) and sosal'schikov (Trematoda).

Epidemiology and biology of helminthisms. The source of helmints can be the infected people (antroponosis helminthisms) and zoo (zoonotic helminthisms).

Helmints have a difficult biological cycle of development, including a few stages: egg, larva (sometimes a few stages), adult worm. An epidemic process depends on the features of biology of parasite. The infected people present a direct danger for circumferential only at contact helminthisms (enterobiasis, hymenolepiasis). At an enterobiasis eggs arrive at maturity in the perianal folds, at hymenolepiasis mature eggs are selected with feces. An infection takes place at a domestic contact with a patient through the articles of everyday life and dirty hands.

At geogelmintiasis immature eggs ripen in the objects of environment, more frequent than all in soil. There they pass the larval stage. An infection takes place by them at the use of food products (green-stuffs, fruit), waters or through dirty hands, containing the invasion eggs of helmint. Larvae can be actively inculcated in an organism through a skin and mucous membranes (at strongyloidiasis).

Biogelmintiasis behave to the 3-th group. Their agents pass the cycle of development with changing of 2—3 hosts. Last host is final (definitive), in him a helmint becomes adult. Other hosts the larval stages parasitize in which - intermediate. Contamination biogelmintiasis can take place per oral or percutaneous by a way. For example, at schistosomiasis, larvae actively inculcated in a skin or at a bite insects (mosquitoes, horse-flies).

Pathogeny. The feature of pathogeny of helminthisms is impossibility of exciter to propagate oneself in the organism of man. Intensity of invasion is determined the amount of eggs (larvae) of helmint, gettings in an organism. Intensity of invasion is determined by expressed of clinical displays illness. For the organism of man the most expressed pathogenic properties are possessed by the larval stages of helmints. In pathogeny of helminthisms select two stages: sharp and chronic (from a few months to many years).

Clinical presentation. Acute stage (from a few days to 2—3 months) characterize migration of larvae and toxic and allergic reactions. Toxic substances (enzymes, proteins, metabolites) have antigen and sensibilizating properties. An inflammatory reaction develops on the ways of their migration. Acute stage are characterize fever, pains in muscles and joints, general intoxication, eruption, edemata, «volatile» infiltrates in lungs, hypereosinophilia. Organic damage of different organs develop therefore: myocarditis, hepatitis, pneumonia, encephalopathy. At a massive invasion the necrotic damage of internals organs are possible. **In a chronic phase** clinical presentation are determined localization of parasite and conditioned the local damage of mucous membranes. For example, Ankylostoma and Cestode injure the mucous membrane of intestine, Opistorchis - biliation ways, Schistosoma - colon and urination ways, Echinococcus cyst is a general bilious channel and cause a subhepatic icterus. Consuming nutritives helmints are instrumental in development of hypotrophy, albuminous failure, hypovitaminosis. At helminthisms development autoimmune reactions, reducing resistance to the bacterial and viral infections. After the release of organism from a parasite circulation of specific antibodies is halted within the limits of 1 year.

Diagnostics. Helminthisms haven't specific clinical symptoms. Laboratory diagnostics has an important value.

A research purpose is an exposure of eggs, larvae, helmints of adult or their fragments (segments). They can be visible macroscopically (for example, ascarides, segments of tapeworm et cetera).

For finding out helmints probe feces, urine, duodenal content, sputum, scrape from a perianal folds, blood, biopsy tissues. For diagnostics of tissue helminthisms use serum methods (RDGA, RCK, elisa and other). Additional instrumental methods used (endoskopy, ultrasonoscope, CT, MRT).

Treatment. For treatment of helminthisms use antihelminthic chemo preparation. Depending on the type of helminthism and possibility of side effects treatment is conducted ambulatory or in permanent establishment. On testimonies a chemotherapy is combined with the use of antihistaminic and antihelminthic and anti-inflammatory preparations, sometimes steroids.

NEMATODOSES

ASCARIDOSIS (syn. - ascariasis, lumbricosis) is the anthroponotic geohelmintosis, belongs to nematodosis.

Etiology. Human Ascarides (Ascaris lumbricoides) are the agent of ascaridosis. Grown-up individuals are fusiform. The female length is 20-40 sm., male – 15-25 sm. Each female lays about 200 000 eggs daily, which are not invasion but getting into the soil with excrements at the optimal temperature (+24-30° C), humidity and aeration in 12-14 days, became matur up to invasion stage, which may cause the disease of a man. At the temperature lower then 12°C development doesn't occur, but vitability of the eggs and developing larvas are preserved.

Epidemiology. Man ill with ascaridosis is the source of invasion. Mechanism of infection is fecaly-oral, which is realized by alimentary way. The ascaride eggs excreted by invasion sources together wich feces get to the external medium, became mature in the soil, contaminate vegetables, berries, fruits and hands, articles of daily use, which become the factors of invasion transmission. The contamination takes place at swallowing of mature eggs, that why the patients ill with ascaridosis can't be the source of invasion for people at everyday contact. At present time the great danger for spreading of Ascaridosis have the private plats of cultivated lands, where sometimes not purified human feces are used for fertilizing soil.

Pathogenesis. From mature eggs swallowed by man, in small intestine the larvas go out, penetrate into intestine wall and bloody capillaries, then by blood migrate into liver, lungs and other organs. Early (migrate) phase of pathogenesis begins, which means sensabilisation by products of Ascarides larvas metabolism and traumatisation of tissues during their migration. During larvas migration inflammatory cellular infiltrates with great amount of eosinophiles (in liver, lungs) are formed, which may have clinical manifestation or develope asymptomatic. In lungs larvas get into the alveoles and bronchioles, move along the small and large bronchus, with the help of ciliary epithelium to stomatopharynx, where swallowing of larvas together with sputum takes place. The larvas getting into intestine become pubertal during 70-75 days. The late phase of pathogenesis (intestinal) depends on parasiting of mature helminth in the intestinum tenue lumen and is characterised with mechanical affection of mucose membrane and also with toxic action of helminth metabolism products on different organs and tissues and first of all on nervous system. Ascorides don't fix itself in intestinum. But are kept, leaning with its ends against intestine wall. That`s why they can descend and ascend along the intestine, to penetrate into stomack and then through esophagus into throat, in respiratory tract and even into frontal sinus.

Symptomes and course. The clinical manifestation of ascaridosis depend on parasites localization and intensity of invasion. Ascaridosis in clinical manifistation has two phases – early (migratory) and late (intestinal). The first phase coincides with larvas migration period, the second phase depends on

helminthes parasiting in intestine and possibly complications. In the early stage of ascaridosis the clinical manifistations are feebly marked, disease development is not noticeable. Sometimes the begining of disease is manifested by obvious fatigue, dry cough appears or with small amount of mucous sputum, rarely mucopurulent. Sometimes sputum has orange staining and has a small blood admixture. The body temperatura normal as a rule or subfebrile. Dry and moist rales are marked in the lungs, some patient have shortening of percutaneous sound. In some cases dry and exudate pleuritis. Physical methods not always reveal changes in the lungs. It is typical for this disease stage the changes on the hands and foot skin as urticaria and little vesicles with transparent contents. At X-ray examination of lungs is marked existence of oval, round, stellate, festone, polyangle infiltrates. Infiltrates may be or solitary or plural, are found in one lobe or all over the lung. They have rough and indistinct out lines. Eosinophilic infiltrates are found for 2-3 weeks; in some cases they disappears, then they appear again in some time. The main difference of infiltrates at ascaridosis – is rapid disappearance without any residual signs. The number of leucocytes as a rule is normal, sometimes leucocytosis is observed. Eosinophilia is typical up to 60-80% (some patients have it), it appears simultaneously with infiltrates in lungs. E.S.R. is normal, rarely accelerated. The late (intestinal) ascaridosis phase is connected with presence of helminthes in intestinum. The patient noted fatigability, appetite lowering, nausea, sometimes vomiting, pain in epigastrium, around umbilicus, in the right iliac region. Some patients have diarrhea, some have constipation. Neurologic sings - dizziness, fatigability, anxious sleep, sometimes hysteric attacks, epileptic cramps, meningism. Some ocular signs – pupil ectasia, anisocoria, photophobia, ambliopia. Cardiovascular signs – some patients note artery pressure lowering, moderate hypochromic or normochromic anemia often are revealed in the blood analysis, eosinophilia is met not always.

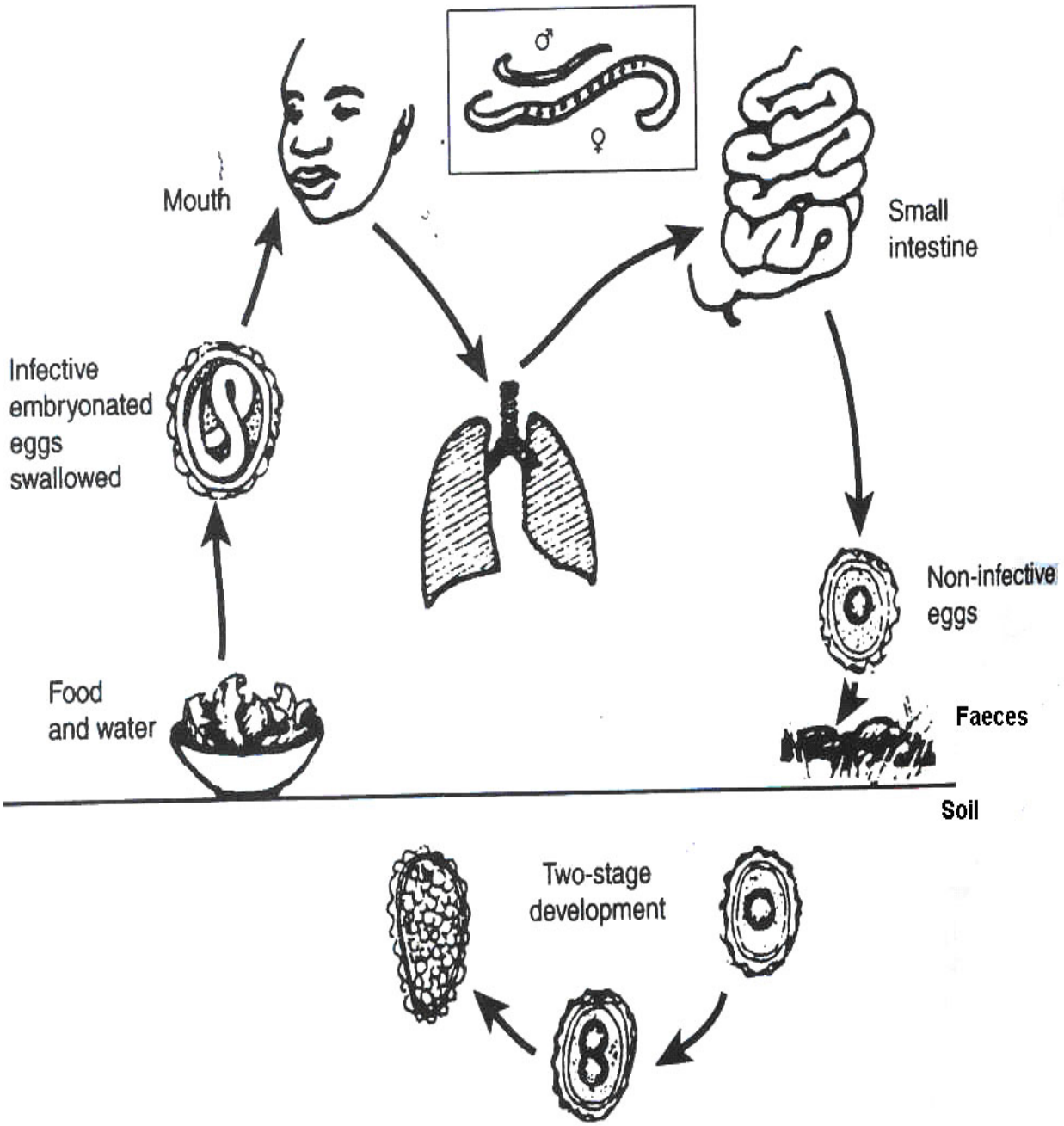
Complications. Purulent cholangitis, liver abscess, acute pancreatitis, appendicitis, intestine obstruction, asphyxia.

Specific diagnosis of ascaridosis in migrate phase on the finding of Ascarides larvas in sputum and caring out serologic reaction to determine specific antibodies in patients blood (reaction on of indirect hemagglutination, latex agglutination with Ascarides antigens). In the intestinal phase of disease the main method is examination of feces and duodenal content on presence of Ascarides eggs.

Treatment. In the early phase of disease desensitizing therapy and antinematodeus medicines of the wide spectrum of action are prescribed: mebendazol (dosage 100 mg twice a day during 3 days), mintezol (50 mg /kg a day, 2-3 times during 5-7 days), albendazol (400 mg one time). For treatment of intestinal ascaridosis are used: levamizol (150 mg one time), pirantel (10 mg/kg of body mass, one time), mebendazol, albendazol. In 2-3 week helminthologic examination must be done (3-4 times). The absence of eggs in feces during this time confirms the effectiveness of treatment. As Ascarides accommodate themself to the life in anaerobic conditions, the high concentration of oxygen is dangerous

for them. That`s why it is necessary to apply moistened oxygen, which is introduced through tube into the stomach on an empty stomach.

ASCARIDOSIS



Profilaxis. In ascaridosis prophylaxis sanitary improvement of populated areas is very important. Fertilizing of the soil is permitid only with refined feces.

ENTEROBIOSIS (syn. - enterobiasis, oxyuriasis) is the contagious anthroponotic helmintosis, belongs to nematodosis.

Etiology. The agent of enterobiasis is seatworm (Enterebius vermicularis or oxyuris vermicularis).

Female proportions are 9-12 mm., male 2-5 mm. Seatworms are found in the distal part of small intestine and proximal part of large intestine. After insemination females move into the lower part of large intestine, crawl out from anal hole and

lay eggs in perianalis folds, which become invasion in 4-6 hours. After lay eggs female died. The term of female life is not more then 1 month.

Epidemiology. The source of invasion is man ill with enterobiosis. The mechanism of transmission is fecal-oral. Disseminated with invasion eggs food, toys, hands are the factors of transmission. The dust way of invasion is also possible – seatworm eggs is rather light and are swallowed with dust. Autoinvasious (as the result of contamination of fingers at scratching of perional region) are observed at patients with enterobiosis.

Pathogenesis. The seatworm eggs get into gastro-intestinal tract . Released from coats the larvas eggs are fixed but sometimes penetrait into the mucous membrane of distal part of small intestine or proximal part of large intestine. In 12-14 days they became puberal. Around invaded seatworm granulomas may be formed, consist of eosinophyles, limphocytes, macrophages, that may be the cause of intestine dyskinesia. The products of helminth metabolism cause the development of toxic-allergic reaction. Seatworm female invading into woman`s genital organs carries the bacterial infection from intestine.

Symptoms and course. Incubative period leasts about 15 days. There are asymptomatic and clinically acute forms of enterobiosis. The main clinical symptom is perianal itching, appearing usually in the evening and at night as a result of seatworm crawling. Itching last 1-3 days then disappears and at reinvasion appears in 2-3 weeks. At massive invasion itching becomes tormenting and constant.

Scratching of anus circle leads to the development of secondary bacterial skin infection. Some patients have intestine disorder – accelerated semi – liquid stool, sometimes with mucus, tenesmus, at rectorhomanoscopy on the mucous membrane punctate hemorrhages, small erosions irritations of mucous tunic of external and internal sphincters. The signs of intoxication may be observed – weakness, fatigue, irritability, lowering of appetite. Subfebrile temperature, urticaria are observed seldom. It is often marked eosinophilia in blood at the beginning of enterobiasis.

