ODESSA NATIONAL MEDICAL UNIVERSITY

Department of urology and nephrology

GUIDELINES
of independent work of students

Academic discipline “Urology”

The theme of independent work of students: Parasitic Diseases In Urology

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Head. Chair prof. F.I. Kostev

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Topic: Parasitic Diseases In Urology -1 h

1. Importance of the theme:

Parasitic diseases of the organs of the urogenital system are related to rare enough diseases. Ukraine is not endemic zone for manifestations of such pathology, but urogenital schistosomiasis, filariasis are spread in the countries with a subtropical, tropical climate, in the CIS - in the countries of Asia. Therefore cases of these diseases have sporadic, delivered character. It accounts for absence of vigilance of doctors to such diseases and mistakes in diagnostics which leads to neglected cases of the disease and long disability of patients.

2. Learning objectives:

To know:

-Epidemiology of parasitic diseases of the organs of the urogenital system in the world and Ukraine.

- The basic pathogenesis of parasitic diseases of the organs of the urogenital system.

- The basic serologic tests for parasitic infections.

- Stages of performing surgical interventions which are used in parasitic diseases.

To be able:

- To interpret the data of ultrasonic examination, excretory urography.

- To establish correctly the diagnosis of parasitic invasions and administer proper treatment.

3. Interdisciplinary integration

Discipline	To know	To be able to
1. Previous disciplines Anatomy Physiology Microbiology	Anatomy of the organs of the urino-genital system Function of the organs of the urino-genital system Morphologic, Serologic properties of	Interpret data of US examination, microbiological investigation, correct sampling of the material

Roentgenology and radiology	parasites US examination of the organs of the urogenital system	
2. Following disciplines Infectious diseases Parasitology Epidemiology	Parasite species, sensitivity to chemicals. Occurrence	
3. Interdisciplinary integration		Make differential diagnosis with objective and nonspecific inflammatory diseases

4. Materials of indidividual work

4.1. List the main terms:

Термин	Определение термина
Schistostomiasis (bilharziasis)	chronic helmintosis caused by blood flukes; causative agent is a helminth *Schistosoma haematobium.*
miracidia	the intermediate forms of the helminth *Schistosoma haematobium*
cercaria	generation of the caudate fluke larvae *Schistosoma haematobium*, which can penetrate into his organism through the intact skin or mucous membranes
bilharzioma	Specific schistostomiasis ulcer or replacement of the granuloma by the fibrous tissue
Filariases	helmintoses, which cusative agents are related to nematode of the families Filarudae Cobbold
Echinococcosis	a chronic disease characterized by development of solitary or multiple cystic formations in the liver, lungs, kidneys and other organs. The causative agent is the larval stage of the gadfly Echinococcus granulosus.

5. The contents of the theme.

Schistostomiasis (bilharziasis) is a chronic helmintosis caused by blood flukes and characterized by primary affection of the urinogenital organs and gastrointestinal tract. Some kinds of blood flukes are known. The causative agent of schistostomiasis of the urinogenital organs is a helminth *Schistosoma haematobium.*

Epidemiology. Schistostomiasis of the urinary system is one of the most widespread urological diseases. It is a big group of tropical biohelminths caused by flukes, related to the class *Trematoda,* genus *Shistosoma.* Schistostomiasis is widely spread in the countries of Africa, Asia, Latin America, especially in Egypt and Iraq. Annually this disease carries away 500,000 lives; according to the WHO up to 300 million people suffer from it, and 600 million more can be infected. Schistostomiasis is a significant social and economic problem in a number of the countries as mainly people of working age fall ill; a ratio of men and women is 5:1. Infection occurs in contact with water containing larvae (cercarias) of blood fluke, in the coastal, well warmed up parts of the rivers, irrigation canals and other reservoirs with slow current and dense vegetation. In the endemic areas the level of contamination with schistostomiasis is the highest in persons of 10-20 years old living in the unfavorable social and economic and sanitary-and-hygienic conditions. Thus, the area of the disease distribution constantly extends due to the population migration, carrying out irrigational works and creation of water basins.