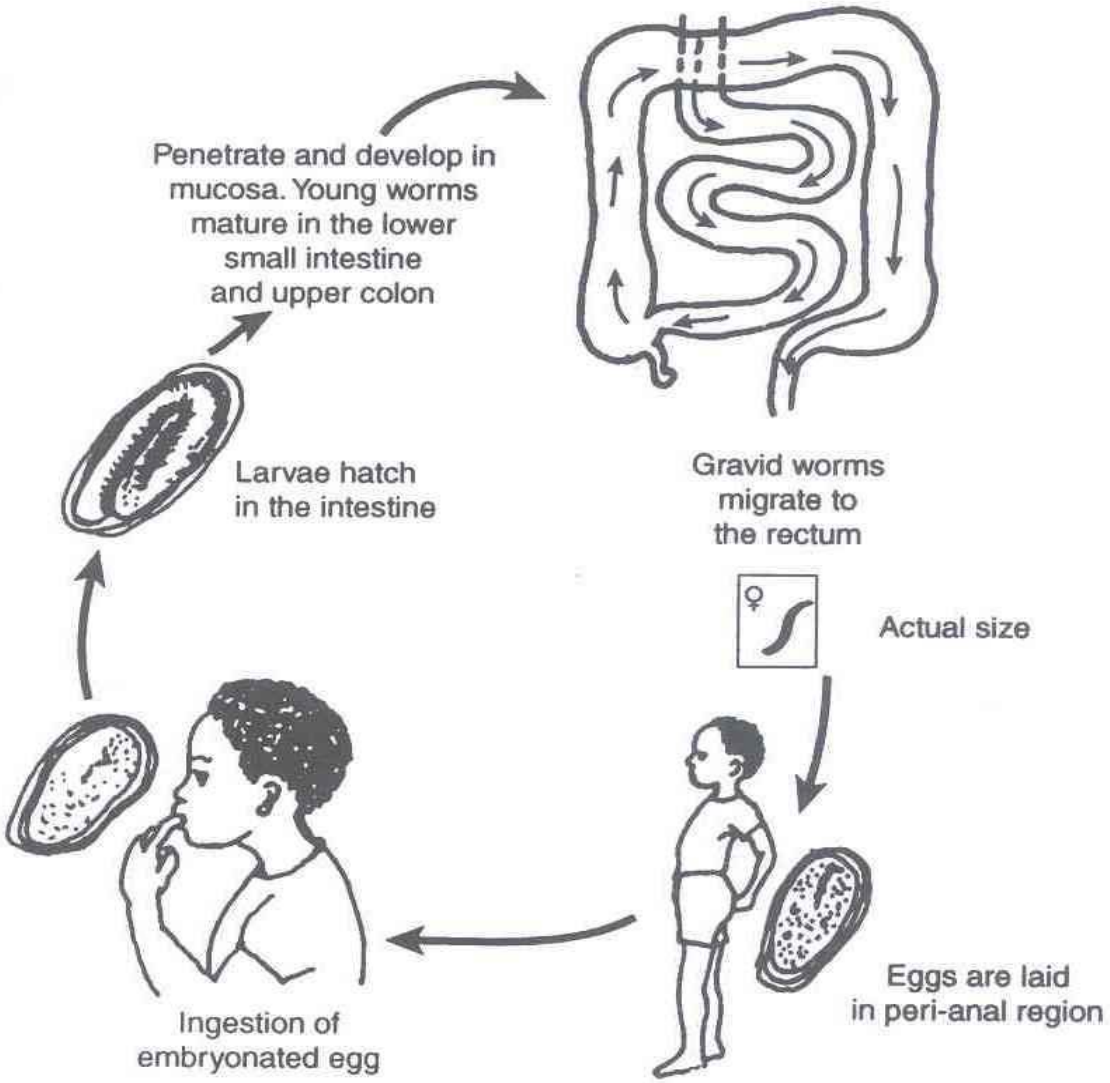
Complication. Appendicitis, sphincteritis, paraproctitis, dysbacteriosis of intestine, women have vulvovaginitis, endometritis.

Specific diagnosis. The discovery of eggs in scrape from perionale folds is the informative method of diagnosis. The scrape is made in the morning defecation with wooden spatula, cotton – wool tampon or with adhesive tenia for the later microscopia. It is necessary to do 3 examinations with interval 3-5 days. Grown up active female seatworms you may often see on the surface of the fresh separated feces of the patient.

Treatment of enterobiosis includes hygienic measures and prescribing of medicines. Hygienic measures to prevent autoinvasion: daily toilet of perionale region, sleep in pants with elastic round the legs, daily change of under wear and linen with following washing and ironing, soda purifaing clysma before sleep. The more effective medicines are mebendazol (0,1 gr one time), pirantel (10mg/kg of

the body massa, one time), albendazol (400 mg, one time). Oinment with anesthesin is prescribed at intese itch in the perionale folds.

ENTEROBIOSIS



Prophilaxis of enterobiosis is just the same in the case of intestine infectious. It is necessary to keep body clean, clean dwelling, working places especially childrens institutions.

TRICHOCEPHALOSIS - (syn. - trichuriasis, whip worm infection) is the anthroponotic geohelmintosis, belongs to nematodosis.

Etiology. The agent is – Trichocephalus trichiuris – nematode 3,5-5,5 sm., parasitizes in large intestine, rarely in lower part of intestine tenue. The anterior part of helminth is starched and looks as hair. Trichuris trichiura is obligate hematophagus. After insemination femalelays eggs in the lumen of large intestine, which get into soil with excrements and become mature active larvas during 17-20 days, at t° 26-30°C, high humidity and good aeration.

Epidemiology. A man is reservoir and source of the agent. The discharge of helminth eggs with feces begins in 1-1,5 month after contamination and leasts for 3-6 years. The mechanism of transmission is fecal – oral. The main factor of

transmission is – vegetables, berries, greenery, cultivated in the soil fertilized with feces. The disease begins at the use in food only mature eggs, that`s why it isn`t dangerous to have direct contact with ill man.

Pathogenesis. When mature (invasion) eggs get into the large intestine the larva leaves it and invades in villus. In 3 days larva gets in intestine lumen. Penetrating with thin cephalic end into thick layer the larvas are fixed in it. In 1-1,5 month pubertal helminth are formed from larvas . The main place of helminth inhabitancy is cecal intestine, sometimes vermiform process rarely other parts of large intestine and lower parts of thin intestine. At this time hairy-like part of Trichuris trichiura body is in mucous, submucous and even muscular layer, the posterior thickened part hangs down in the intestine lumen. Such arrangement of Trichuris trichiura is connected with peculiarities of feeding. – it absorbs blood vessels, situated in intestine wall. In the place of Trichuris trichiura invasion and fixation in mucous layer of thin intestine, edema and infiltration are developed, sometimes hemorrhage. Invasion of parasites deep in mucous tunic stipulate the constant irritation of interoreceptons causing the reflex of disorder of stomach functions, duodenum and CNS. The important meaning in trichicephalios pathogenesis is the sensabilization of organism with parasite metabolites.

Symptoms and course. The weak invasion of Trichuris trichiura doesen't cause serious disorders in thick intestine and has subclinical course. At serious invasion the main symptoms are: nausea, sometimes vomiting, appetite lowering, sialorrhea, stomacache localizing in right iliac region, meteorism. The patients often notice irritability, insomnia, sometimes intestine headache may appear. At trichcephaliosis instable stool or moderate diarrhea often appears, which is stupulated with disorder of water absorption in thick intestine as the result of the damage of mucous tunic and interoreceptors irritation. At the blood analysis moderate eosinophillia and hypochromic anemia are observed.

Complications. Proctosigmoiditis, rectum prolapse, appendicitis, hipochromic anemia. Specific diagnosis is based on finding of Trichuris trichiura eggs in feces. Use of the methods of enrichment essentially improve the effectiveness of parasitologic examinations, which must be done some times in succession.

Treatment. It is preferably to prescribe mebendazol (0,1gr one time after meal), medamin(10 mg/kg during 3 days), albendazol (0,4 gr one time). So as Trichuris trichiura become accustomed to live in anaerobic conditions, the high content of oxygen is dangerous for them. That`s why it is necessary to use moistened oxygen which is prescribed per rectum during 5-7 days.

Prophilaxis. Timely discovery and treatment of infected people, protection of environment from fecal contamination, observance of the rules of personal hygiene, necessary to wash vegetables and fruits.

ANCYLOSTOMIDOSES (syn. - ancylostomiasis, hookworm disease) is the anthroponotic geohelmintosis, belongs to nematodosis.

Etiology. Ancylostomidoses unite two helminthoses similar in epidemiologic and clinical manifestations. The agent of ancilostomosis is Ancylostoma duodenale, necatoriasis agent is Necator americanus. The proportions of female a.duodenale 10-13*0,4-0,6 mm., male 8-11*0,4-0,5mm; the proportions of female n.americanus 7,6-13,5*0,3-0,35 mm., male 5,5-10*0,2-0,25 mm. Necator eggs are similar with the eggs of ancylostoma.

Epidemiology. The agent of infection is ill man, excreted in environment immature eggs of helminths . The development of larvas in external environment is possible at t° from 14 to 40° C (optimal – 27-30° C), high humidity of soil and good aeration. The direct contact with ill man has no danger for other people. The infection with ancylostomosis often takes place through contaminated hands, vegetables, fruits, greenery. The infection with necatorosis takes place at walking bare foot.

Pathogenesis. Ancylostoma and nekator are localized in thin intestine, mainly in duodenum and jejunal intestine. The larvas of ancylostoma get into organism per os and develop in intestine without migration. Necator larvas usually invade activly through cutis, penetrate into the bloody capillaries, migrate along large and small circle of blood circulation. When they reach the lungs, they get into intestine through pneumanic tract, larynx and throat, where in 4-5 weeks develop up to grown – up helminth. Fixation of helminth to mucous tunic of intestine is accompanied with local damage of tissues and origin of microhemorrhages. Helminths feed with blood and excrete special anticoagulantes, which cause long – term hemorrhages. The term of helminth life is 3-5 years and even more.

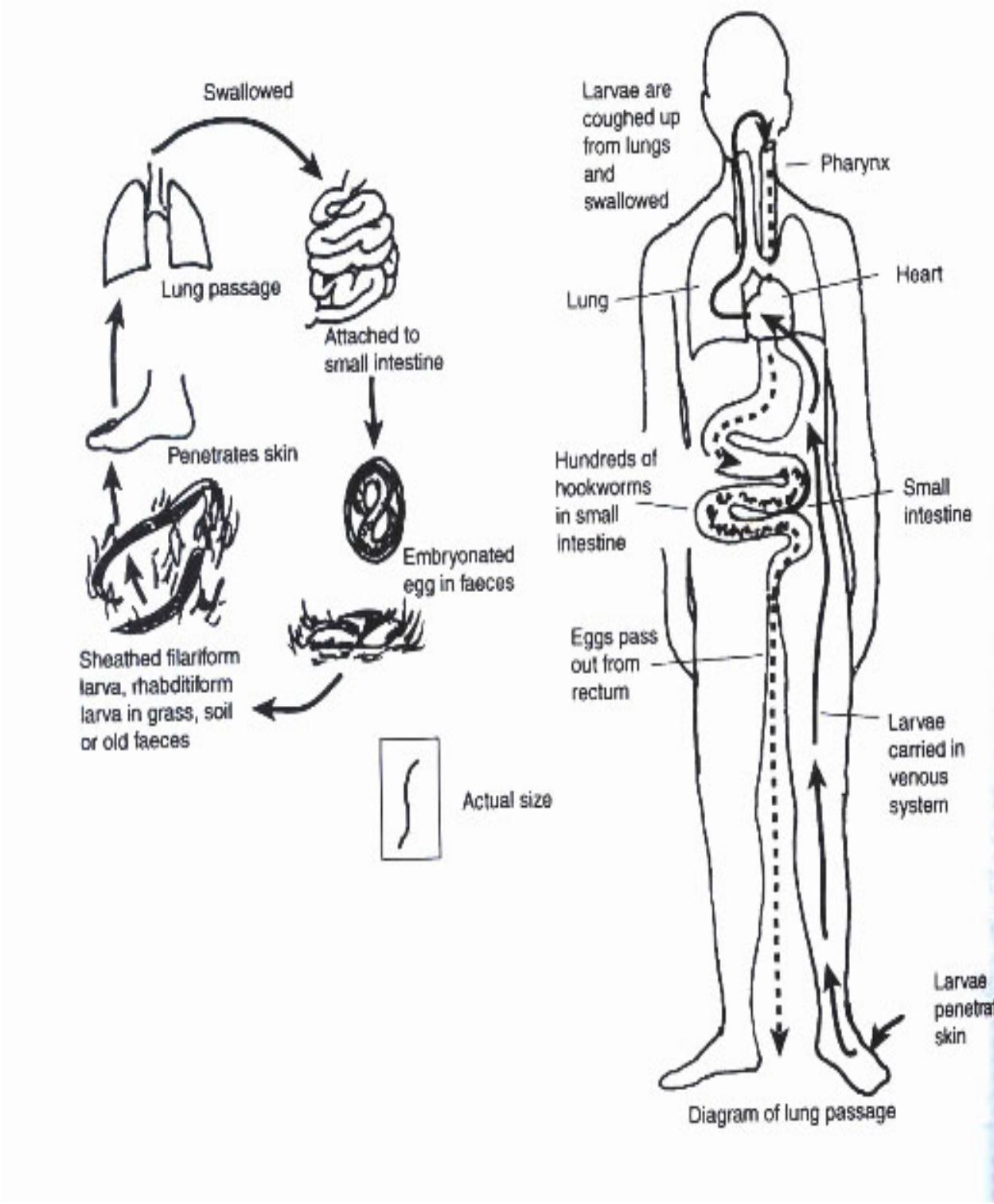
Symptoms and course. If the larvas penetrate through skin, the early manifestations are connected with their migration along organism. In the early phase of ancylostomidoses eosinophillia infiltrates in lungs and vascular pneumonia with fever and high (up to 30-60% eosonophilles in blood is discribed) Tracheitis and laryngitis with voice hoarsness and even aphonia are observed.

The intestine phase id manifested in 30-60 days after contamination – pains in the stomach, vomiting, diarrhea and fatigue appear. Pains in epigastric region remind ulcer and duodenum pains. At feces examination (coprocytogram) erythrocytes are found. The Gregersen reaction practicaly always is positive. The characteristic sign of ancylostomides is hypochromic anemia developing in a great number of patient, wich sometimes has serious form. The patients complain of fatigue, dyspnea, noise in ears, increased mental and physical defatigation, dizziness, darkness in the eyes, loss of weight, appetite lowering or increase (rarely). The patients often eat clay, lime, paper, lick metal things, salt, soap. The blood smear analyses show anisopoikilocytosis, microcytosis, hypchromia of erytrocytes.

Complications. The signs of the first stage of ancylostomidosis are – skin infection in the region of entry hilus accompaning with local inflammatory reactions. In the migration phase – allergic manifestations even Quinque edema, laryngospasm, in the intestine phase – hypochromic anemia. At long parasiting the

severe exhaustion may appear, children may have delay of mental and physical development.

ANCYLOSTOMIDOSES



Specific diagnosis. Feces or duodenal content are examined with method of native smear on a large glass with the aim of discovery ancylostomid eggs.

Treatment. Levamisole (120 -150 mg before sleep, one time), mebendazole (100 mg 2 times a day during 3 days), albendazole (400 mg one time), pyrantel pamoate (11 mg / kg body weight one time a day during 3 days). Ferrum medicines for treatment of ferric-deficiency anemia prescribed per os or

parenteral. It is necessary to prescribe folic and ascorbic acids simultaneously. At obvious allergic reactions antihistaminic medicines are used.

Prophylaxis. Discavery and treatment of patients, sanitary measures, personal hygiene. In focuses of ankylostomiasis you should not walk barefoot and lay on the ground. The soil infected with helminthes are covered salt through each 10-15 days.

STRONGYLOIDIOSIS is the intestinal nematodosis, anthroponosis, percutaneus and peroral geohelmintosis.

Etiology. Agent - Strongyloides stercoralis. Male has the length 0,7 mm and width 0,04 – 0,06 mm. The female length 2,2 mm, width 0,05 × 0,03 mm. Development of helminth takes place without intermediate owner. Pubertal female are localized in the thick part of mucose tunic of duodenum, at intensive penetraits into stomach, mucose tunic of intestinum tenue, pancreatic and biliary ducts. Inseminated females lay eggs. From eggs appear larvas. The larvas get to the external environment with exrements, where they transformed into filarideus larvas (homogonia) or into free-living pubertal males and females (heterogony). They can lay eggs. Filarideus larvas may repeatedly invase the sick man, penetrate into mucose tunic of intestine or skin perianal region (autosuperinvasion).

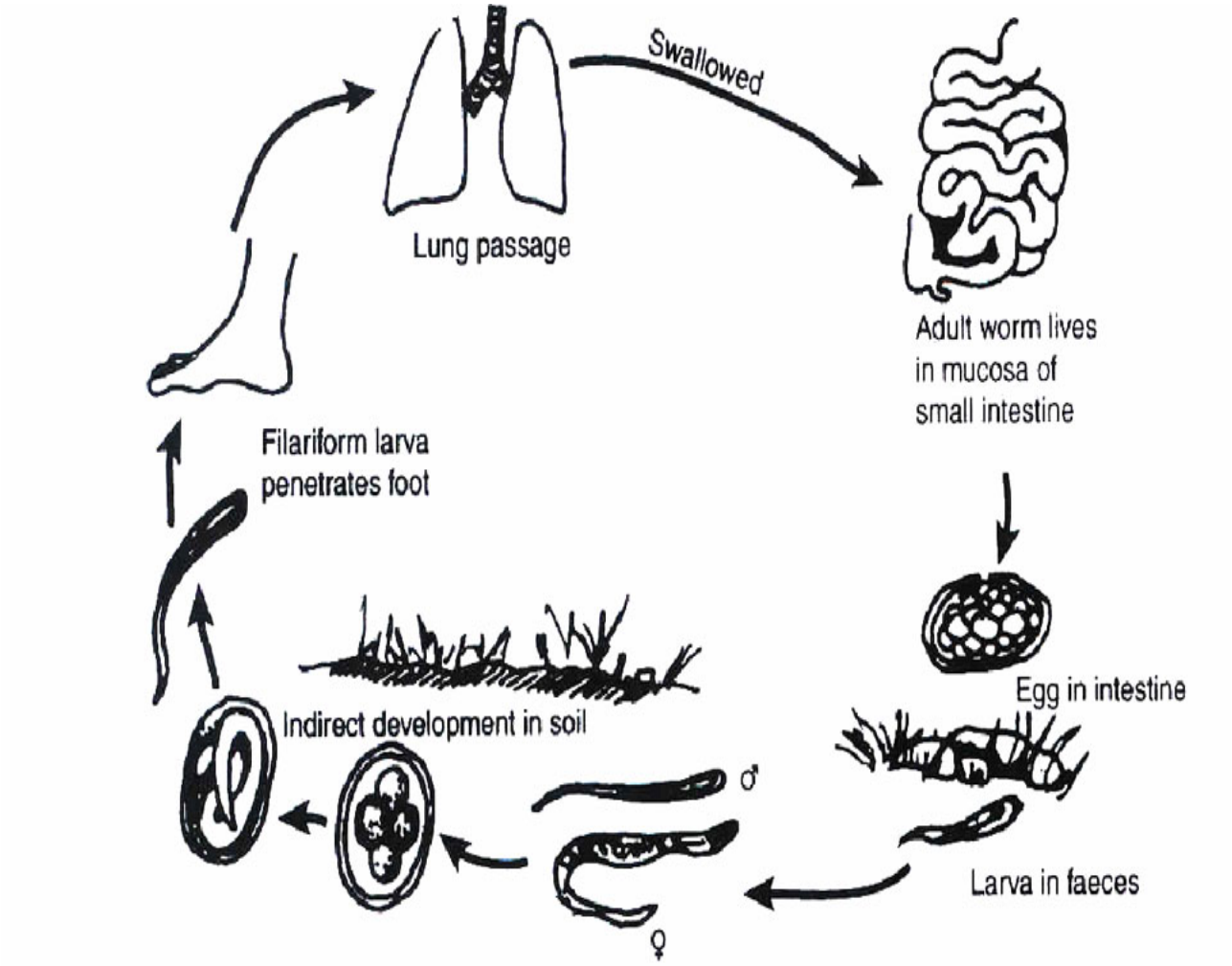
Epidemiology. The sick man is a source of infection. Contaminated soil is the source of infection (percutaneus way) penetrating through skin. There are alimentary way (if the patient eats fruits and vegetables), water way and intraintestinal autoinfection. Strongyloidosis is widely spread in the countries of east and south Africa, south-east Asia, South America.

Pathogenesis. At infection through the skin larvas penertait into the tissue through the sweaty glands and hairy follicles into bloody and lymphatic vessels. The larvas penetrait into the heart and then into lungs with the current of blood and lymph. Through alveolas, bronchus, trachea larvas penetrait in the mouth cavity and then are swallowed and penetrate into intestine. Intestinal phase develops in 20-30 days after contamination.

Symptoms and course. There are such stages in clinical course - early (acute, migratory) and late (chronic, intestinal). Incubation period is short, in 1-2 days appear dermal sings, dermal itching, nettle rash (urticaria) or papula, local edemas, appear eosinophilic infiltrates. Nausea, dull pains in epigastrium, constipations or alternation of constipation with diarrhea may be noticed. At obvious manifestations may appear nausea with vomiting, acute pain in epigastrium or in stomach, periodic diarrheas up to 5-7 times a day. Liver is enlarged and indurated. In peripheral blood eosinophillia is revealed up to 70-80 %, at long invasion secondary anemia appears. At a serious forms of strongyloidosis diarrheas a permanent. Organism dehydration, serious secondary anemia, cachexia may appear. Headache, dizziness increased fatiguability may appear (nervous system). Symptoms of duodenitis, enterocolitis, rarely angiocholitis and hepatitis are observed. If there is no treatment helminthosis has

long, chronic course. In the late (chronic) stage of the disease the symptoms appear in 4-5 weeks.

STRONGYLOIDES



There are clinical forms of strongyloidosis: intestinal, allergotoxic, duodeno-gastro-vesical, mixed. There are three stage of desease: mild, middleserious and serious. There is also asymptomatic form. The patient complains of loss of appetite, belching, heart-burn, nausea, vomiting, pain in different parts of the stomach, stool disorder at intestinal and duodeno-gastro-vesical forms. Diarrhea is the main symptom. Stool may be up to 15-20 times a day, watery, sometimes with admixture of mucus and blood.

Allergotoxic form is characterized by urticaria, dermal itching, myalgia, arthralgia. Some patients have allergic myocarditis, bronchitis, asthenovegetative syndrome, polyarthralgia as manifestation of allergia. Affection of digestive tract at this form of the disease is manifested by moderetely expressed dyspeptic disorders and abdominal pain.

Complications. Ulcerous affection of intestine, perforating peritonitis, necrotic pancreatitis, intestinal bleeding, miocarditis, meningoencephalitis, asthenic syndrome, cachexia.

Diagnosis. Diagnosis is confirmed at finding of parasite larvas in duodenal contents and in excrements, made according to Berman's method. Berman's

method is based on thermotropism of larvas (ability of the active exit from feces into the warm water.). It is possible to find larvas and pubertal parasites in sputum and urine in the migrate stage.

Treatment. Etiotropic therapy - albendazol, carbendacim, mebendazol. Albendazol is prescribed 400-800 mg a day, 1-2 times, during 3-5 days. Carbendacim and mebendazol are taken per os 10 mg / kg a day 3-5 days. It is recommended to do 1-2 course of etiotropic therapy. It is also recommended desensitizing medicines and spasmolytics. The treatment is effective if at the secondary examination of excrements and bile, which are made in 1-2-3 months after treatment, the parasite larvas are not found. Dispansory observation are recomended for 6 months with monthly control examination.

Prophylaxis. Finding and treatment sick man, organisation of sanitory measures, observance the personal hygiene.

TRICHINELLOSIS is a nematodosis, peroralis biohelminth, accompanied by fever, muscle pain and allergic manifestations.

Etiology. Agent of this disease is Trichinella spiralis. The body length of the female is 1,5-0,8 mm before insemination and 4,4 mm after insemination, the body length of male is about 2.2 mm. The parasite body is round and narrow in front. The male died after insemination. Just the some organism of the animal is for Trichinella final and intermediate owner.

Epidemiology. Domestic and wild animals are the source of infection. The wild animals (wolves, foxes, boars, badgers, bears, etc.) are the source of infection in natural focus. Rats, pigs are the source of infection in synatropic focus. Contamination may be due to eating of raw meat or not enough thermal treatment of pigs or wild animals meat (boar, bear**).** Trichinellosis is noncontact helmintosis and the sick man is not dangerous. Trichinellosis is spread everywhere, receptivity is high, season – summer and autumn.

Pathogenesis. There are two stages in the development of invasion: intestinal and migration. Into the man's organism parasite get with animal meat, which contains alive larvas in capsule. The capsule dissolves under the action of gastric juice, larvas in intestinum tenue penetrate into mucosal layer. Female begin to product alive larvas in 4-7 days. From intestine larvas are spreading into organism by blood the migration stage begins. Further development of parasite may be only in transversostriatal muscles. In skeletal muscles infiltrates are formed which make a capsule around larva from connective tissue. Inside the capsule larva develops to invasion larva. In capsule larva remains viability for 5-10 years. The larva migration is accompanied by common allergic reaction.

Symptoms and course. Incubative period lasts from 10 to 25 days. The. typical symptoms - edema of eyelids and face, muscular pains, fever, polymorphic itching rash, eosinophillia. Myalgia of different localisation is typical during the first days of disease: pain in ocular masticatory muscules and in tongue, back, legs. This pain is absent during the complete rest and appears at the movement, or palpation of muscles. At the same time fever raises to 39-40 °C and may remains

for 2-3 weeks. The tissue eruptions of erythematopapular character are localised on intestinal surface of extremities and trunk. Face and neck edema spreads to trunk and extremities. The course of disease may be latent, weak middle and serious. A latent course – subfebrile condition, weak muscular pain, face pastosity, weak eosinophillia. Intestinal manifestations is absent. At a week course – the raise of temperature up to 39 °C, temperate muscular pains, face puffiness, eosinophillia up to 20%.

At middle course - high temperature during 7-8 days, then 7-10 days subfebrile condition, intensive muscular pains, tissue puffiness, itching eraption, conjunctivitis. The signs of other organs affection appear - lungs (cough, rale), heart (tone deafness, decreased blood pressure, tachycardia). The lymphatic nodes may be enlarged. The pain in the stomach appers, eosinophillia up to 40%. Duration of disease for 3-4 weeks.

At the serious course disease begins 1-2 days after contamination. Nausea, vomiting, diarrhea, abdominal pains appear. Intoxicative syndrome, dermal allergic manifestations, high fever, hypereosinophillia (up to 80 - 90%), muscular pain, signs of affection of different organs - lungs, heart, digestive tract appear.

Complications - myocarditis, pneumonia, meningoencephalitis, abdominal syndrome, affection of liver, kidney, phlebitis, thrombosis of large vessels.

Diagnosis may be confirmed at finding Trichinella larvas in meat or in bioptate of patient muscle. Reaction of connection of complement, reaction of calceprepitation and reaction of precipitation are used for serological diagnosis. The serologic reaction is necessary to repeat in dynamics. The greatest number of antibodies are found on 4-12-week of disease. You may find migratory focuses in lungs, at X-ray of muscles you may find encapsulated larvas as small calcified formations.

Treatment. Such patients must be hospitalized. Recommended medicines are mebendazol and albendazol. Dosage of mebendazol is 10 mg/kg per day, 3 times after meal. Dosage of albendazol is 400 mg twice a day, after meal. Course of treatment is 14 days. At prescription of etiotropic remedies destruction of Trichinella in muscles and intestine is observed. It may be manifestated by increasing of clinical manifestation of disease. Due to such condition glucocorticosteroids are prescribed simultaneously. Prednisone daily dosage is 30-80 mg and desensitizing remedies. Convalescents are discharged from the hospital after disappearance of edematic and allergic syndromes, reconstruction of motor ability, ECG normalization, absent of lung changes. Patients must be under sanitarium observation during 12 months.

Prophylaxis. Sanitary - veterinary inspection and sanitary educational work.

TOXOCAROSIS is a nematodosis, tissue zoonosis helminthosis with affection of inner organs and eyes.

Etiology. Round worms of Ascaridida group, Anisakidae family. Toxocara genus are the agents of Toxocara canis. Toxocara canis is a helminth of dogs family and have important epidemiologic meaning for a man. Toxocara mystax is a

helminth of a feeling family (cats), whose role in man's pathology has not yet proved. The size of puberal helminth Toxocara canis is from 4 to 18 cm. Toxocara canis female lays more than 200 thousand eggs a day. The Toxocara canis eggs have almost round form. The puberal invasion eggs have alive larvas. When the eggs are in the soil, they preserve vitable and invasiveness.

Epidemiology. The disease is spread everywhere. Dogs are the source of contamination. The dogs excrete Toxocara eggs with excrements contaminate the soil. In the external environment at favorable temperature and humidity the invasion larva is formed within the egg in 5 days. People are infected while eating the Toxocara eggs. The sick people are not source of invasion.

Pathogenesis. The infection of a man takes place while eating the Toxocar invasion eggs. The larvas are discharged from eggs in proximal part of intestinum tenue. The larvas penetrate into the blood flow through mucos tunic of thin intestinum, then into the liver and right part of the heart. Larvas continue to migrate from pulmonary artery and get into the left part of the heart, then larvas are delivered with arterial blood to organs and tissues. They circulate along vascular system and reach the place where the vessel diameter don't make it possible to move further (larva diameter 0.02 mm). Here larvas leave the blood flow and penetrate into surrounding tissue. The Toxocar larvas settle in liver, lungs, heart, kidneys, pancreas, brain, eyes. The larvas keep vitability during months up to 10 years. Part of them may activate and continue migration. The other part incapsulates and distracts inside the capsule. During migration the larvas traumatized tissues, leave hemorrhage, necrosis, inflammatory changes. The leading role in the development of immunopathologic reactions in the sensibilisation by Toxocara antigens. Formation of granulomas in liver, lungs, pancreas, myocardium, mesenteric lymph nodes, brain is typical for such state. Granulomas are formed as the result of allergic reaction of slowed type.

Symptoms and course. The clinical manifestations of Toxocara depend on parasites localization. There are 2 forms: visceral and ocular Toxocarosis.

Visceral Toxocarosis is manifested by recurring fever during some weeks or months. Temperature is more often subfebrile rarely febrile. It is possible to have lung affection sush us: bronchitis, pneumonia, seldom bronchial asthma. On the lungs roentgenogrammetry of such patients multiple or singular infiltrates, strengthening of lungs outline are seen. The enlarged liver is determined often, rarely the enlargement of spleen and lymphatic nodes. It is possible to have eritematoseus or urticarial rash on the skin. It is possible to have convulsions, epileptoed attacks, paresis and paralysis, behavior changes during the affection of central nervous system.

Eosinophillia is the most permanent sign of Toxocarosis, is often accompanied with leukocytosis and increased ESR. In biochemical blood analysis shows moderate increasing of bilirubin and small increasing in liver ferments activity.

If a man is infected with small number of Toxocar larvas ocular Toxocarosis is developed. At ocular Toxocarosis granulomas, uveitis, chronic endophthalmitis,

abscess in vitreous body, neuritis of visual nerve, keratitis or existence of migrate larva in vitreous body are developed.

Complications. At migration of Toxocara larvas into brain epileptiform attacks, paresis, paralysis are observed. At affection of visual nerve blindness may occur.

Diagnosis are made on the base of histological examination of bioptates of affected tissues. For serologic diagnosis the method is used immunofermental analysis is used.

Treatment. For treatment Albendazol (10 mg / kg of the patients weight) during 10 -20 days. Ditrazin citras is prescribed 3 mg / kg of the patients weight a day 2-4 weeks. Antihistaminic medicines are prescribed simultaneously. At ocular Toxocarosis the same schemes of treatment are used as at visceral Toxocarosis. In some cases surgical methods are used.

Sanitaruim examination are made during 1 year.

Prevention and measures. Measures directed to the source of invasion (dogs examination and treatment, catching the stray dogs, equipping the special places for domestic dogs etc.). Observance of the rules of personal hygiene (washing hands after contact with soil or animals, cleansing of greenery, vegetables and other food-stuffs, which may contain parts of soil etc.). Observance of the rules public hygiene (defense kids places for play from stray dogs).

DIROPHYLARIOSIS is a transmissible zoonotic biohelminthosis, nematodosis, characterized by parasiting of nematode Dirophylaria in subcutaneous fat of a man and is manifested by migratory tumor on the different parts of the body.

Etiology. The agent belongs to the round worms Nematoda class, Spirurina group, Spiruromorpha subgroup, Filarioidea family, Dirofilaria. Some worm types are described, from which D. Repens and D. Immitis are widely spread. Nematode has the filament body, covered with cuticle. The males proportion are 47-70x0,37-0,45 mm. Females proportion are 100-170x0,46-0,55 mm. Dogs are the obligate owners. They have such helminthes under their skin. Intermediate owners and vectors are Aedes, Culex and Anorheles mosquitoes. Which suck in the blood of the infected dogs and swallow the Microfilariae. In dogs organism Microfilariae grow at the temperature 24-28° C and in 15-20 days they migrate into the lower lip of the mosquito.

Epidemiology. Reservoir and source is canis family. Contamination of a man takes places by transmissible way through the mosquito-bite genus Aedes, Culex and Anorheles. The source of mosquito contamination are invasive dogs. The men for Dirophylariosis is in accidental owner. In the man's organism female doesn't give birth to microfilariae. A man is a biological deadlock and isn't the source of infection.

Pathogenesis. While mosquito sucks blood the invasive Microfilariae elements get into the skin. Here they begin to develop and active move in subcutaneous fat. Sometimes around helminths is formed thin connective tissue

capsule. In the pathogenesis base is a toxicoallergic reactions and mechanical influence of helminths on tissues of subcutaneous fat. Diseases may last for a long time asymptomatic. Allergic manifestation are observed rare and only in the acute period.

Symptoms and course. Incubation period is from 1-2 months up to 2 years. The clinical picture depends on the place where helminths is located. The first sign of disease is appearance on the place of a bite. Sometimes it accompanied by itching, burning. The skin over it is slightly hyperemic and slightly adematic. Typical symptom of dirophylariosis is the migration of agent – movement of swell-like formation or helminth itself under skin, especially after the local treatment. Distance of the movement is more than 10 sm. Speed of movement up to 30 sm for 1-2 days. At parasite migration through subcutaneous fat to the new place new induration appears and the former place no marks are left. Specific sign of dirophylariasis is sensation of motionless and crawling in induration or tumor. Headache, nausea, weakness, fever, pain in the place of helminth localization with irradiating along nerve trunk may be the symptoms of this desease. Eosinophillia is not typical.

At ocular dirophylariosis eyelids, conjunctiva, anterior chamber, sclera, orbit are affected. At affection of eyebrows skin and eyelids Quincke edema is developed. The eyelids are swollen, there is itching, watery eyes. Under the skin dense nodules or tumor are formed. The vision acuity doesn't go down. If the conjunctiva is affected, conjunctivitis develops accompanied with intense pains, tearness and itching. Conjunctiva is edematic and during some days, through it you may see curved helminth body. All sing disappear completely after it migrates into orbit or is removed surgically.

Complications. Pyosis of subcutaneous nodes, secondary inflammatory processes at eyes affection.

Diagnosis. Clinical diagnosis is difficult. The important meaning has the epidemiological anamnesis – mosquito-bites. Parasitological diagnosis is also difficult because of absent of Microfilaria in blood microfilariae, eosinophillia is not typical. Diagnosis of dirophylariosis is set retrospectively as the result of surgery of removing of subcutaneous node, in which the dirophylaria is found.

Treatment. The optimal method of treatment is surgical removing of helminth. If the correct diagnosis was set without surgery, diaethylcarbamazine 2 mg / kg per a day 10-30 days, antiallergic drugs, glucocorticoids are prescribed. Antibacterial drugs are prescribed at suppuration (pyosis) of nodes and appearance secondary bacterial infection. The prognosis of disease is favorable.