Etiology and pathogenesis. The causative agent of urinogenital schistostomiasis is trematodes *Schistosoma haematobium.* Males and females of the parasite differ in the size: length of the helminth male is from 4 up to 15 mm, width - 1 mm, length of the female is up to 20 mm, width – 0.25 mm. Eggs of the helminth are very small, of only 0.1 mm in diameter, of the oval form, with a thorn on one of the poles. The developmental cycle of flukes is connected with a change of hosts. Two forms are distinguished: the larval form is inside of a snail-mollusc (the intermediate host); pubertal - in the human organism (the final host). Three kinds of molluscs can be intermediate hosts: *Bulinus trancatus, Bulinus becari* and *Bulinus wrisht.* Migration of flukes occurs as follows. Being discharged from the human organism with urine, eggs of the parasite get into the water of any fresh-water reservoirs. In the water membranes of the eggs are quickly broken off, and the intermediate forms of the helminth - miracidia are released. They require the intermediate host for the further development. For *Schistosoma haematobium* such intermediate hosts are fresh-water molluscs of several kinds *(Bullinus truncatus, Bullinus forskali, Bullinus tropicalis, Physopsis Africana,* etc.). The optimum temperature of the water for their life is 20-25°C, temperature of the water below 0°C and above 50°C causes destruction of molluscs. Molluscs prefer reservoirs with a slow current (not more than 25 cm/s). A great role in distribution of molluscs belongs to presence of food: specific vegetation, monocelled seaweed as well as microorganisms, detritus. Sharp increase of the mollusc population is marked after a season of rains. Miracidium penetrates into the mollusc body where it lives from 4 to 8 weeks. During this period there is a further development of the helminth. It is established that in the intermediate host there are cycles of agamic multiplication, giving rise to generation of the caudate fluke larvae - cercaria. Every day several thousand cercarias are discharged from the infected mollusc into the water, which life expectancy of one-two days. When the person is in the water infected with helminths, cercaria can penetrate into his organism through the intact skin or mucous membranes. Penetration of cercaria is promoted by presence of five pairs of glands at the head end, discharging lytic substances. Except for microscopic wound at the site of penetration of cercaria there are no others changes on the skin observed. The head and body penetrate, and the tail is torn away forming metacircaria. Approximately in 30 min after getting into the human's organism cercaria get into capillaries of the integument, and then in the venules and larger blood vessels due to active movement and lysis of the tissues. Through the veins cercaria reach the right atrium and right ventricle of the heart and get into the pulmonary

capillaries. It takes some days during which some larvae destroy the fine blood vessels that are accompanied by hemorrhagic manifestations. In five days from the moment of penetration through the integument cercaria reach the portal vein and settle in its fine intrahepatic branches. In 3 weeks after infection of the person the larvae migrate in the mesenterial, duodenal venous plexus as well as in the venous plexus of the bladder. One of the peculiarities of flukes is that their adults parasitize not in the intestinal lumen as the majority of helminths but mainly in veins of the bladder or intestines. By the 10-12-th week larvae of the parasite reach maturity and females start to lay eggs in the vascular bed. The spasm of the vessel promotes penetration of an egg through the vascular wall and to the exit into the surrounding tissues. More often eggs of the flukes are localized in the submucous layer of the bladder, in rare cases they can be found in the mucous membrane of the bladder as well as in the muscular layer. Due to lytic enzymes released by the germ being in the egg -miracidium, eggs of the flukes are able to "bore" the bladder mucous membrane to get in its lumen. Then the eggs get into the environment with a current of urine, and the cycle of development is repeated. Thus, in the migration cycle of the flukes it is possible to distinguish three periods: 1) prehepatic - from the moment of penetration into the vascular bed before getting into the portal vein of the liver; 2) hepatic - development of the parasite in the system of the portal vein where it reaches maturity and distinction in sex; 3) posthepatic – the female settles in the genicoform canal of the male, and the male carries the female on itself, moving against the bloodstream, going to the venous plexus of the small pelvis, in men – to the vesical-prostatic plexus. Migration occurs until the width of the vessel lumen allows it. Then the female leaves the partner and lays eggs. In laying the eggs of the parasite in the submucous layer of the bladder or in the prevesical part of the ureter the reaction to the foregn body is observed characterizing by accumulation of eosinophils, huge cells, histiocytes. The mucous membrane of these organs is hyperemic and edematous. Later on, the formation of specific schistostomiasis granuloma - bilharzioma is observed at these sites. Specific schistostomiasis ulcer or replacement of the granuloma by the fibrous tissue may subsequently occur at the place of this infiltrate. Massive character of this process leads to "shrinkage"of the bladder. Besides, the dead eggs can calsify.

Semiology and clinical course. Clinical symptoms of schistostomiasis of the urinogenital organs are divided into the general and local ones. *The general symptoms* are associated with toxic and allergic reactions to products of vital functions and disintegration of the parasite or its eggs. *Local symptoms* can be conditionally divided into the cutaneous and uric ones. The first symptoms are assocated with penetration of the helminth larvae through the skin, the second ones - with traumatization of the bladder walls by fluke eggs. By the clinical course there are distinguished acute and chronic stages of schistostomiasis. The acute stage lasts about 2 weeks. It is characterized by manifestations of dermatitis at the site of penetration of cercaria lasting up to 5-6 days, fever and general malaise are characteristic of it. Depending on sensitization of an organism expressiveness of the skin manifestations can be from insignificant itching up to serious enough toxic and allergic affection. At the moment of penetration of cercaria through the skin the person feels pain, as injection of a needle. Cough with discharge of dense sputum, sometimes hemoptysis can be observed during migration of parasites due to passage of the helminth through the pulmonary vessels. There are also symptoms of intoxication - headache and pains in the extremities, hyperhidrosis. Leucocytosis and eosinophilia are determined at this time. Sometimes the liver and spleen are enlarged. Expressiveness of the general symptoms depends on individual sensitivity of the patient and massiveness of invasion by parasites. Signs of intoxication are intensified on the average in 10-12 weeks after infection, i.e. during laying eggs by the flukes. Patients complain of weakness, fast fatigue, indisposition, pains in the lower abdomen, more often in the region of the right subcostal area, headache and morbidity in the muscles, stable elevation in the body temperature. From the moment of fixing eggs in the bladder wall schistostomiasis tubercles start to appear around the eggs, and microabscesses with the subsequent fibrous changes of the affected tissue develop. During this period the most frequent symptom is *terminal hematuria* - discharge of blood at the end of urination. In massive