Sanitarium observation is set for 6 months. Recovered after are taken off registration at the clinical recovery and absence of formation of new subcutaneous nodes.

Prophylaxis. Control of mosquitoes, discovery and dehelmenthisation of invased dogs, the measures of personal prophylaxis, defence from mosquito bites.

CESTODOSES

TAENIARHYNHOSIS is antraponotic biohelminthosis with chronic course.

Etiology. Causative agent is Taeniarhynchus saginatus. Adult worm of T. Saginatus consists of scolex, neck and tape body. Scolex has 4 suckers. Body (strobila) consists of 1000–2000 hermaphroditic segments (proglottids). Body length is about 4-7 meters. The terminal proglottids have uterus with eggs. One mature proglottid has about 170 thousands eggs. Eggs are stable in environment especially at low temperature. They can survive in water during 33 days, in grass – up to 159 days.

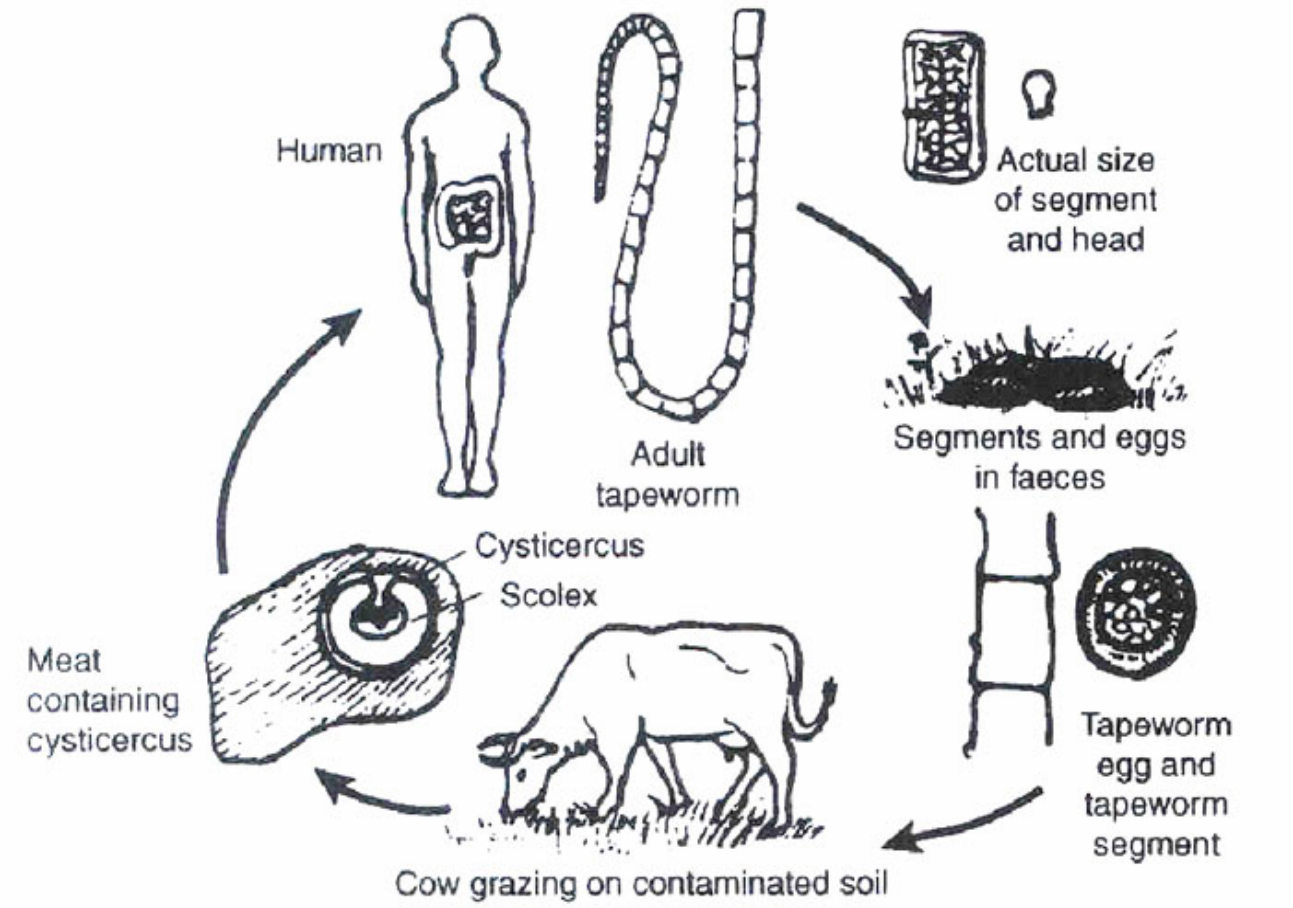
Epidemiology. Taeniarhynhosis is widespread but is most prevalent in Middle and Southeast Asia, Transcaucasia, Africa, South America and Australia. It is more spread in countries with large livestock sector. Source of invasion is ill human. Adult helminthes can live in human body for many years (25 years).

Human is the only definitive host for T. saginata, which inhabits the upper jejunum. On the 75^{th}-91^{st} day after invasion mature proglottids start excreted. They are released almost every day. Eggs contained larvas (oncospheres) are infective for the intermediate host such as herbivores (cattle, buffalo, yak) hence T. Saginatus is biohelminth. Taeniarhynhosis doesn't belong to contact helminthosis and ill human is not contagious for other people. Cattle become infected by eating fodder contained human faeces with oncospheres. The embryo released after ingestion invades the intestinal wall, enters the bloodstream and is carried to intramuscular connective tissue, where it transforms into the cysticercus. It is round shaped formation up to 1 cm in diameter with a scolex inside. They become infective within 12 weeks and remain viable in the living host for 2 years; they are viable in stored, chilled meat for several weeks but are killed at –20°C within 1 week. Humans become infected after ingesting of raw or undercooked infected meat (shashlik etc., tasting of raw minced meat). After the cysticercus is ingested, it takes 2 months for the mature adult worm to develop. There is no formation of immunity to T. Saginatus.

Pathogenesis. T. Saginata has toxico-allergic influence into human body, leads to mechanical damage and abnormality of nutritive absorption in intestine due to mucous membrane injury.

Clinical features. Invasion with T. saginatus is often asymptomatic. Fecal passage of proglottids may be noted by patients. The proglottids are often motile and can crawl out of the anus without defecation especially at night. Due to this patients can find proglottids in bed which is typical only for Taeniarhynhosis. Patients may experience perianal discomfort when proglottids are discharged. Pruritus ani is common. Mild abdominal pain or discomfort, nausea, change in appetite, weakness, diarrhea and weight loss can occur. T. saginatus occasionally obstructs the small intestine, pancreatic duct, or bile duct. Proglottids are recorded in the gallbladder, and eggs have been found in gallstones.

TAENIA SAGINATA



Specific diagnostics. The diagnosis is made by the detection of eggs or proglottids in the stool. Eggs may also be present in the perianal area; thus, if proglottids or eggs are not found in the stool, the perianal region should be examined with use of a cellophane-tape swab. The eggs are indistinguishable from those of T. solium so laboratory will give conclusion: “eggs of teniid are found”. Distinguishing T. saginata from T. solium requires examination of mature proglottids by macroscopic examination which is the main method of investigation for Taeniarhynhosis. Mature segments of T. saginatus are 2 cm in length and 0,5 cm in width. Each gravid segment has 15–38 uterine branches (in contrast to 8–12 for T. solium).

Treatment. Praziquantel, Mebendazole, Niclosamide, pumpkin seeds and other medicine can be used.

Niclosamide. A single morning dose of 2 g niclosamide is given to adults and older children on an empty stomach; the tablets should be chewed. Children of 2 to 6 years should receive 1 g, and those below 2 years, 500 mg.

Praziquantel is given in a single dose of 10 to 20 mg/kg after a light breakfast.

Phenasalum is given in dose of 2 g in 3 hours after a light breakfast and 1 g on an empty stomach next morning.

Mebendazole (vermox) 300 mg 2 times a day during 3 days.

After either drug the proximal part of the worm disintegrates in the gut and the scolex cannot be found. Failure of proglottids to reappear within 3 to 4 months indicates cure.

In case of bad tolerance to antihelminthic drugs pumpkin seeds are used. Patient has to take easily digestible food and do enema every morning during two days before treatment starts. In the evening of the last day before treatment saline purgative has to be taken and next morning on an empty stomach enema has to be done. 500 grams of pumpkin seeds are chopped. Twice more amount of water is added. Put it in steam bath for two hours, filtrate through gauze then remove oily film. It should be taken on an empty stomach during 30 minutes. In 2 hours after taking decoction saline purgative has to be given.

Regular medical check-up. In three months after course of treatment investigation of faeces has to be done 4 times with one month interval. After negative results of laboratory examinations patient doesn't need to do regular medical check-up anymore.

TAENIASIS is antraponotic biohelminthosis with chronic course.

Etiology. Causative agent is Taenia solium. It is 2-3 meters in length and consists of scolex, neck and strobila. Scolex has four suckers and a double row of hooks. Strobila may have 1000 segments (proglottids). The terminal proglottids have uterus with up to 50 000 eggs. Eggs are stable in environment. They can survive in desiccation condition during 10 months or under snow during winter. But they are sensitive to high temperature. Direct sunbeams kill them in soil during 2 days.

Epidemiology. Taeniosis is widespread, especially in India, China, Africa, Latin America.

Source of invasion is ill human. T. solium can cause two distinct forms of infection in humans: adult helminthes live in small intestine of human and cause Taeniosis; larval forms inhabit the tissues and cause cysticercosis. The definitive host for T. Solium is humans. Segments get detached from strobila and excreted in environment with faeces. Eggs contain oncospheres and are infective for both humans and animals.

T. solium is biohelminth. Maturation of larva takes place in intermediate hosts such us pig, wild boar, cat, camel, hare, monkey. They become infected with oncospheres by eating feedstuff contaminated with patient's faeces. In intestine of intermediate host the embryo released after ingestion invades the intestinal wall and is carried with blood to almost all organs where in 2-3 months it transforms into the cysticercus with about 1 cm in diameter. Cysticercus is round-shaped formation with scolex and neck of parasite inside. In animals (most of all in pigs) they are mainly localized in intermuscular connective tissue. The cysts are most numerous in the tongue, masseter, heart, and diaphragm, but also occur in the brain.

Invasion of human with Taeniosis happens due to eating of invasive, not proper cooked meat (shashlik, blood beefsteak and other). Cysticercus localized in pig is resistant to high and low temperature, high concentration of salt. In small

intestine of human scolex comes out from cysticercus and attaches to mucous membrane. In 2 months helminth becomes sexually mature and starts detaching and releasing segments.

Susceptibility to Taeniosis is common. Immunity isn't formed.

Human can be as definitive as intermediate host for T. solium. Oncospheres can enter human body by two ways. The first one is with vegetables and verdure (green onion, dill, parsley) contaminated with patient's faeces. The second one is due to entering mature segments into stomach from intestine during reversed peristalsis (for example, in case of vomiting). Oncospheres entered into stomach release embryos which penetrate the intestinal wall and are carried with blood all over the body. In human body cysticercus is often found in brain and eye and rarely in skin, muscles and other tissues.Therefore human with Taeniasis can be course of Cysticercosis invasion for himself and other people.

Pathogenesis is the same as in case of Taeniarhynhosis.

Clinical features. Invasion with T. solium may be asymptomatic and patients become aware of the infection by noting passage of proglottids in their faeces. Some patients complain of nausea, abdominal pain, weakness and sleep disorders. Symptoms are not associated with pruritus ani. The proglottids do not migrate actively *per anum*.

Specific diagnostics of taeniosis. The main method of examination is macroscopical which can reveal released gravid proglottids in the faeces (often in short chains). Mature segment of T. solium has 8–10 uterine branches. The eggs of T. solium and T. saginatus have similar structure therefore if only eggs are found laboratory gives conclusion that these are eggs of teniid.

Clinical features of cysticercosis. The clinical manifestations of cysticercosis are variable and depend on localization and number of parasites. Cysticerci can be found anywhere in the body but are most commonly detected in the brain, skeletal muscle, subcutaneous tissue, or eye. The most often cysticerci are localized in brain. Epilepsy, the most common presentation of neurocysticercosis, is usually the primary or sole manifestation of the disease. Seizures occur in 50 to 80 per cent of patients with parenchymal brain cysts or calcifications but are less common in other forms of the disease. Seizures may be generalized, focal, or Jacksonian. Other common focal signs include pyramidal tract signs, sensory deficits, signs of brainstem dysfunction, and involuntary movements. These manifestations usually follow a subacute or chronic course, making neurocysticercosis difficult to differentiate clinically from neoplasms or other infections of the central nervous system. Focal signs may occur abruptly in patients who develop a cerebral infarct as a complication of subarachnoid neurocysticercosis. When cysticerci develop at the base of the brain or in the subarachnoid space, they may cause chronic meningitis or arachnoiditis, communicating hydrocephalus, or strokes.

Neurocysticercosis may present with increased intracranial pressure, usually from hydrocephalus secondary to cysticercotic arachnoiditis, granular ependymitis, or ventricular cysts. In these cases, intracranial hypertension develops subacutely and progresses slowly. Signs of increased intracranial pressure, including

headache, nausea, vomiting, changes in vision, dizziness, ataxia, or confusion, are often evident. Patients with hydrocephalus may develop papilledema or display altered mental status. Lymphatic pleocytosis with prevalence of eosinophils and elevated level of protein are found in CSF in patients with neurocysticercosis.

Cysticercosis of eye causes visual impairment and sometimes results in blindness. Lesion of orbit leads to exophthalmus.

Muscular pseudohypertrophy, a rare presentation, is caused by heavy cysticercal infection of skeletal muscles giving a 'Herculean' appearance. The few cases reported are all from India. Muscular and subcutaneous cysticerci are far less common in American than in African or Asian patients with neurocysticercosis (See table).

The clinical manifestations of cystecercosis in dependence on localization of cyctecercs

Symptoms	Eyes	Brain		
		Cerebral hemispheres	IV Ventricle	Basis
Headache		+	+	
Dizziness		+	+	
Nausea		+	+	
Vomiting		+	+	
Epileptic attacks		+		
Disorders of psychics (dimension, gallucinations)				+
Dispnoe			+	
The harsh of the state in impairment the change of the body position			+	
Disorder of vestibular conduction				+
Paresis, paralyses			+	
Progressive impairment of the vision	+			

Specific diagnostics of Cysticercosis. Immunological tests such as immune enzyme analysis with cysticerci diagnosticum, instrumental methods such as computed tomography (CT), magnetic resonance imaging (MRI), ophthalmoscopy and ultrasound examination.

Treatment. Patients with Taeniosis have to be treated in hospital. The drug of choice is Extract of male fern (Filicis maris rhizoma) which has paralytic effect on muscles of parasite. Prescription the drugs which kill parasite may lead to digestion of parasite and releasing eggs from its segments that can result in development of Cysticercosis. Patient has to take easily digestible food during 2 days. In the evening of the 2nd day saline purgative (Magnesium Sulfate) has to be taken and next morning on an empty stomach cleansing enema has to be done. Then during 30 minutes patient has to take 4-7 g of Filicis maris rhizoma (1 capsule which has 0.5 g every 3 minutes). In 30-60 minutes after taking the last capsule saline purgative has to be used. If patient doesn't have stool during 3 hours after taking purgative, cleansing enema has to be done. Helminth excreted with the faeces has to be examined under the microscope to find scolex. If scolex is not found, 1-3 enemas have to be done.

Filicis maris rhizome is very toxic. Pumpkin seeds are less toxic than Filicis maris rhizome but they have less antihelminthic activity. They can be prescribed if patient has contraindication to Filicis maris rhizome. Patient has to take easily digestible food and do enema every morning during two days before treatment starts. In the evening of the last day before treatment saline purgative has to be taken and next morning on an empty stomach enema has to be done. 500 grams of pumpkin seeds are chopped. Twice more amount of water is added. Put it in steam bath for two hours, filtrate through gauze then remove oily film. It should be taken on an empty stomach during 30 minutes. In 2 hours after taking decoction saline purgative has to be given.

The recommended treatment for cysticercosis is albendazole, 15mg/kg in 2–3 doses over 8–28 days, repeated as necessary. Praziquantel is also used in a dosage of 50mg/kg/day in three doses for 15 days. The cyst may also be surgically removed. Surgery (and not chemotherapy) is recommended for intraocular cysticercosis.

Regular medical check-up. In two months after treatment of Taeniasis investigation of faeces has to be done 4 times with one month interval.

Regular medical check-up of patient with Cysticercosis lasts during 1 year. One time in 6 months they have to be examined by neurologist and ophthalmologist, and control instrumental examinations such as ophthalmoscopy, computed tomography and other have to be done.

DIPHYLLOBOTHRIOSIS is zoonotic biohelminthosis with chronic course.

The causative agent is diphyllothrium latum. The adult worm is the longest tapeworm (up to 25 m). It attaches to the ileal and occasionally to the jejunal mucosa by its suckers (bothria), which are located on its elongated scolex. The adult worm has 3000–4000 hermaphroditic proglottids. The terminal mature segment has uterus, which is opened in human intestine and releases about 1-2 million eggs daily into the faeces. Eggs are stable in environment especially in low temperature. They can survive in water or under snow during winter time.

Epidemiology. Diphyllobothriosis is spread in North Europe, East Mediterranean, USA, Canada, Alaska, Central Africa, Russia and Ukraine.

The source of invasion is human, dog, cat, pig, bear, fox and other fish-eating animals such us seal, mink, walrus. The adult worms inhabit in small intestine. In human body they can live up to 25 years. Infected human is not contagious for other people.

D. latum is biohelminth which needs tree hosts for its lifecycle. With definitive host's faeces egg reaches water. If temperature of water is 10-20 ºC and there is enough oxygen, egg hatches and releases a free-swimming embryo that can be eaten by small freshwater crustaceans (Cyclops or Diaptomus species), who are the fist intermediate host. After an infected crustacean containing a developed procercoid is swallowed by a fish, the larva migrates into the fish's flesh and grows into a plerocercoid, or sparganum larva. Consequently fish (pike, perch, burbot, chum salmon, hunchback salmon and other freshwater fish) is the second intermediate host, which has larva of D. latum in its body, liver, hardroe. Humans acquire the invasion by ingesting infected raw or not enough salted fish or caviar. Hot or cold smoking can destroy larva in fish. Frying fish within 20-40 min, heating it to 54°C for 5 min or freezing it at –18°C for 24 hours also kills larva.

Susceptibility to Diphyllobothriosis is common.

Pathogenesis. Negative influence on human body is result of mechanical, toxico-allergic affect of helminth. Because the tapeworm absorbs large quantities of vitamin B12 and interferes with ileal B12 absorption, vitamin B12 deficiency can develop. It leads to appearance of megaloblastic anemia.

Clinical Manifestations. Most D. latum infections are asymptomatic, patients become aware of invasion by noting proglottids in faeces. Some patients complain about weakness, nausea, vomiting, transient abdominal discomfort, urticaria, diarrhea and weight loss. Occasionally, invasion can cause acute abdominal pain and intestinal obstruction; in rare cases, cholangitis or cholecystitis may be produced by migrating proglottids. Months or even years after invasion clinical features of anemia (such us pale skin) appear. 2-3 % of patients have B12 deficiency anemia. They feel pain and paresthesia in tongue. Some of them have atrophic Hunter's glossitis with bright red painful spots and fissures on tongue. There are tachycardia and hypotonia. . There are decrease number of erythrocytes (to $1.5\text{-}2.0\times10^{12}$/liter), low hemoglobin level, high colour index, megaloblasts, Jolly's bodies and Cabot's rings in general blood analysis.

Specific diagnostics. The diagnosis can be confirmed by the detection of the characteristic eggs in the stool. The oval shaped eggs possess a single shell with an operculum at one end and a knob at the other. Discharged chains of gravid segments are also diagnostic. The width of proglottids (up to 1.5 cm) is longer then length. There is rosette shaped uterus in the middle of proglottid.

Treatment. Praziquantel, Niclosamide, Phenasalum, Mebendazole, pumpkin seeds and other medicine can be used.

Praziquantel is used in a single dose of 10 mg/kg body weight.

Praziquantel (5–10 mg/kg once) is highly effective.

Niclosamide. A single morning dose of 2 g niclosamide is given to adults and older children on an empty stomach.

Phenasalum is given in dose of 2 g in 3 hours after a light breakfast and 1 g on an empty stomach next morning.

Mebendazole (vermox) 300 mg 2 times a day during 3 days.

In case of bad tolerance to antihelminthic drugs pumpkin seeds are used. Patient has to take easily digestible food and do enema every morning during two days before treatment starts. In the evening of the last day before treatment saline purgative has to be taken and next morning on an empty stomach enema has to be done. 500 grams of pumpkin seeds are chopped. Twice more amount of water is added. Put it in steam bath for two hours, filtrate through gauze then remove oily film. It should be taken on an empty stomach during 30 minutes. In 2 hours after taking decoction saline purgative has to be given.

Treatment of evident anemia should be started before using anthelmintic medicine. Parenteral vitamin B12 should be given if B_{12} deficiency is manifest.

Regular medical check-up of patient with Diphyllobothriosis lasts during 4-6 months. In three months after course of treatment control microscopic examination of faeces has to be done 2-3 times with one week interval. After negative results of laboratory examinations patient doesn't need to do regular medical check-up anymore.

ECHINOCOCCOSIS is biohelminthosis, which is caused in humans by the larval stage of the Echinococcus granulosus and characterized by formation of hydatids (cyst with daughter cysts, each containing several protoscolices) in different organs of human body.

Etiology. The causative agent of hydatidic echinococcosis is Echinococcosis granulosus. Adult helminth lives for 5–20 months in the jejunum of canines. It reaches 5-6 mm in length with 3-4 proglottids. There are 4 spherical suckers and rostellum with two rows of hooks on a scolex. Last gravid segment has about 800-1000 eggs that are morphologically similar to Taenia eggs. The eggs are stable in environment and can survive during 6 months in temperature +1+20 ºC.

Epidemiology. Echinococcosis is found on all continents. It is common in Australia, New Zealand, China, central Asia, the Middle East, the Mediterranean region, eastern Africa, and parts of South America.

Echinococcus granulosus has both intermediate and definitive hosts. The adult tapeworm is found in the small intestine of the definitive host, usually dogs or other canines (wolfs, jackal, foxes). Gravid segments are excreted with faeces or crawl out from the anus without act of defecation. They release eggs contained larvas (oncospheres) which are infectious for the intermediate hosts (sheep, cattle, horses, pigs, goats, camels, human and others). Human becomes infected by ingestion of eggs with contaminated food (vegetables, berries) or water, by contact with infected dogs which have eggs on their hair. Inhalation way of transmission can be also realized by inspiration of oncospheres in lungs with dust.

After humans ingest the eggs, oncospheres escape from the eggs, penetrate the intestinal mucosa, enter the portal circulation, and are carried to various organs,

most commonly the liver and lungs, rarely brain. Larvae develop into fluid-filled unilocular hydatid cysts that consist of an external membrane and an inner germinal layer. Daughter cysts develop from the inner aspect of the germinal layer, as do germinating cystic structures called brood capsules. New larvae, called protoscolices, develop in large numbers (up to 100) within the brood capsule. The cysts expand slowly over a period of years. Size of the cyst can be different from 1 to 40 cm in diameter and bigger. Larvae stay viable inside the cyst for many years. Human with Echinococcosis is not dangerous for other people.

There are two types of focus of Echinococcosis. They are natural and anthropurgic foci. Natural foci of Echinococcosis (wild cycles) involve wild predators such as wolves and others (definitive hosts) and herbivorous animals (intermediate hosts). Predators get Echinococcus granulosus by ingesting infected with cysts organs of herbivorous animal (liver, lungs, kidney). In their intestine scolices release from cyst, then attach to mucous of small intestine, grow and become adult helminthes.

Anthropurgic foci are formed with dogs (definitive hosts) and farming animals (intermediate hosts). The infection is widely distributed in most parts of the world where sheep are raised and dogs are used to herd livestock. Infected dogs contaminate pasture with eggs of Echinococcus granulosus. Sheep and goats become infected by eating grass with eggs. Sometimes human feed the dogs with internal organs of ill animals. When a dog ingests infected meat containing cysts, the life cycle of Echinococcus granulosus is completed.

Human can get Echinococcosis in natural and антропургический foci. Susceptibility to Echinococcosis is common.

Pathogenesis. Negative influence of helminth on human body is result of mechanical and toxico-allergic affect. Echinococcus cyst can press host's tissues even up to their death. In case of intrahepatic localization cyst may compress big biliary ducts and cause cholestasis. It may also compress big hepatic vessels and lead to portal hypertension. Rupture of a cyst may result in anaphylactic shock due to sensitization of human organism by massive releasing of foreign proteins.

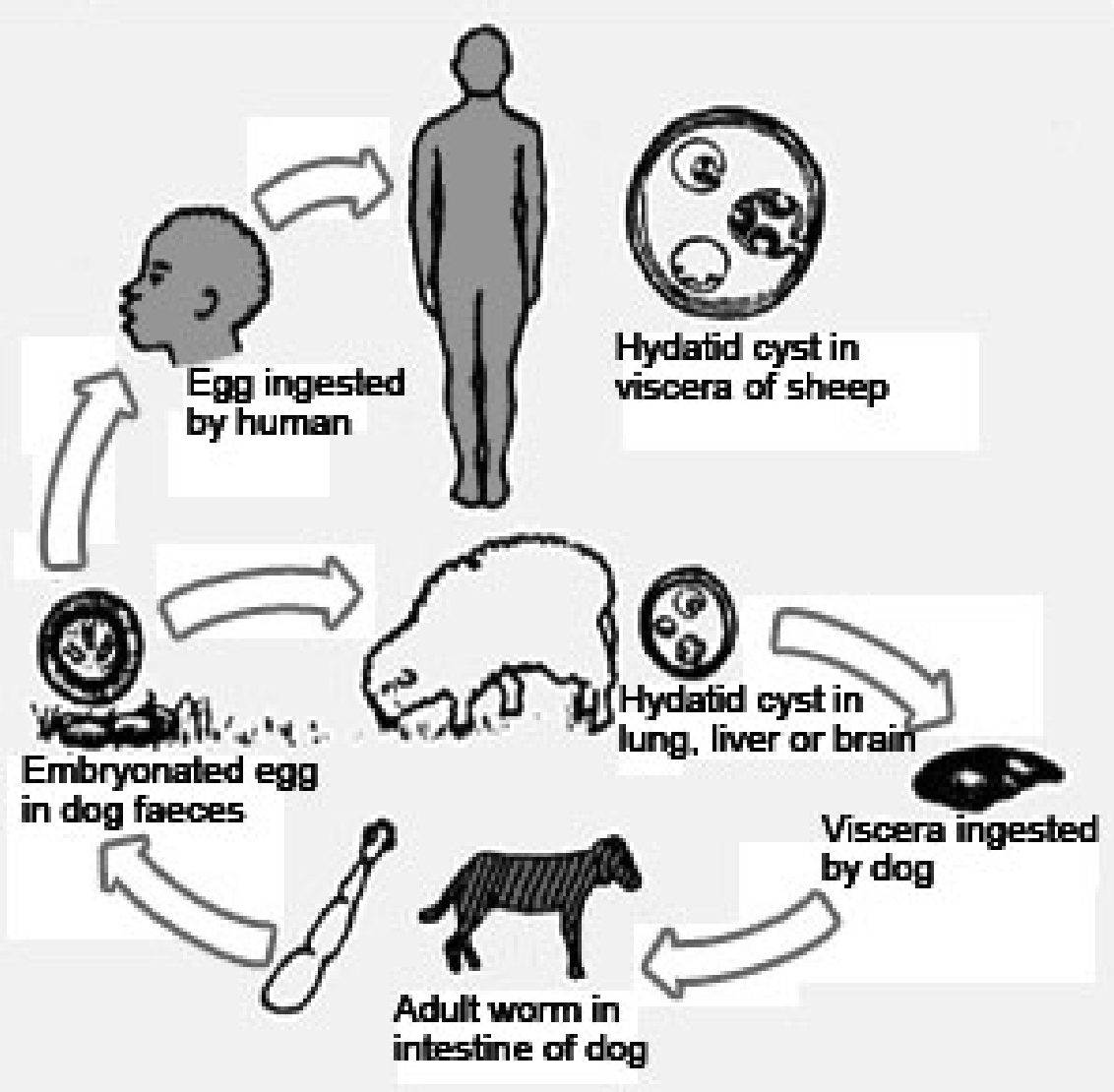
Clinical Manifestations. Since a period of years elapses before cysts enlarge sufficiently to cause symptoms, they may be discovered incidentally on a routine x-ray or ultrasound study. Clinical manifestations depend on localization and size of the cysts. The liver and the lungs are the most common sites of these cysts.

Patients with hepatic echinococcosis can have lost of appetite, belching, abdominal pain. Cyst can be palpable in the right upper quadrant in case of its superficial localization in low parts of right lobe of the liver. Compression of a bile duct or leakage of cyst fluid into the biliary tree may mimic recurrent cholelithiasis, and biliary obstruction can result in jaundice. Rupture of or episodic leakage from a hydatid cyst may produce fever, pruritus, urticaria, eosinophilia, or anaphylaxis. Hydatid cysts may become secondarily infected with bacteria presenting as a hepatic abscess.

Features of lung involvement depend on localization of the cyst. If it is close to pleura, pain syndrome is the early manifectation. If it localizes close to pulmonary trunk, patient can have cough, haemoptysis, dyspnoea.

Other presentations are due to the involvement of bone (invasion of the medullary cavity with slow bone erosion producing pathologic fractures), the CNS (space-occupying lesions with intracranial hypertension and epilepsy), the heart (conduction defects, pericarditis), and the pelvis (pelvic mass).

ECHINOCOCUSS



Complications. One of the most severe complications is rupture of echinococcus cyst, which can occur spontaneously or at surgery and may lead to multifocal dissemination of protoscolices, which can form additional cysts. Rupture of the cyst may also lead to allergic manifestations such as pruritus, oedema, dyspnoea, anaphylactic shock and appearance of peritonitis, pleuritis, meningitis or other inflammatory reactions.

Specific diagnostics. A number of serological tests have been developed for detection of antibody to specific echinococcal antigens, for example, an immune-enzyme analysis, a Western blot assay. Cysts in the liver elicit positive antibody

responses in 90% of cases, whereas up to 50% of individuals with cysts in the lungs are seronegative. That's why a negative test does not exclude the diagnosis of echinococcosis. Another drawback in serological diagnostics is cross-reactivity with sera of patients with Taenia solium.

Radiographic and related imaging studies are important in detecting and evaluating echinococcal cysts. Plain x-rays will define pulmonary cysts of E. granulosus – usually as rounded masses of uniform density – but may miss cysts in other organs unless there is cyst wall calcification (as occurs in the liver). MRI, CT, and ultrasound reveal well-defined cysts with thick or thin walls. However, the most pathognomonic finding, if demonstrable, is that of daughter cysts within the larger cyst. This finding, like eggshell or mural calcification on CT, is indicative of E. granulosus infection and helps to distinguish the cyst from carcinomas, bacterial or amebic liver abscesses, or hemangiomas. In contrast, ultrasound or CT of alveolar hydatid cysts reveals indistinct solid masses with central necrosis and plaquelike calcifications.

Examination of sputum, duodenal juice, faeces can be done to find protoscolices in case of rupture of the cyst into hollow organs.

In general blood analysis can be seen nonpermanent eosinophilia to 15 % and increased ESR.

Treatment. Therapy for cystic echinococcosis is based on considerations of the size, location, and manifestations of cysts and the overall health of the patient. Surgical removal of hydatid cysts remains the treatment of choice in many countries. In some countries PAIR (*p*ercutaneous *a*spiration, *i*nfusion of scolicidal agents, and reaspiration) is now recommended instead of surgery. PAIR is contraindicated for superficially located cysts (because of the risk of rupture), for cysts with multiple thick internal septal divisions, and for cysts communicating with the biliary tree. For prophylaxis of secondary peritoneal echinococcosis due to inadvertent spillage of fluid during PAIR, the administration of albendazole (15 mg/kg daily in two divided doses) should be initiated at least 4 days before the procedure and continued for at least 4 weeks afterward. PAIR, when implemented by a skilled practitioner, yields rates of cure and relapse equivalent to those following surgery, with less perioperative morbidity and shorter hospitalization.

Surgery is the treatment of choice for complicated *E. granulosus* cysts (e.g., those communicating with the biliary tract) or for areas where PAIR is not possible. For *E. granulosus*, the preferred surgical approach is pericystectomy, in which the entire cyst and the surrounding fibrous tissue are removed. Albendazole should be administered adjunctively, beginning several days before resection and continuing for several weeks after it.

Chemotherapy. Benzimidazole compounds have been shown to be effective against hydatid disease. The administration of 3-4 courses of albendazole in a dose of 10 to 15 mg/kg body weight per day (divided in two doses) for 28 days with drug-free periods of 2 weeks is used. This regime cures approximately one-third of cases of liver hydatid disease and causes partial regression of cysts in another third of patients. Small liver or lung hydatid cysts may be treated with albendazole. Albendazole is also indicated, when surgery is contraindicated. Mebendazole may

also be used, although it is less effective than albendazole. Albendazole, mebendazole, and other benzimidazole compounds should not be used in pregnant women because of their potentially teratogenic effects. Since benzimidazoles are potentially hepatotoxic, liver enzymes should be monitored before and during treatment.

Regular medical check-up of patient with Echinococcosis after operation lasts not less than 8 years. Patients should be examined by different doctors (gastroenterologist, pulmonologist, neuropathologist and others, dependently on prior localization of cyst) not less than 1 time in two years. Instrumental and serological examination should be done to them.

HYMENOLEPIASIS NANA Infection with H. nana, the dwarf tapeworm, is the most common of all the cestode infections. H. nana is endemic in both temperate and tropical regions of the world. Infection is spread by fecal/oral contamination and is common among institutionalized children.

Etiology and Pathogenesis. *H. nana* is the only cestode of humans that does not require an intermediate host. Both the larval and adult phases of the life cycle take place in the human. The adult—the smallest tapeworm parasitizing humans—is 2 cm long and dwells in the proximal ileum. Proglottids, which are quite small and are rarely seen in the stool, release spherical eggs 30–44 m in diameter, each of which contains an oncosphere with six hooklets. The eggs are immediately infective and are unable to survive for >10 days in the external environment. When the egg is ingested by a new host, the oncosphere is freed and penetrates the intestinal villi, becoming a cysticercoid larva. Larvae migrate back into the intestinal lumen, attach to the mucosa, and mature into adult worms over 10–12 days. Eggs may also hatch before passing into the stool, causing internal autoinfection with increasing numbers of intestinal worms. Although the life span of adult *H. nana* worms is only 4–10 weeks, the autoinfection cycle perpetuates the infection.