affection of the mucous membrane of the bladder total hematuria can be observed. In development of cystitis symptoms frequent and painful urination is also characteristic. Some patients have sharp pain in the urethra during or at the end of urination. In long presence of the parasite in the person's organism schistostomiasis gets a chronic course when the period from the moment of laying eggs till their appearance in patients' urine can take some months. There are distinguished mild, of moderate severity, severe and very severe forms of the disease course. In the mild form patients have no complaints, urination disorders are insignificant, working capacity is kept; in the course of moderate severity disuria is expressed distinctly, the liver and spleen are enlarged, anemia develops; the severe form is characterized by frequent aggravations of chronic cystitis, lasting for years, expressed disuria. Not infrequently complications develop including cirrhosis of the liver, strictures of the ureter, hydronephrosis, and microcystis. Patients lose work ability. This form is insusceptible to treatment and can result in a lethal outcome. The clinical course of the disease depends on the degree of distribution of the specific process in the organs of the urinary system and character of proliferative and regenerative processes in the tissues leading to development of complications of the disease (stones of the kidneys and bladder, hydronephrosis, chronic renal failure, cancer of the bladder).

Complications of schistostomiasis of the urinogenital organs can be conditionally divided into early and late. Secondary infectious-inflammatory affections are related to early complications of the urinary organs and genitals - cystitis, pyelonephritis, epidimiitis, and prostatitis. Late complications develop against the background of long persistence of the causative agent in an organism. They include tumours of the bladder and ureter, stricture of the ureter, hydronephrosis.

Diagnosis is based on the data of the epidemiological anamnesis, clinical manifestations and results of the laboratory and instrumental studies. In establishing the diagnosis of schistostomiasis of great significance is the fact of staying of the patient in the endemic focus of the disease. While analysing clinical symptoms a combination of the general manifestations, skin rash with disuria and hematuria should cause doctor's vigilance. Of crucial importance in diagnosis of schistostomiasis is microscopy of urine. An absolute sign of the disease is detection of fluke eggs in urine in ovoscopy. It is known that fluke eggs are released with urine most intensively about noon; however, all daily portion of urine is usually investigated for their detection. If it is impossible, urine is taken from 10 a.m. up to 2 p.m. The collected urine is infused in high jars, supernatant fluid is poured out, and sediment is centifugated. Microscopy of the sediment is made in a slightly blacked field of vision. Due to non-uniformity of the eggs discharge with urine, repeated analyses are necessary. The analysis of urine is likely to reveal hematuria, proteinuria, and leucocyturia. Cystoscopy is necessarily carried out on suspicion of schistostomiasis. Focal hyperemia of the mucous membrane with the smudged vascular pattern can be considered as the earliest manifestation of schistostomiasis affections of the bladder. However, this sign is rather nonspecific. Detection of schistostomiasis tubercles is more informative - a few formations of yellow color towering above the mucous membrane of a pin head in size. A characteristic sign of schistostomiasis tubercles is absence of hyperemia zone around them and their regular arrangement in the bladder. In the chronic course of the disease the mucous membrane is pale due to bloodstream disorder in the bladder wall.

A pathognomic sign of schistostomiasis at this stage is presence of "sandy spots" on the mucous membrane of the bladder, representing dead calcified flukes eggs, being in the submucous layer and visible through thinned, not enough vascularized mucous membrane of the bladder. "Sandy spots" specify duration of schistostomiasis invasion.

On cystoscopy it is also possible to reveal polypoid growths of the mucous membrane of the bladder, submucous haemorrhages, erosion and ulcers. Polypoid growths resemble a papilloma externally and frequently are a source of hematuria. The cause of bleeding can also be schistostomiasis ulcers. They have characteristic external signs – crater-like edges and irregular shape.