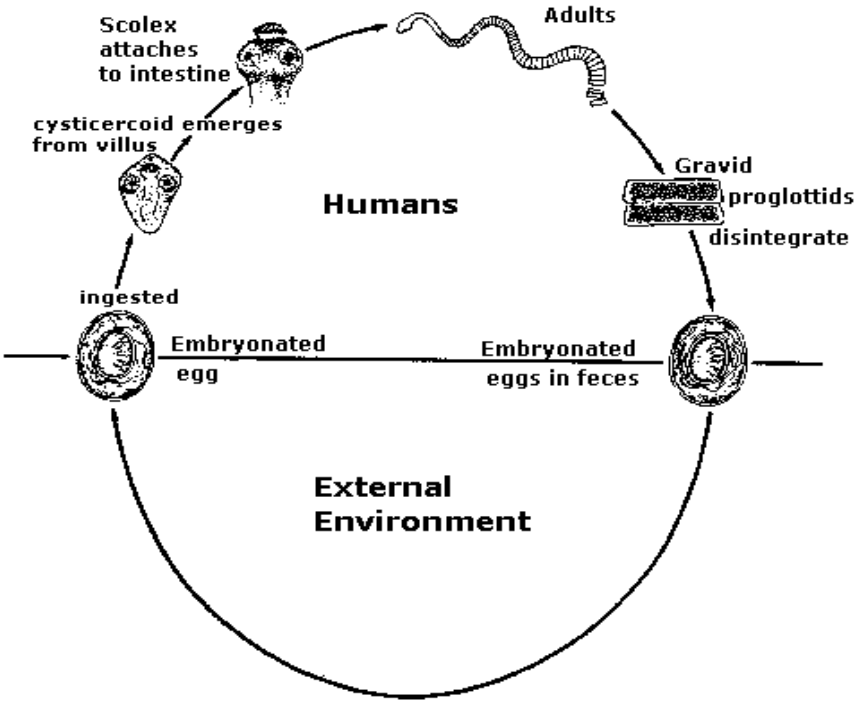
The life cycle of Hymenolepis nana starts, when microscopic eggs are passed with the stool of an infected human. They then get ingested either by rodents, humans (definite hosts) or insects (intermediate hosts). If a person ingests eggs (from contaminated fingers, water, food or soil), oncospheres (hexacanth larvae) hatch in the small intestine.

A larva penetrates an intestinal villus and develops into a cysticercoid. A cysticercoid develops to look more like an adult having a scolex (head) and a neck. It bursts out of the villus, attaches to the intestinal mucosa and matures into an adult in the last part of the small intestine, ileum. Its long neck starts producing segments, proglottids, which make up the body.

A proglottid absorbs nutrients from the surroundings and grows bigger before it detaches from the tail. Each proglottid has both male and female reproductive organs. It copulates with itself or with other proglottids of the same individual or nearby tapeworms. A gravid proglottid releases thousands of eggs

through its genital atrium or when its membrane disintegrates. Eggs are immediately infective when passed with the stool and cannot survive more than 10 days in the external environment.

Hymenolepis nana (dwarf tapeworm)



Clinical Manifestations. *H. nana* infection, even with many intestinal worms, is usually asymptomatic. When infection is intense, anorexia, abdominal pain, and diarrhea develop.

Some proglottids release eggs or disintegrate themselves already in the small intestine. Larval oncospheres (hexacanth embryo) might hatch prematurely and penetrate villi without leaving the body resulting in autoinfection. Hymenolepis nana does not necessarily need an intermediate host to complete its life cycle. Larvae can develop in spite of the high temperature of a human body. Adults live 4–6 weeks, but internal autoinfection allows hymenolepiasis to persist for years.

Hymenolepiasis is usually asymptomatic in adults. But prolonged infection or multiple tapeworms especially in children can cause more severe symptoms. The worms eat your food and cause inflammation of the intestinal mucosa. The inflamed tissue will have a reduced ability to absorb nutrients. People with little food to begin with and those who are weakened by other diseases suffer the most. Hymenolepiasis symptoms sometimes include: anal itching, diarrhea (can be bloody), headache, increased appetite or loss of appetite, insomnia, muscle spasms nausea, nervousness, seizures, stomach ache, vomiting, weakness, weight loss.

Diagnosis. Infection is diagnosed by the finding of eggs in the stool.

Treatment. Praziquantel (25 mg/kg once) is the treatment of choice, since it acts against both the adult worms and the cysticercoids in the intestinal villi. Nitazoxanide (500 mg bid for 3 days) may be used as an alternative.

Prevention. Good personal hygiene and improved sanitation can eradicate the disease. Epidemics have been controlled by mass chemotherapy coupled with improved hygiene.

HYMENOLEPIS DIMINUTA, a cestode of rodents, occasionally infects small children, who ingest the larvae in uncooked cereal foods contaminated by fleas and other insects in which larvae develop. Infection is usually asymptomatic and is diagnosed by the detection of eggs in the stool. Treatment with praziquantel results in cure in most cases.

TREMATODOSES

Class flukes (Trematodoses) includes the agent of fascioliasis, fascipsidosis, clonorchiasis, paragonimiasis, schistosomiases, opisthorchiasis.

These trematodes belong to the Opisthorchidae. Some species of the genus Opisthorchis cause infections in humans occasionally while other species do so quite frequently. Opisthorchis felineus has endemic foci in water reservoirs and river deltas. It is a common parasite in Siberia. Snails of the genus Bythinia are its first intermediate host. Normal final hosts include dogs, cats and pigs. The adult worm is found in the bile ducts. Recurrent cholangitis may occur. People become infected by eating an infected fish.

Opisthorchis viverrini is common in humans in North Thailand. Bithynia snails are the first intermediate host. Various freshwater fish are intermediate hosts. The adult worm is found in the bile ducts where it is responsible for recurrent cholangitis, but the majority of infections are subclinical.

Opisthorchis sinensis (previously called Clonorchis sinensis) is a very common parasite in Southeast Asia. The adult worms are found in the biliary tract or the Wirsung duct in the pancreas. The first intermediate hosts are snails (Bythinia, Assiminea, Melanoides, Parafossarulus). Subsequently fish become infected. Cats, dogs, pigs and fish-eating carnivores are the normal final hosts. People become infected by eating infected fish. The parasitosis is a problem not only because of the direct damage to the biliary tract and the risk of pancreatitis, but also due to the risk of bile duct carcinoma (cholangiocarcinoma) for those infected.

Etiology. Organisms: are Platyhelminth trematodes. Adult is 10-25 mm long x 3-5 mm wide. Reservoir: cats and dogs. Intermediary hosts: fresh water snail and fish. Magnitude: –13.5 million infected worldwide. Up to 26 % of Asian immigrants in USA have liver flukes. Acquisition: ingestion of fresh-water fish, raw, pickled, smoked, or dried.

Epidemiology. The natural-focal zoonosis. Biohelmintiasis. The definitive hosts are human and animals (cats, dogs, pigs). The intermediate hosts are fresh

water mollusk from the family Bithynia; fishes from the family. The factors of the transmission is fish with larvae (metacercariae).

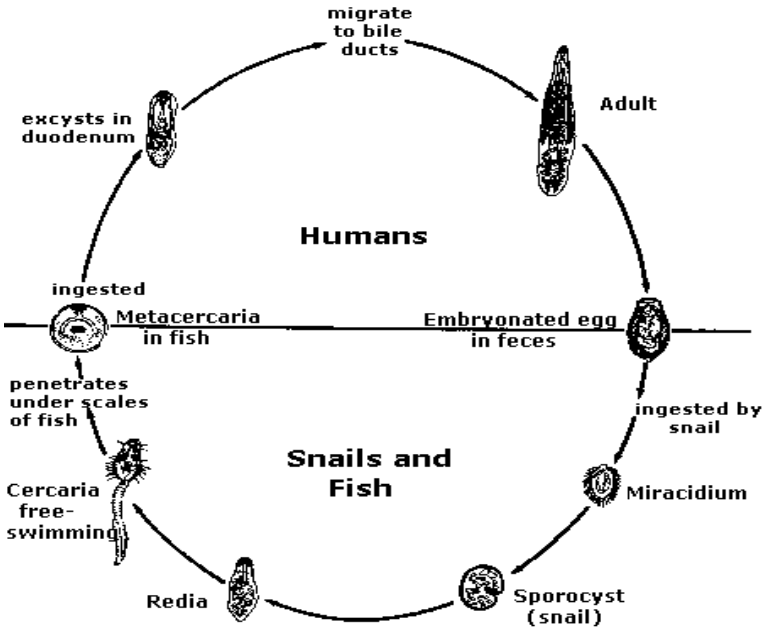
Life Cycle: Water is contamination. Eggs operculate an release miracidia.

Miracidia is eaten by fresh-water snail and changes to sporocyst then to rediae.

Rediae matures to cercaria and is released into the water and penetrates the skin of a fish transforming into a cyst that matures to metacercaria. Human eats fresh water fish. Metacercaria excyst in duodenum and migrate inside the lumen into biliary duct, GB, and pancreatic duct. Then matures to adult and lays eggs after 4 weeks.

Metacercariae penetrates to bile ducts, gall bladder, intrahepatic bile ducts and ducts of the pancreas

Early acute phase characterized toxic action of the products of the destruction of larvae, toxic-allergic reactions, lesion of the walls of bile ducts and ducts of pancreas. Late chronic phase characterized allergic action of helminthes, toxic and mechanic actions.



Clinical Manifestations. Acute phase. Light infection are asymptomatic but may be repetitive and cause heavy parasite burden. Heavy infections cause symptoms for less than 1 month, including fever, diarrhea, epigastric pain, anorexia, tender hepatomegaly, and sometimes jaundice. Leukocytosis and eosinophilia are very common. Eggs appear in stool 1 month after infection.

Chronic phase is due to the invasion of adult worms in the biliary tree, pancreatic duct, and/or GB. If < 100 flukes, usually asymptomatic. Moderate infestations (< 1000) cause anorexia, nausea, abdominal fullness and distress.

Severe infestations (1000-20000) cause intrahepatic and extrahepatic biliary obstruction. May have repetitive cholangitis. Sometimes worms migrate into the liver. Cholecystitis may occur. Secondary biliary cirrhosis may develop. Patient may have jaundice and hepatosplenomegaly. Patients have higher risk development of cholangiocarcinoma, proportional to the parasite burden. Patients may develop cholangiohepatitis with formation of intraductal strictures and stones, and with recurrent cholangitis.

Complications: abscessis of the live, cholangitis, rupture of parazitive cyst, peritonitis, primary cancer of the liver.

Diagnosis. Specific diagnosis: Holding of ovoscopy of feces or duodenal aspirate. Need to use concentration techniques. Serum Immunoblot has 92% sensitivity for active infection. Monoclonal ELISA test sensitive and specific to 100%. Non-specific diagnostics: eosinophilia, leukocytosis, biochemical methods, USD, cholecystography.

Treatment. Praziquantel 75 mg/kg divided in 3 doses x 1 day. In very heavy clonorchiasis, 2 days of therapy may be needed. Pathogenetic therapy.

Prophylaxis. The guarding of the water reservoirs. Sanitary control of the processing of the fish products.

Appendix№1

Professional algorithm for formation practical skills of the diagnostics of the nematodoses (ascariasis, trichuriasis, trichinosis, enterobiasis (pinworm), filariases, ancylostomiases (hookworms), strongyloidiases, dracunculiasis).

Professional algorithm for formation practical skills of the diagnostics of the nematodoses (ascariasis, trichuriasis, trichinosis, enterobiasis (pinworm), filariases, ancylostomiases (hookworms), strongyloidiases, dracunculiasis).

№	Task		
1.	Possess of the methods of clinical examination of the patient with nematodoses	1. To elucidate complaint of the patient.	To determine complaints of the patient, which are typical for syndromes: - general intoxication - allergic manifestations - disorders of gastrointestinal tract . To pay attention on appearance and dynamics of: - fever; - irritation; - headache; - disorder of the sleep; - disorder of the stool; - rash; - pain in the epigastrium area
2.	To take the history	2.To take the history (anamnesis) I. Anamnesis of the disease II. Life history III. Epidemiological anamnesis 3. Objectively examination	To establish the diseases in the last, anamnesis of vaccination. Use into food dirty fruits and vegetables, insufficiency thermal processing pork, beef or fresh water fish, water plants; presence in the endemic areas (opisthorchiasis) Remember: presence, dynamics of the symptoms depend from the period of the disease, severity of the course, age of the patient, concomitant pathology Pay attention on: - temperature;

			- edema of the face; - itching; - skin rash; Pay attention on: - abdominal pain; - decreased of appetite, heartburn; - hepatomegaly; - meteorism; - jaundice; - symptoms of peritonitis; Pay attention on: - tachycardia;
3.	Prescribe laboratory and instrumental investigations, interpret the results of investigations	1. 1. blood count. 2. urinanalyses. 3.investigation of feci 4. biochemical methods 5. ultrasound investigation 6. cholecystography 7. fibro-esphago-gastro-duodenoscopy I. General examination: - skin, mucous membranes; II. Gastrointestinal tract: - tongue;- percussion and palpation of abdomen; III. Cardiovascular system: IV. Nervous system	Pay attention on anemia, leukocytosis, eosinophylia. Finding of the eggs or proglottids in feci Dysproteinemia, changes of the biochemical tests (increase of total bilirubin and it's fractions, ALT, AST and other) - decrease of arterial pressure; - muffed heart sound. - disorder of heart rhythm; - sleeplesness - irritation - fatigue - dizziness - headache

ASCARIDOSIS

Etiology Ascaris lumbricoides

Epidemiology Anthroponosis. Peroral helmints.

The mechanism of the transmission is fecal-oral

Pathogenesis	Early (migratory) phase		Late (intestinal) phase
Clinics ↓	↓	↓	↓
General toxic syndrome	Allergic syndrome	damage of the respiratory tract	damage of the gastrointestinal tract
– increase the temperature – arthralgia - mialgia	– itch - skin rash	– bronchitis – pneumonia – rhinopharingitis - Leffler's syndrome	– decrease of appetite – nauseas – meteorism – disorder of the function of intestine – abdominal pain – fatigue, bad sleep – headache

Complications

– acute appendicitis
– mechanic jaundice
– purulent cholecystitis
– abscess of liver
– pancreatitis
– obstruction of the intestine (till perforation)
– perforation of the intestine, peritonitis
– asphyxia

Diagnostics

– leukocytosis
– eosinophilia (30-40%)

Specific diagnostics

- larvae in the sputum
- ovoskopy
- serological methods (RIHA, immunofermentive method)

Treatment

– Albendasolum
– Mebendazole
– Antihistaminic remedies
– **Prophylaxis** the personal hygiene; sanitary-hygienic measures

ENTEROBIOSIS

Etiology	Enterobius vermicularis
Epidemiology	Anthroponosis. Peroral contageous helminthiasis. The mechanism of the transmission is fecal-oral
Pathogenesis	The infection is realized in oral cavity The maturation of the invasive eggs with larvae is finished in 4-6 hours on the skin of the human. Larvae in the distal section of the small intestine → rectum (female) → eggs on the perianal area and perineum The mechanic lesion of the mucous membrane → inflammatory changes, hemorrhages
Clinical manifestations	– itch – pyodermia – headache – disorder of sleep – irritation – dizziness
Compilations	– appendicitis – perforation with peritonitis
Diagnostics	– perianal scrape

Treatment

Etiotropic – mebendazole
(treatment is effective only in observance of the rules of the personal hygiene)

Prophylaxis

– dehelmintization of the patients
– sanitary-hygienic measures
in the foci chemioprophylaxis is performed

TRICHOCEPHALOSIS

Etiology Trichocephalus trichiuris

Epidemiology Anthroponosis. Peroral biochelminthiasis

The mechanism of transmission is fecal-oral

The factors of transmission are vegetables, fuits, water

The infection of the human is realized in use

with the food only matured eggs

Pathogenesis The eggs with invasive larvae → penetrations into the mucous membrane of the small intestine → the large intestine

↓ ↓

Traumatization of the wall of the large intestine → admixture of the bacterial flora → inflammatory reaction → formation of the infiltrates (typhlitis)

Penetration into the mucous and submucous layers → constant irritation of the interoreceptors → disorders of the function of the stomach, duodenum and central nervous system

Clinical manifestations

↓ ↓

- diarrhea
- hemocolitis
- tenesmus
- pain in the right hypogastrium
- hypochromic anemia
- weakness
- dizziness
- decrease of artherial pressure tachycardia

Compilations
- rectal prolapse
- appendicitis
- hypochromic anemia

Diagnostics
- eosinophilia
- hypochromic anemia
- finding of the eggs of helminths in feci

Treatment
- albendazolum
- mebendazolum

Prophylaxis

- detection and treatment of the patient
- sanitary-hygienic measures
- washing of vegetables and fruits

ANCYLOSTOMIDOSES (ANCYLOSTOMOSIS AND NECATOROSIS)

Etiology	Ancylostoma duodenale / Necator americanus	
Epidemiology	Anthroponosis. Percutaneous and peroral heohelminthiases Main route of transmission - percutaneous The susceptibility is general	
Pathogenesis	**Early (migratory) phase**	**Late (intestinal) phase**
	↓	↓
	– allergic reaction – perivascular infiltration of the lungs	Fixation of ancylostoma to mucous membrane of the small intestine (hypoalbuminemia)
	↓	↓
Clinical manifestations	– itch, urticar rash, increase the temperature, cough with asthmatic component (sputum with blood)	– nausea – vomiting – pain in the epigastrium – diarrhea – anemia is leading symptom (in 3-6 month) – pain in the muscles – dry skin – edema of the low extremeties
Compilations	– infection of the skin in the area of the entrance gate → sepsis, tetanus, erythypilas, carbuncule – Quinke's edema, laryngospasm	– hypochromic anemia
Diagnostics	**General methods**	**Biochemical methods**
	– leukocytosis – eosinophilia – increase the ESR – hypochromic anemia	– decrease general protein – general of serum Fe
	Specific methods: finding of the eggs of helminths in the feci	

Treatment

Etiotropic therapy: albendazole, levamizole, mebendazole, thiabendazole

Pathogenetic therapy: treatment of anemia and hypoalbuminemia, desensibilization

Prophylaxis dehelmintization of the patients sanitary-hygienic measures devastation of soil

STRONGYLOIDIASIS

Etiology — Strongyloides stercoralis

Epidemiology — Anthroponosis.

Percutaneous and peroral geohelminthiasis

The factors of the transmission are soil, vegetables, fruits

The contingents of risk is miners, earthworkers

Pathogenesis — In infection through the skin larvae → into the tissue → blood and lymphatic vessels → heart → lungs → oral cavity → intestine

Clinical manifestations

Migratory phase	**Intestinal phase**
– allergic and general toxic reactions – skin itch – rash – Leffler's syndrome	– damage of mucous membrane – disorder of the absorbtion of the fats and – hypoproteinemia

Compilations

- ulcerous damage of the intestine
- perforative peritonitis
- necrotic pancreatitis
- intestinal hemorrhages
- myocarditis
- meningoencephalitis
- chaxexia

Diagnostics

General clinical methods	– eosinophilia (till 40%) is marked in early phase – anemia, accelerated ESR are marked in late phase
Biochemical methods	– dysproteinemia – increase level of bilirubinum
Specific methods	– finding of the larvae in sputum, duodenal secretum, feci

Treatment

Migratory phase	Intestinal phase
– pathogenetic therspy (antiallergic remedies)	– thiabendazole

Prophylaxis

- detection and treatment of the patient
- general sanitary mezures
- in endemic areas it is necessary to use only boiling water
- defense of the open parts of the body from larvae
- devastation of the soil (10% NaCl, mineral fertilizations)

Trichinellosis

Etiology Trichinella spiralis

Zoonosis. Peroral biohelminthiasis.

Epidemiology The sourses of infection

↓ ↓

Natural foci (beers, wolfs, foxes) Anthropurgic foci (pigs, dogs, cats)

The factor of the transmission is insufficient thermal processing of the meat

Immunity is no prolonged. It is possible repeated infection

Pathogenesis Larvae are freed from the cyst walls in the stomach and in the small intestine by acid-pensis digestion → blood → skeletal muscles (incapsuling) → A cyst wall develops around the larva and may eventually calcify. Larvae may remain viable for several years..

The leading factor of pathogenesis – sensilization → allergic vasculitis

Clinical manifestations

- enteritis, nausea, abdominal pain
- periorbital edema and edema of the face
- mialgia
- fever
- eosinophylia (50-90%)
- skin rash

Compilations

- myocarditis
- pneumonia
- meningoencephalitis
- hepatitis
- nephritis
- systemic vasculitis
- thrombophlebitis
- thrombocytopenia

Diagnostics

- trichinelloskopy (investigation of the meat)
- immunological methods
- skin allergic test

Treatment

Etiotropic

- vermox
- thiabendazole

Pathogenetic

- desensibilizative remedies
- desintoxication therapy

Prophylaxis

- investigation of the meat of presence for Trichinella spiralis

Appendix№2

Professional algorithm for formation practical skills of the diagnostics cestodoses (diphyllobothriasis, hymenolepiasis, echinoccosis ,pork warm disease and cystecercosis, beef worm disease), and trematodoses (fascioliases, opisthorchiasis)

№	Task		
1.	Possess of the methods of clinical examination of the patient with cestodoses and / or trematodose s	1. To elucidate complaints of the patient.	To determine complaints of the patient, which are typical for syndromes: - general intoxication - allergic manifestations - disorders of gastrointestinal tract .
2.		2. To take the history (anamnesis) I. Anamnesis of disease	To pay attention on appearance and dynamics of: - fever; - irritation; - headache; - disorder of sleep; - disorder of stool; - rash; - pain in the epigastrium area To establish the diseases in the last, anamnesis of vaccination. Use into food dirty fruits and vegetables, insufficiency thermal processing pork, beef or fresh water fish, water plants; contact with dogs; presence in the endemic areas (opisthorchiasis)
		II. Life history	Remember: presence, dynamics of the

			symptoms depend from the period of the disease, severity of the course, age of the patient, concomitant pathology
		III. Epidemiological anamnesis	
			Pay attention on: - temperature; - edema of the face; - itching; - skin rash;
			Pay attention on: - abdominal pain; - decreased of appetite, heartburn;
		3. Objectively examination	- hepatomegaly; - meteorism; - jaundice; - symptoms of peritonitis;
			Pay attention on: - tachycardia; - decrease of arterial pressure; - muffed heart sound. - disorder of heart rhythm;
		I. General examination: - general state of the patient; -skin, mucous membranes;	- sleeplesness - irritation - fatique - dizziness
		II. Gastrointestinal tract: - tongue;	- headache

		- percussion and palpation of the abdomen; III. Cardiovascular system: IV. Nervous system	
3.	3. Prescribe laboratory and instrumental investigations, interpret the results of the investigations	1. blood count. 2. urinanalysis 3.investigation of feci 4. biochemical methods 5. ultrasound investigation 6. cholecystography 7. fibro-esphago-gastro-duodenoscopy	Pay attention on anemia, leukocytosis, eosinophylia. Finding of the eggs or proglottids in feci Dysproteinemia, changes of the biochemical test (increase of total bilirubin and it's fractures, ALT, AST and other)

THE BEEF TAPEWORM

Etiology Taenairhynchus saginatus

Epidemiology Anthroponosis. Peroral biohelmintiasis.

The source of the invasion and definitive host is human. The intermediate host is cattle.

The factor of the transmission is insufficient thermal processing of the meat

Pathogenesis

cysticercus → small intestine → larvae fixate to mucous membrane

↓ ↓

In 2 month 2 месяца → adult worm → discharge of proglottids filled by eggs

Mechanic action

– lesion of the mucous membrane in the site of the fixation of parazite
– irritation of the interoreceptors
– disorders of the absorbtion
– intoxication
– sensibilization

Clinical manifestations

– abdominal pains (especially right hypogastrium area)
– nauses
– meteorism
– disorders of appetite
– disorders of stool
– hypoacidic gastritis
– it is typical the active exit of proglottids act of the defication (especially at the night)

General toxic symptoms

– weakness
– fatique
– disorders of the sleep
– irritability
– headache
– dizziness

Complications

– acute appendicitis acute pancreatitis acute cholecystitis
– occlusive ileus

Diagnostics

General clinical methods

– moderately eosinophilia
– moderately anemia

Specific methods

– revealation of the eggs and proglottids of helminth in the feci

Treatment

– niclosamide paromomycin praziquantel

Prophylaxis

– dehelminthization of the patients
– devastation of the soil
– veterinary-sanitary control
– the maintenance of the roles of the terminal processing of the meat

PORK TAPEWORM AND CYSTICERCOSIS

Etiology Taenia solium

Epidemiology Anthroponosis.

Peroral biohelminthosis

The source of the invasion and definitive host is human. The intermediate hosts are pigs, dogs, cats, sheep

The factors of transmission is use into the food meat of the pig with cystecercs

Pathogenesis Taenia solium arised in use of the meat with larvae of the parasite. The adult helminth parasites in the organism of human. The human is the definitive host. Cysticercosis arises in the penetration of the eggs of helminth into the intestine. The human is an intermediate host

Taenia soleum	Cystecercosis
In infection by cystecercs in 2-3 months → adult helminth → small intestine	In infection by eggs of T. soleum in 2-2,5 month → cystecercs → brain, myocardium, skeletal musculature, eyes
↓	↓
Mechanic influence	– compression of the tissues – inflammatory process around parasites – toxic and allergic action
– the lesion of the mucous membrane in the site of the fixation of parasite – irritation of interoreceptors – disorder of the process of absorbtion – intoxication – sensibilization	

Clinical manifestations

– moderate marked dyspeptic manifestations and general toxic syndrome	– the clinical manifestations is dependence on localization (brain, eyes, subcutaneous, cellulose)

Diagnostics General clinic methods moderate eosinophilia

Taenia soleum – finding of the helminth's eggs in the feci
– ovoscopy

Cystecercosis In dependence on localization (ophtalmoscopy, roentgenogram, computed tomography)

Serological methods ELISA

Treatment ***Taenia soleum***- Filicis maris acterium

Cystecercosis - surgical

– **Prophylaxis** Relevation and treatment of the patients: general sanitary measures; veterinary control

DIPHILLOBOTHRIASIS

Etiology (Diphylobotrium latum)

Epidemiology Zoonosis. Peroral biohelminthiasis

The source of the infection and definitive host is human.

Intermediate host is a copepod (crustacean)

The ancillary host is a fresh water fish

Infection of the human is realized only in the use into the food of the thermic insufficiency cooking fish

Pathogenesis

Mechanic influence	Toxicoellergic influence
Attachment to the intestinal wall → attachment by bot to mucous membrane → athrophy and necrotization of the mucous membrane	Sensibilization by products of the metabolism of helminth → endogenic hypo- and avitaminosis B_{12} and folic acid
↓	↓

Clinical manifestations

– weakness – dizziness – signs of the damage of the gastrointestinal tract – it is possible dynamic or occlusive obstruction ileus	– anemia – pallor – glossitis – loss of tonque papillae – achilia

Compilations

– anemia
– obstruction of the intestine (till perforation)

Diagnostics

– finding of the helminth's eggs in the feci
– hyperchromic anemia
– revealation of the eggs and proglotids of D.latum

Treatment

Etiotropic therapy	Pathogenetic therapy
– fenasal – Filicis maris acterium – praziquantel – vermox	– treatment of anemia

Prophylaxis

– treatment of the patients
– the quarding of the water reservoirs from the pollution by feci
– fish tapeworm infection is prevented by throught cooking of freshwater fish

ECHINOCOCCOSIS

Etiology Echinococus granulosus

Epidemiology Zoonosis. Peroral biohelmintisis.

There are 2 types of the foci:

1. The natural foci (wild animals: beers, elks, wolfs, foxes)

2. Synantropic foci (sheeps, goats, dogs) The definitive host is dog.

Pathogenesis Eggs of helminths → fixation on the mucous membrane of duodenum → oncosphere → V.portae → liver (or brain, heart, kidney) → formation of the hydatid cyst in the site of the penetration

↓ ↓

Toxico-allergic action of the metabolic products of Echinococcus

↓

The local damage

- compression of the tissues
- the disorders of trophics
- necroses, granulation
- replacement of the perished by connective tissue

Clinical manifestations

- weakness, fatique
- headache
- increase of the temperature
- dyspeptic manifestations
- skin rash, itch

Location of hydatid cysts:

Liver (usually in the right lobe)	– jaundice (cholestasis) – pain in the right part of the chest – pain in the right subribal area – on palpation – immovable compact formation
Lungs	– dyspnea – pain in the chests – cough
Brain	– clinics of the tumor

Complications The rupture of hydatid cyst → anaphylactic shock

The cyst may rupture:

1. into the biliary tract → cholangitis intermittent ductal obstruction

through the capsule of the liver → into the peritoneal cavity → dissemination of scolices → formation of daughter cysts

Complications – an acute inflammatory reactions (peritonitis, meningitis, pleurisy)
– the suppurating of the cysts → abscess
– in prolonged invasion → amyloidosis of the parenchymatous organs

echinococcosis of the liver

There are 3 stages
– mechanic jaundice
– cirrosis of the liver
– portal hypertension

echinococcosis of the lungs → massive hemorrhage

echinococcosis of the brain → epileptic attacks, loss of the vision, pareses, paralyses

echinococcosis of the ovaries → rupture of the salpinx → hemorrhage

Diagnostics
– leukocytosis
– anemia
– accelerated ESR
– eosinophylia
– roentgenograms
– USD
– computed tomography
– serological reactions (complement fixation, hemagglutination and other)
– a skin test (Casoni)

Treatment – the leading method of the treatment is surgical

Prophylaxis
– roles of the personal hygiene
– helminthological investigation and dehelminthization of the dogs

HYMENOLEPIASIS

Etiology Hymenolepis nana

Epidemiology Peroral contagenous helminthiasis.

The human is the main sourse of the invasion.

The human is the definitive and intermediate host

The ancillary hosts are rats

The mechanism of transmission is fecal-oral

The factors of the transmission

are vegetables, fruits, flies, cockroach

Pathogenesis eggs → stomach → penetration into the mucous membranes of the upper sections of the small intestine → larvae

(cyctecercoided) → eggs → environment

Mechanic lesion

– edema, hyperemia of the all layers of the intestine wall
– dystrophy, necrosis of the epithelium

The part stays in the intestine

(autoinvasion)

– the products of the helminth's metabolism cause
  – intoxication and allergization
  – damage of the fermentative system
  – dysbacteriosis
  – disorder of the functions of the liver

Clinical manifestations

The damage of the gastrointestinal tract

– nausea
– vomiting
– decreased appetite
– salivation
– abdominal pain
– diarrhea
– moderate hepatomegaly

The damage of the nervous system

– weakness
– fatique
– headache
– dizziness
– irritation
– seizures
– epileptic attacks

Sensibilization

– urticaric rash
– vasomotoric rhinitis
– asthmatic bronchitis
– Quinke's edema

Complications
– dysbacteriosis

Diagnostics
– moderate eosinophilia
– identification of the double membrane eggs in the stool

Treatment
– praziquantel niclosamide paramomycin

OPISTHORCHIASIS

Etiology — Opisthorchis felineus

Epidemiology — The natural-focal zoonosis. Biohelmintiasis

The definitive hosts are human and animals (cats, dogs, pigs)

The intermediate hosts are fresh water mollusk from the family Bithynia; fishes from the family

The factors of the transmission is fish with larvae

(metacercariae)

Pathogenesis

Metacercariae → bile ducts → gall bladder → intrahepatic bile ducts and ducts of the pancreas

↓ ↓

Early acute phase	Late chronic phase
Toxic action of the products of the destruction of larvae → toxicoallergic reactions → lesion of the walls of bile ducts and ducts of pancreas	– allergic action of helminths – toxic action – mechanic action

Clinic

↓ ↓

– diarrhea – allergic rash – pain in the right hypogastrium (in the patient which came to endemic regions; – asymptomatic course in the inhabitans of the endemic regions	– pain in the area of the liver and gall bladder – dyspeptic manifestations – hepatomegaly – ictercic sclerae

Complications
- abscessis of the liver
- cholangitis
- rupture of parazitive cyst
- peritonitis
- primary cancer of the liver

Diagnostics
- eosinophilia
- leukocytosis
- ovoscopy
- biochemical methods
- USD, cholecystography
- Serological methods in chronic stage

Treatment
- Praziquantel
- Pathogenetic therapy

Prophylaxis
- The guarding of the water reservoirs
- Destruction of mollusks
- Sanitary control of the processing of the fish products

FASCIOLOPSIASIS

Etiology — Fasciola hepatica

Epidemiology — Zoonosis with natural foci. Peroral biochelminthiasis.

The sourse of infection and definitive hosts are human, cattle, sheep, hourses, rodents.