Sometimes on suspicion of schistostomiasis invasion biopsy of the site of pathologically changed mucous membrane of the bladder is made during cystoscopy. The tissue taken for biopsy is crushed in a drop of glycerin between subject glasses and investigated under a microscope. On suspicion of schistostomiasis it is necessary to carry out radiological examination to all patients - plain and excretory urography. It is possible to reveal calcified organs of the urinogenital system on the plain roentgenogram. It is known that the dead fluke eggs become calcified. It allows to see contours of the foci of calcification in the wall of the bladder or ureter, seminal vesicles in the form of "honeycomb" as well as secondary stone formation in the kidneys and ureters due to strictures of the distal parts of the ureter of schistostomiasis genesis. Excretory urography gives the information on patency of the ureter and presence of the hydronephrotic transformation. The most typical schistostomiasis localizations of strictures of the ureter are distal and intramural parts. The descending cystography allows estimating the size of the bladder that is of great importance in diagnosis of microcystis. Intracutaneous allergic tests with schistostomiasis antigene are used on inspections in the endemic foci and epidemiological investigations in the risk groups. Specificity of serologic methods of investigation is insufficiently high but sometimes there are made reactions of complement binding, indirect hemagglutination, immunoenzymic analysis with the blood serum of the patient.

Differential diagnostics. Presence of tubercles in the bladder is also characteristic of tuberculosis of the bladder. Differential signs of schistostomiasis etiology are absence of regularity in the arrangement and hyperemia nimbus around these tubercles.

It is necessary to differentiate polypoid growths of the mucous membrane characteristic of schistostomiasis affections from a tumour of the bladder. Frequently the final diagnosis is established only after endovesical biopsy. In revealing sites of calsification on roentgenograms it is necessary to make a differential diagnosis of urolithic disease and secondary stone formation in the bladder.

Treatment. The modern medicine has highly effective medical products for treatment of schistostomiasis. It is necessary to note that medicamentous therapy is successful in uncomplicated schistostomiasis. In development of complications it is frequently necessary to use surgical methods. Prasiquantel (biltricid) is highly effective in all schistostomiases. The preparation is administered perorally in the dose of 20-60 mg/kg by 1-3 intakes in short courses. Other frequently used preparation is niridazol (ambilgar) administered perorally to the adult in a daily dose of 25 mg/kg during 5-7 days; the dose is divided into morning and evening intake. Metrifonat is a preparation of reserve in schistostomiasis invasion; it is administered unitary in the dose of 7.5-10 mg/kg. Other preparation of reserve is hicanton (etrenol) adminitered unitary intramuscularly in the dose of 2-3 mg/kg. Last years stilbocaptate (astiban) began to be applied by 8-10 mg/kg, intramuscularly, weekly; the course of treatment is 5 weeks. Surgical treatment is performed in development of complications, more often in stenoses of the ureter. Efficacy of the therapy is estimated on the basis of long (several months) and careful clinical and helminthic studies as relapses of the disease are possible. Serologic reactions are used to control efficacy of specific therapy of schistostomiasis. They become negative in 3 months after disappearance of helminthic invsions. In complications of the basic disease (stenoses of the ureter, stones of the kidneys and bladder, etc) operative treatment is performed, which should be preceded by the course of medicamentous therapy.

The prognosis is favorable under the condition of duly administration of specific therapy.

Filariasis. Filariases are helmintoses, which cusative agents are related to nematode of the families Filarudae Cobbold.

For filaria a human is the final host. Adult helminths parasitize in the lymphatic vessels, connecting tissue of the subcutaneous cellular tissue and walls of the body cavities; larvae (mictofilaria) circulate in the blood or concentrate in the superficial layers of the skin. Intermediate hosts (carriers of filaria) are various blood-sucking insects (mosquitoes, gadflies,

midges, black gnats.). The main filariases of the human are Wuchereriasis, Brug's filariasis. Wuchereriasis and Brug's filariasis are helminthiases with the chronic course. They are characterized by fever, lymphadenitis, and retrograde lymphangitis of the extremities, orchitis, funiculitis and abscesses basically of the allergic nature in the initial stage as well as development of elephantiasis of the extremities and mammary glands, chyluria or hydrocele.

Etiology. The causative agents of Wuchereriasis are Wuchereria bancrofti, of Brug's filariasis - Brugia inalayi – helminths-nematodes, having the elongated threadlike form with thinning at the ends. Development of filaria occurs in a change of hosts, final host of Brugiainalayi is a human and some kinds of monkeys, intermediate hosts are various kinds of mosquitoes. Mature filaria parasitize in the lymph nodes and vessels.

Helminth females bear larvae – microfilaria, which do not vary morphologically and do not grow in the human organism. They parasitize in the blood system. Microfilariae of Wuchereria bancrofti of the periodic strain (Microfilaria nocturna) are in the vessels of the lungs in the daytime, and move ahead in the peripheral vessels at night. Microfilariae of Wuchereria bancrofti of the subperiodical strain, revealed in the zone of the Pacific ocean and consequently received the name W. pacifica, are in the peripheral blood all the day round but their number noticeably increases in the afternoon. The periodical strain of Brugia inalayi is inherent only to the human while the subperiodical one is also encountered in monkeys. Both strains of Brugia inalayi are characterized by night peak of microfilariemia, which is encountered much less often in the periodical strain.