The intermediate host is snail mollusks

The factors of the transmission are water flowers, vegetables

Pathogenesis — Penetration of larvae of F. Hepatica into intestine → liver and bile ducts, pancreas, brain, eyes

Acute migratory phase	Late (chronic) phase
- toxicoallergic action of the helminth's products - there are eosinophilic infiltration and necroses of the hepatocytes in the liver	– toxicoallergic action of helminth's products → mechanic action

Clinical manifestation

Acute phase	Subacute phase	Chronic phase
General toxic and allergic manifestations – increase of the temperature – skin rash – arthralgia – hepatomegaly, jaundice		– pain in the right hypogastrium area – enlargenment of the liver – diarrhea – loss of weight – disorders of the activity of the gastrointestinal tract – admixter of the secondary flora
Complications	– anemia – cachexia	– purulent meningitis – cholecystitis – flegmone of the gall bladder – abscess of the liver – mechanic jaundice – acute pancreatitis – fibrosis of the liver – myocarditis
Diagnostics	– eosinophilia anemia leukocytosis biochemical methods – the increase of the activity of ALT and AST – dysproteinemia specific diagnostics – serological methods (reaction complement fixation) – finding of the helminth's eggs in the feci	
Treatment	– chloxylum praziquantel antibiotics (in complications)	

Prophylaxis: don't use into the food water plants in the foci of fasciolopsiasis; boiling of the water; garding of water reservoirs from the pollution; sanation of the infected animals

Appendix№3

Control questions for self control:

1. What helminthiasis are treated to the class of the round warms?
2. What is the mechanism and factors of the infection in ascaridosis?
3. What are the phases of the pathogenesis in ascaridosis?
4. What are the clinical manifestations of the migratory phase in ascaridosis?
5. What are the complications of ascaridosis?
6. What is the blood count of the early (migratory) phase in ascaridosis?
7. What are the principles of the therapy of ascaridosis?
8. What are the principle ways of the infection in ancylostomidosis?
9. What etiotropic and pathogenesis therapy should be administered in ancylostomidosis?
10. What is the mechanism of the invasion in trichocephalosis?
11. What are the cardinal signs of trichocephalosis?
12. What methods are used for diagnostics of trichocephalosis?
13. What are the factors of the transmission of the infection in trichocephalosis?
14. What are the ways of the transmission in enterobiosis?
15. What are the criterions of the diagnosis in enterobiosis?
16. What is the source of the infection in Trichinellosis?
17. What is the mechanism of the invasion in Trichinellosis?
18. What are the cardinal signs of Trichinellosis?
19. What are the complications of Trichinellosis?
20. What methods are used for diagnostics of Trichinellosis?
21. What are the factors of the transmission of the infection in Trichinellosis?
22. What the features of the nutrition of Trichinellosis?
23. What are the principle criterions of the diagnosis in Trichinellosis?
24. What is life cycle of the agent of strongyloidiases?
25. What are the ways of the transmission in strongyloidiases?
26. What are the contingents of the risk for infection in strongyloidiases?
27. What are the main clinical symptoms in strongyloidiases?
28. What are the clinical stages of the diseases in strongyloidiases?
29. What are the complications of strongyloidiases?
30. What laboratory methods are used for diagnostics of strongyloidiases?
31. What therapy should be administered in strongyloidiases?
32. What is the source of the infection in Toxocarosis?
33. What is the mechanism of the invasion in Toxocarosis?
34. What are the visceral signs of Toxocarosis?
35. What are the complications of Toxocarosis?
36. What methods are used for diagnostics of Toxocarosis?
37. What are the factors of the transmission of the infection in Toxocarosis?
38. What are the principle criterions of the diagnosis in Toxocarosis?
39. What is the source of the infection in dirophylariosis?
40. What is the mechanism of the invasion in dirophylariosis?

41. What are the complications of dirophylariosis?
42. What methods are used for diagnostics of dirophylariosis?
43. What are the factors of the transmission of the infection in dirophylariosis
44. What therapy should be administered dirophylariosis?
45. What are the principle criterions of the diagnosis in dirophylariosis?
46. What helminthiases are regarged to the class “cestodoses”?
47. What is the definitive and intermediate host in diphillobothriasis?
48. What are the complications of diphyllobothriasis?
49. What is epidemiology of beef tapeworm?
50. What is the life cycle Taenia soleum (pork worm)?
51. What are the clinical manifestations of Taenia soleum?
52. What is the mechanism of invasion in cystecercosis?
53. What are the clinical manifestations in cystecercosis?
54. What is the life cycle of Hymenolepis nana?
55. What is epidemiology of hymenolepiasis?
56. What are the leading clinical syndromes in hymenolepiasis?
57. What is the etiology of echinoccosis?
58. What is the epidemiology of echinoccosis and contingent of risk?
59. What are the leading factors of the pathogenesis of echinoccosis?
60. What is the possible localization of cyst in echinoccosis ?
61. What are the clinical manifestations in dependence on localization of cyst in echinoccosis ?
62. What are the complications of echinoccosis ?
63. What methods are used for diagnostics of echinoccosis ?
64. What is the principal method of the treatment in echinoccosis ?
65. Which helminth a are regarded to the class of trematodoses?
66. What are the features of the life cycle trematodoses?
67. What is the epidemiology of opisthorchiasis?
68. What are the phases of the pathogenesis of opisthorchiasis?
69. What are the clinical manifestations of the late (chronic) stage of opisthorchiasis?
70. What are the complications of opisthorchiasis?
71. What methods are used for diagnostics of opisthorchiasis?
72. What are the clinical manifestations of fascioliases?
73. What treatment should be administered in trematodoses?
74. What methods are used for prophylaxis of cestodoses?
75. What methods are used for prophylaxis of trematodoses?

Appendix№4

Task for self control:

1. The patient of 42 years old complains on acute headache, tenderness of the muscles of the extremities. The general weakness, fever, edemas around the eyes developed a week ago. The physician diagnosed influenza and prescribed Amixin IC. An improvement didn't come. His wife also fell ill. She complaints of muscle pain, bad condition. They had eaten fried pork, bought 12 days ago. The temperature is 38, 3°C, the face is edematous. The muscles of extremities are painful. The abdomen is soft. Stool is 2 times per day. In the blood count: Нв-133 g/l, L-15,0, B-1 %,E-40 %, U-1 %,B-7 %,S-3%,L-8 %, M-6%, ESR-25 mm /h.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

2. The patient of 40 years old was referred with complaints of high temperature to 39°C, pains in the eyes and muscles. The disease began with general weakness, digestive disturbances. The patient had pork, bought from the neighbour 2 weeks ago. Objectively: there are edema of the face, plentiful exudative-papular eruption on the body, adynamia, symptoms of myocarditis. In the blood count eosinophilia (45 %) is marked.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

3. The patient of 42 years old complains of acute headache, tenderness of the muscles of the extremities. The general weakness, fever, edemas around the eyes developed a week ago. The physician diagnosed influenza and prescribed Amidopyrinum. An improvement didn't come. His wife also fell ill. She complaints of muscle pain, bad condition. They had eaten fried pork, bought 12 days ago. The temperature is 38,3°C. the face is edematous. The muscles of extremities are painful. The abdomen is soft. Stool is 2 times per day grueled. In the blood count: Нв-133 g/l, L-15,0, B-1 %,E-40 %, U-1 %,B-7 %,S-3%,L-8 %, M-6%, ESR-25 mm /h.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

4. The patient of 25 years old complains of weakness, nausea, pain in the right iliac area, stool 3-4 times a day, without pathological admixtures, periodically meteorism. She has been sick for 2 years. The sharp increase of the appetite, and following its decrease was marked in the beginning of the disease. At the last time she paid attention on discharge of the tape formations in defecation and in the bad in the morning. These formations have white color and size 1-2 cm in diameter. Epidemiological anamnesis: she is a cook. She frequently taste uncooked beef mince. Objectively: The skin and visible mucous membrane are pink. The tongue is coated with white fur. The peripheral lymphatic nodules are no palpated. The pulse rate is 72 beats per minute, rhythmical. Heart sounds are muffled. The lungs

are without peculiarities. The abdomen is soft, inflated and painless during palpation. The liver and the spleen are no palpated. There is eosinophilia and anemia in general blood analysis.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

5. The patient of 21 years old complains of pain in the epigastrium, heartburn, nausea, decreased appetite, weakness, loss of weight, irritability, constipations, alternated by diarrhea. She works as an accountant on a fish farm. On examination: paleness of her skin and mucous membranes, the tongue of bright red color with flatted papillas are reveled, the pains are on percussion of the breastbone. The liver and spleen are enlarged a little. There are decreased level of hemoglobin, erythrocytes of large sizes, color index – 1.2 in general blood analysis. The number of eosinophiles in increased. Gastric acholia is determined in the investigation of the gastric contents.

6. The woman of 36 years old is a worker of the fish plant. She came to the polyclinic with complaints of heartburn, unstable stool, weakness. The itching rash periodically developed on the body. On examination: her skin is pale, single elements of urticaric rash are marked. The stomach is soft, slightly painful around the umbilicus. On examination of the blood: hyperchromatic anemia, eosinophilia to 9% are marked.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

7. Gambia. A mother asks for advice. Her daughter’s faeces contained several worms. You ask the length of the parasites. What do you think if the mother replies 30 cm, or 1 cm, or 3 cm?

8. Is there a clinically important difference between *Taenia solium* and *Taenia saginata*? Is it possible to differentiate the eggs under a microscope?

9. Congo. A 29-year-old man has been coughing for five weeks. There is eosinophilia. Sputum for acid-fast bacilli is negative. Your colleague asks whether the man ate crabs a few months ago. What diagnosis is he considering?

10. Mexico. Epilepsy is common in the region where you work. Which parasitic cause needs to be ruled out? What would you advise as prevention?

11. Brazil. A woman has had problems for one week with a swollen, puffy face, chiefly around the eyes. Do you consider trichinellosis, Chagas’ disease or nephrotic syndrome? What do you do? Are there simple tests which can help in your diagnosis?

12. Vietnam. A man has diarrhoea. Examination of the faeces for parasites shows: "Countless eggs of *Trichinella spiralis*". What do you think and what do you do?

13. Northern Thailand. You are asked if eating raw fish is dangerous. What is your answer, what are your reasons?

14. Jamaica. A 15-year-old girl is suffering from anal itch. There are no haemorrhoids and repeated Scotch tape tests have shown no oxyurids. She has not noticed any *Taenia* proglottids. There are a few itching lines moving under the skin. What do you think and what do you do?

15. Tobago (Trinidad). Which worms lead to important anaemia?

16. Haiti. A girl has had fever for 2 months and is clearly emaciated. She coughs often. In the stools *Ascaris* eggs are observed. What do you think?

17. If all the snails in an area are destroyed, will this have an effect on nematode, trematode or cestode infections?

18. Do all the trematode infections transmitted by food involve hermaphrodite parasites?

19. Farouk is a deeply devout Muslim and works as an archaeologist in rural Mexico, together with his German friend Jurgen and his American colleague John. Jurgen is a vegetarian and John likes his daily portion of meat. Can Farouk and Jurgen develop cysticercosis? Can John?

20. See last question. If Farouk should develop cysticercosis, should he then ask himself whether he has sinned by eating “impure” pork?

21. Lesotho. A Swiss family of 4 people. The father suffers regularly from anal itch. He has noticed oxyurids and taken mebendazole (Vermox®). After a month the same symptoms return. The whole family is now treated with Vermox®. However, there is another relapse after 4 weeks. Do you now consider resistance, exogenous re-infection or incomplete treatment?

22. Congo. You suspect trichinellosis in a patient. A small muscle biopsy is surgically removed from the quadriceps. This muscle fragment is pressed between 2 glass slides. Can you look at the whole biopsy to find the encapsulated larvae with a simple magnifying glass or do you need a microscope?

23. Bolivia. You are working in the northern Altiplano, between Lake Titicaca and the capital. This is a region with many animals (sheep, cattle, pigs, goats, horses, donkeys, llamas, alpacas). Would this information be important to explain the high incidence of fasciolasis?

Appendix№5

Tests for self control

1) Where do the eggs of Ascaris lumbricoides develop into invasive stage? A. in intestines of human, B. in perianal folds during 4-6 hours, C. in the ground during 2 weeks by temperature 25ºC, D. in lungs, E. in liver of human.
2) The source of Enterobiosis invasion is infected: A. cattle, B. pig, C. human, D. cat, E. fish.
3) What is the main method of diagnostics of Enterobiosis? A. indirect hemagglutination test, B .immune-enzyme analysis, C coprogram, D. investigation the scrape from the perianal skin, E. coproculture.
4) For what helminthiasis percutaneous way of transmission is typical?: A. strongyloidiasis, B. necatoriasis, C. ankylostomiasis, D. B and C are correct, E. everything is right.
5) At serious ascariasis following surgical complications are typical, except:
A. asphyxia, B. suppurative cholangitis, C. rectal prolapse, D. intestinal bstruction, E. abscesses of the liver.
6) Which method of treatment of Enterobiosis is the most correct: A. anthelmintic drugs combined with strict hygiene regime, B. anthelmintic drugs ombined with daily night spasmolytic enemas, C. 3-4 courses of treatment with metronidazole, D. surgical treatment, E. daily night enemas with ammonia soda to prevent perianal itching.
7) Indicate which type of worms belongs to contagious?:
A. trichuriasis, B.teniasis, C. enterobiasis, D. opisthorchiasis, E. ascariasis.
8) The patient 30 years old has weakness, fever up to 39 ° C. On R-graphy eosinophilic lung infiltrates migratory nature were revealed. What disease the patient has? A. pulmonary tuberculosis, B. lobular pneumonia, C. ascariasis, migration phase, D. polisihmentarna pneumonia, E. lung cancer.
9) The patient complains of weakness, poor appetite, intermittent abdominal pain. In ovoskopy study of feces roundworm's eggs were found. What treatment should be prescribed to patient for deworming? A. hygienic treatment, B. levamisole, C. ceftriaxone, D. norfloxacin, E. fenasal.
10) The patient with ancylostomiasis has manifestation of iron deficiency anemia (decreased absolute number of red blood cells, decreased hemoglobin and color index). Indicate the cause of this clinical manifestation in ancylostomiasis? A. bleeding, B. hookworm are true hematophah, C. toxic-allergic action of ankilostom metabolites, D. perforation of vessels, E. violation vascular permeability.
11) In the specific diagnostics of trichinellosis the main is to detect: A. ovum in feces, B. helminthiasis in feces, C. larva in biopsy material, D. ovum in scraping from perianal folds, E. eosinophilia in blood.
12) The symptoms of which helminthic invasion are: acute onset, fever, muscular pains, conjunctivitis, allergic rash? A. echinococcosis, B. ascaridiasis, C. diphyllobothriasis, D. opisthorchiasis, E. trichinellosis.

13) The patient A. has headache, deltoid muscles pains, appearance of skin rash. The medical examination has detected painfulness of muscles of left shoulder joint. The eosinophilia in the blood is up to 40%. The rontgenography of left shoulder joint has shown calsifications (size 3mm x 1,2mm) with effect of perifocal inflammation in soft tissues of deltoid muscle. What disease is characterized by these clinical signs? A. nonspecific polyarthritis, B. trichinellosis, C. leptospirosis, D. toxicoallergic dermatitis, E. extrapulmonary tuberculosis.
14) Trans-dermal route of transmission is typical? A. trichocephalosis, B. teniasis, C. strongylosis, D. opisthorchiasis, E. ascariasis.
15) Leading syndrome in trichinellosis? A. immunosuppressive, B. toxic-allergic,C. adynamic – neurotic, D. dyspepsial, E. all not true.
16) The development of mature forms of Trichinella are: A. on the microvilli in the small intestine, B. in the striated muscle, C. in the submucosal layer of the small intestine, D. in the large intestine, E. in the stomach.
17) The development of the larval stage of Trichinella are: A. on the microvilli in the small intestine, B. in the intermuscular septa of the striated muscle, C. in the submucosal layer of the small intestine, D. in the large intestine, E. in the stomach.
18) Transmissible mechanism of transmission is typical: A. dirophylariosis, B. teniasis, C. strongylosis, D. opisthorchiasis, E. ascariasis.
19) By biohelminths include all, exept: A. difilobotrios, B. strongylosis, C. teniasis, D. teniarinhosis, E. trichinellosis.
20) Dogs are the source of contamination: A. teniasis, B. opisthorchiasis, C. strongylosis, D. toxocarosis, E. ascariasis.
21) What is the agent of pork worm disease? A. Taenia soleum, B. Hymenolepis nana, C. Taeniarhynchus saginatus, D. Trichinella spiralis, E. Dyphylobotrium latum
22) What are the principal clinical syndromes in pork worm disease? A. respiratory syndrome, B. cholestatic syndrome, C. dyspeptic syndrome, D. meningeal syndrome, E. everything is right
23) How is the source of invasion in case of cystecercosis? A. cattle, B. dog, C. pig, D. human, E. fish
24) What methods are used for diagnostics of cystecercosis? A. General blood analysis, B. biochemistry methods, C. computed tomography, D. investigation of faeces, E. nothing is right
25) The invasion of the human with Dyphillobotirum latum is possible to get after eating: A. fresh water fishes, B. sea fishes, C. pork, D. beef, E. milk.
26) What is the duration of the life of the adult forms in echinoccosis? A. It is continued from 6 months till 1 year, B. It is continued 1 week, C. It is continued 10 weeks, D. It is continued more than 10 years, E. It is continued 7 days,
27) What is epidemiology of echinoccosis? A. It is transmissive helminthiasis, B. It is heohelminthiasis, C. It is biohelminthiasis, D. It is contagious helminthiasis, E. It is especially dangerous disease
28) What is the possible localization of hydatid cyst? A. liver, B. lungs, C. brain, D. everything is right, E. nothing is right

29) What is treatment should be administered in pork tapeworm? A. vermitin (phenasalum; Niclosamidum), B. Filicis maris rhizome, C. mebendazol, D. decaris (levamysol), E. chloxil

30) How is the definitive host in case of Taeniarhynhosis? A. cattle, B. dog, C. pig, D. human, E. fish

31) What is epidemiology of hymenolepiasis? A. It is peroral helminthiasis, B. It is percutaneous helminthiasis, C. It is contagious helminthiasis, D. It is transmissive helminthiasis, E. It is especially dangerous disease

32) What is epidemiology of fascioliasis? A. It is anthroponosis, B. It is zoonosis, C. The definitive host is human and cattle, D. The intermediate hosts are the pigs, E. The intermediate hosts are mollusks,

33) What is epidemiology of fascioliasis? A. It is anthroponosis, B. It is zoonosis, C. The definitive host is human and cattle, D. The intermediate hosts are the pigs, E. The intermediate hosts are mollusks.

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