Adult filaria are capable of parasitizing in the human organism for a long time (up to 12, and according to some information to 17 years), microfilariae – for about 12 months.

Epidemiology. A source of Wuchereriasis is a sick person or parasite-carrier, a source of Brug's filariasis is a person and some monkeys. Direct carriers of infection are mosquitoes. During a sting of a mosquito invasion forms of microfilaria get into the skin, actively penetrate into the bloodstream and are brought in the tissue by a flow of the blood. Transformation of microfilariae into mature forms occurs later in 3-18 months after their penetration into the person's organism.

Wuchereriasis is endemic for some the countries of Africa, Asia, it is encountered in India, China, Japan, Central and South America, on the islands of the Pacific and Indian oceans.

Brug's filariasis is widespread in the countries of Asia - in India, Ceylon, in Thailand, Vietnam, Laos, Cambodia, China, Japan, Indonesia, and Malaysia.

Pathogenesis. Pathogenesis of Wuchereriasis and Brug's filariasis is based on toxic-allergic reactions, mechanical influence of helminths on the lymphatic system and secondary bacterial infection. As many other helminthiases, Wuchereriasis and Brug's filariasis can not give the expressed clinical picture in some cases. In general sometimes there are no clinical manifestations of invasion. Asymptomatic Wuchereriasis or Brug's filariases are observed when parasites do not occlude the lymphatic vessels and do not cause inflammatory changes in the surrounding tissues. Patients with such forms of infection are revealed casually in detection of microfilariae in their peripheral blood. Wuchereria and Brugia in the lymphatic vessels including those in the chest duct are weaved among themselves into glomuses, which cause delay in the lymph flow and lymphostasis. Parasites cause inflammatory induration of the lymphatic vessel walls that finally leads to their occlusion as a result of stenosis or thrombosis. Thrombosed lymphatic vessels often rupture. Because of long lymphangitis and lymphadenitis in various parts of the body elephantiasis can develop. The changed endothelium of the lymphatic vessels, the foci of necroses in the lymph nodes and surrounding tissues are favorable places for development of coccal infections with formation of abscesses. As a result of vital activity of parasites and especially during their disintegration, substances, which lead to sensitization of the organism with local and general allergic reactions – eosinophilia, skin eruptions, etc are formed.

Symptoms and course. Allergic manifestations can develop approximately in 3 months after infecting. Microfilariae are revealed in the blood not earlier than in 9 months.

1 stage. The disease begins with various allergic manifestations. Painful elements like exudative erythema appear on the skin especially on hands, lymph nodes are enlarged in the inguinal

areas, on the neck and in the armpits, painful lmphangites, funiculitis, orichoepididimitis, synovitis with the outcome in fibrous ankylosis, in women - mastitis often develop. Hydrocele develops in the prolonged recurrent course of funiculitis and orchoepididimitis. The fever is characteristic, bronchial asthma and bronchopneumonia develop quite often.

In 2-7 years after infection II stage of the disease ensues, which is characterized basically by affections of the skin and deep lymphatic vessels with development of varicosis, lymph flow disorder, ruptures of these vessels. Painful lymphangites with regional lymphadenitis develop. At this time the patient has the expressed phenomena of intoxication against the background of high body temperature and severe headaches within several days. Vomiting is often observed, sometimes a delirious condition develops. The attack usually comes to an end with profuse sweating. As a result of rupture of the lymphatic vessels there is lymphorrhea and reduction in lymphadenitis intensity.

Phases of relative well-being are periodically replaced by the aggravations of the disease. Dense bands remain at the sites of lymphagites; there is also fibrous induration in the affected lymph nodes. The enlargement of the inguinal and femoral lymph nodes is characteristic. Initial swelling of the lymph nodes does not cause pain, however, in subsequent development of lymphangites there are severe pains in the nodes. The affection can be uni-or bilateral, the node size is from small up to 5-7 cm in diameter. The so-called lymphoscrotum is frequently develops (chylous impregnation of the tunica vaginalis) and chyluria. Lymphoscrotum is clinically manifested by the enlargement of the scrotum. The dilated lymphatic vessels are easily determined on palpation of the scrotum skin. In ruptures of these vessels a lot of quickly coagulating lymph outflows. The outflow of the lymph from the damaged vessels may last for some hours. Chyluria or lymphuria is often encountered in the patients with wuchereriasis or Brug's filariasis in the countries of Northern Africa, India and China. The patient notices that urine has got a milky-white shade. In some cases urine becomes pink or even red, sometimes it is white in the morning and red in the evening or vice versa. Presence of the blood in urine alongside with lymph is explained, obviously, by ruptures of the fine blood and dilated lymphatic vessels. Microfilariae are discharged in urine only at night. Sometimes it is preceded by slight pain above the pubis or in the inguinal areas. The characteristic feature is delay of urine due to coagulation of lymph and formation of flakes in the urinary tracts. In lymphuria there is admixture of lymph in urine, protein in a significant amount, admixture of blood is possible but there are no traces of fat. Lymphocytes are found in the urine sediment.

Usually bodies of the dead filariae are completely resolved or calcified. However, in some cases the dead parasites are the cause of development of abscesses, which lead to severe complications, such as empyema, peritonitis, purulent inflammation of the genitals.

Due to damage of the lymphatic vessel walls in wuchereriasis, the microbes can get into the surrounding tissues and in the blood that may result in development of sepsis. Hemolytic streptococcus is often found in the blood of such patients.

The obstructive stage of the disease is characterized by elephantiasis. In 95% of cases elephantiasis of the lower extremities develops, a little bit less often - the upper extremities, genitals, separate parts of the trunk and in very rare cases – of the face. Clinically, elephantiasis is manifested by quickly progressing lymphangitis with addition of dermatitis, cellulitis in a combination with fever, which can serve the basic symptom of the disease in some cases and is a consequence of addition of a bacterial infection. In due course the skin becomes covered with warty and papilomatous growths, there are sites of eczema-like changes of the skin, unhealing ulcers. The legs can reach the huge size, they get a kind of shapeless blocks with thick transversal folds of the affected skin. The weight of the scrotum usually makes 4-9 kg, and in some cases - up to 20 kg. There was described a case when the weight of the scrotum of the patient has reached 102 kg. In case of elephantiasis of the face the upper eyelid is more often affected. In Brug's filariasis elephantiasis occurs usually only on the extremities, the affection is more often unilateral, the skin remains smooth.

Treatment. Specific treatment is given by Ditrazin (synonyms: Carbamazin, Loxuran, Hetrazan, Bancid, Notezin). The preparation is especially active to microfilaria but works in adult Wuchereria and Brugia as well as it is likely to kill or sterilizes females.

Mode of administration of the preparation: 1 day - 50 mg inside after meal once a day, 2 day - 50 mg 3 times a day, 3 day - 100 mg 3 times a day, 4-21 day - 2 mg/kg 3 times a day. To reduce irritation of the gastroentestinal tract (nausea, vomiting, and loss of appetite) the preparation is necessary to take after meal. Of other side-effects there may be headache, sleeplessness, allergic reactions in the form of fever, skin eruptions, etc. Allergic reactions arise due to sensitization of an organism by products of disintegration of parasites and are stopped by corticosteroid preparations and others desensitazing drugs.

After treatments with ditrazin microfilaria usually appear in the blood again in some months, therefore courses of treatment are repeated by clinical indications.

Pathogenetic therapy is given by antiallergic preparations (corticosteroids, etc.), which reduce the inflammatory process in the lymphatic vessels and thus the outflow of the lymph. In elephantiasis elastic bandage is used for reduction of edema of the affected organs; at the late stages of elephantiasis it is necessary to have surgical intervention. In elephantoid fever and other signs of secondary infection antibiotics are indicated.

Prognosis. The disease is characterized by a long course if patients with Wuchereriasis and Brug's filariasis have not passed a course of treatment. Elephantiasis leads to stable disability and physical inability. Lethal outcomes come in addition of secondary infection, especially in development of empyema, peritonitis and abscesses near to the vital organs.

Echinococcosis of the kidneys.

Echinococcosis is a chronic disease characterized by development of solitary or multiple cystic formations in the liver, less often - lungs, kidneys and other organs.

Etiology. The causative agent is the larval stage of the gadfly Echinococcus granulosus. The mature form of the helminth - cestodes - is 2-7 mm in length. The larval stage growing, developing and living in the person's organism for years, is presented by a cyst of the round or oval form filled with fluid. Mature forms parasitize in the small intestines of a dog, a cat. The intermediate hosts of ecchinococcus are a sheep, a horse, a pig, a human.

Epidemiology. The disease is widespread in the countries with the developed pasturable cattle breeding. On the territory of the CIS countries it is more often registered in Moldova, republics of Transcaucasia and Central Asia, in Kirghizia, Ukraine, and Belarus. The basic source of invasion is domestic dogs, less often - wolves, jackals. Mature eggs are discharged with excrements of animals, polluting their wool and environment. Infection of the person occurs in contact with the affected animals, in picking up berries and herbs, drink of water from the sources polluted with helminth eggs. Due to peculiarities of epidemiology the disease is more frequently encountered in certain professional groups (workers of slaughterhouses, shepherds, tanners). The possibility of transplacental transmission of helminthiasis is also proved.

Pathogenesis. In the gastroentestinal tract of the person the oncospheres of echinococcus leave the membrane and discharged larvae penetrate into the mesenterial blood vessels and spread by the bloodstream. Most part of the larvae are left in the liver, some part gets into the lungs (through pulmonary circulation). The insignificant part passes the filter of the lungs and brain and gets into the kidneys, bones. A fibrous capsule is formed around the cyst in the liver by the end of the 5-th month. Echinococcal vesicle has a complex structure. The external (hyaline) membrane consists of numerous concentric plates not containing cells that are important for diagnosis. It is lined with a germinal layer from within, which gives rise to the corpuscles of the vesicle (protoscolexes and nidifugous capsules). Not infrequntly secondary (filial) and tertiary (grand) vesicles are quite often formed inside the primary (maternal) vesicle. One cyst (solitary affection) or several (multiple Echinococcosis) can develop in the affected organ, the sizes of the cysts vary considerably - from 1-5 up to 40 cm and more in diameter. The echinococcal cyst

grows expansively, moving aside and compressing tissues of the host, which get atrophied and necrotized.

Parasitic antigenes exert a sensitizing effect especially expressed in multiple echinococcoses. The immune system of the host is not able to destroy helminth completely, it is associated with presence of some adaptive mechanisms in echinococcus.

Symptoms and course. Echinococcosis is more often revealed in persons of the middle age. In uncomplicated cases the diseases lasts for years and can be revealed casually (on scheduled photoroentgenography) or on purposeful inspection (in the foci) in absence of clinical manifestations (preclinical stage of echinococcosis). In clinically expressed stage the course of echinococcosis depends on localization of cysts, their size, developmental rate, complications, variants of combined affection of the organs, reactivity of the host's organism. Pregnancy, severe intercurrent diseases, alimentary disorders promote more severe course of the disease, fast growth of the cysts, disposition to ruptures and dissemination of the causative agent.

In localization of the cyst in the right lobe of the liver, the painful syndrome is similar to those in cholecystitis. There is marked loss of weight, loss of appetite, in localization in the left lobe there is heartburn, eructation, vomiting. In superficial localization of the cyst, it can be palpated. In the neglected cases there is protein- synthetic dysfunction of the liver – dysproteinemia with decrease in albumins, prothrombin and increase of gamma-globulins. Manifestations of echinococcosis of the lungs are determined by localization of the cyst. Even a small cyst, located near to the pleura, manifests itself by a painful syndrome, and in localization at the bronchial trunk clinical symptoms are manifested by cough and vascular disorders. Echinococcosis of the kidneys is quite often diagnosed only in revealing echinococcuria. A nagging pain in the lumbar area, disuric disorders may precede detection of scolex fragments in the urine sediment. Much less often there may be found echinococcosis of the brain, mediastinum, mammary gland, intestines; it is extremely rare to find echinococcosis of the bones, hypodermic celular tissue.

Complications in echinococcosis are encountered often (up to 30 %), sometimes being the first clinical manifestation of the disease. Suppurations of the cyst (addition of the secondary bacterial flora are frequent in destruction of echinococcus), as well as intensification of pains, fever, hyperleucocytosis. There may be cholangites, opening of the cyst in the abdomen and pleural cavity with development of peritonitis, pleurisy. Compression of the bilious ducts leads to mechanical jaundice, less often – biliary cirrhosis, amyloidosis. In compression of the vessels of the portal system there are signs of portal hypertension. Echinococcosis of the lungs may become complicated by repeated pulmonary bleedings, acute cardiovascular insufficiency. The most threatening complication is rupture of the cyst, which can be provoked by a blow, weight lifting, rough palpation. Rupture of the cyst is accompanied by a sharp painful syndrome and manifestations of the allergic reaction of a various degree of expressiveness, up to development of anaphylactic shock.

Diagnosis and differential diagnosis. Diagnosis of echinococcosis in the initial stage is complicated because of obliteration and nonspecificity of the clinical manifestations and is based on the analysis of data of the clinical, radioisotope, radiation and immunologic investigations. Presence of operations for echinococcosis, echinococcosis in other members of the family in the anamnesis allows to assume probable etiologic diagnosis. Radiation (radiological), radio isotope (scanning, scintiography) methods of examination, ultrasonic and especially computer tomography and techniques with use of a magnetic-nuclear resonance allow to estimate prevalence of the process. In some cases the diagnostic laparoscopy (caution – a cyst cannot be punctated because of danger of dissemination) is indicated.

It is more often necessary to differentiate echinococcosis with neoplasms of the corresponding organs. The main role belongs to the immunologic methods. Reactions of indirect hemagglutination (RIHA), latexagglutionation, double diffusion in the gel, immunoelectrophoresis (IEP) and counterimmunoelectrophoresis (CIEP), fluorescing antibodies (RFA), immunoenzymic method (IEM) are widely used; their information value reaches 90-97

%. Intracutaneous test with echinococcal antigene (Casonni reaction) is inexpedient due to frequent development of severe allergic reactions, especially in repeated investigations.

Treatment. In uncomplicated echinococcosis of the liver medicamentous treatment is possible: albendasole (a synonym – zentel) in the dose of 10 mg / (kg/ day); the dose is divided into two intakes, treatment lasts 3 months. In relapse or widespread of the process operative treatment is indicated. A single cyst can be removed or drained under the control of ultrasonic investigation with introduction of 95% ethyl alcohol with mebendasole. All patients are registered in the dyspensary. They have examination after surgery 1-2 times a year, which includes the blood count, determination of bilirubin, ALT, ACT in the blood serum, estimation of proteinogram, serologic (IFA or RIHA) and ultrasonic investigations (computer tomography). In absence of signs of relapse and stable negative serologic reactions within 5 years the patients can be struck off the register.

*The prognosis i*s serious due to possibility of development of complications menacing to life.

Malaria. Malaria is a protozoan transmissive disease with alternation of feverish attacks and the periods of apyrexia, enlargement of the liver and spleen, anemia with possible development of hemolytic jaundice. The reservoir and source of invasion are the human (the patient or parasite-carrier) and females of mosquitoes of the genus *Anopheles.* The mosquito becomes infected after sucking the blood of the person containing mature gametocytes. The latter overflow the blood after 2-10 attacks of three-day or four-day malaria, and in tropical malaria – from the 7-10th day of the disease. Duration of this period makes about one year in tropical malaria, it is a little bit more in three-day and oval-malaria, tens years - in four-day malaria. In the endemic areas the basic sources of infection are children. In adults the amount of circulating gametocytes and duration of carriage is much less as a result of developing immune responses. *The mechanism of transmission* is transmissive. The possibility of infection is not excluded in blood transfusions or application of the infected instruments (syringes, needles). There may be transplacental or intranatal transmission of the causative agent. *Natural susceptibility of people* is general; however, there are groups, relatively unsusceptible to malaria.

The basic epidemiological signs. Now the malaria is widespread on the territories of 90 countries of the globe, half of which are located in Africa. On the territory of the former Soviet Union the active foci exist in Tajikistan, Uzbekistan and Azerbaijan. In the majority of the European countries (including Russia) there are marked brought in cases of malaria.

Clinical picture. Some periods consistently replacing each other are distinguished in the course of the disease. *The incubation period.* It lasts 1-3 weeks, in four-day malaria - up to 6 weeks. In cases of three-day or oval-malaria the inactive condition of bradysporosoites in the liver can lead to lengthening of the incubation period up to 2 years and more. *The prodromal period.* The majority of patients, invaded by the causative agents of three-day and oval-malaria, develop weakness, reduction in working ability, headache, arthralgia and myalgia. Pains in the liver and spleen are sometimes possible. The prodromal period lasts from several hours to 2-3 days. Development of the prodromal period is uncharacteristic of tropical and four-day malaria. *The period of initial fever.* It is characteristic of primary infection. It is manifested by general malaise, progressing weakness, headache, myalgia, arthralgia, pain in the lumbar area. Objective data on examination of the patient are scarce: there may be moderately expressed catarrhal phenomena in the fauces and tachycardia. Enlargement of the liver and spleen, their induration and morbidity to palpation are revealed only in the end of this period. Nonspecificity and moderate expressiveness of the clinical signs of the disease create difficulties in determination of the cause of a feverish condition. However, in taking the detailed hourly anamnesis of the diseases it can be noted that elevation in the body temperature within several hours is accompanied by fever of a various degree of expressiveness, and in reaching the maximum level of fever (it is usually at night) the fever is replaced by a feeling of heat, at the same time there may be dryness in the mouth and thirst. By the morning the body temperature subsides, though does not reach normal indices that is accompanied by sweating, sometimes significant. The state

of health of the patient is often noticeably better on the 2nd day of the disease than on the first day. Duration of the period of initial fever makes 3-5 days.
The clinical picture of the feverish attack has much in common in all forms of malaria and passes three stages: fever, heat and sweating.
Stage of fever. Headache, dryness in the mouth, quite frequent muscular pains, pains in the lumbar area as well as in the liver and spleen area are characteristic. The body temperature with fever of a various degree of expressiveness quickly increases. The tachycardia develops. The skin becomes pale, dry; there are marked cyanosis of the lips, nose and tips of the fingers. Duration of the stage is from 1 up to 3 hours.

Stage of heat. Increase of intensity of the above-stated complaints and significant deterioration of the state of health of the patient are characteristic. The body temperature is at a level 39-40 °C and higher, the fever is replaced by a feeling of heat, dizziness and vomiting. There may be disorder of consciousness, delirium, hallucinations, and spasms. The skin of the patient is dry and hot, extremities are often cold. The face is hyperemic, scleras are injected, there may be herpetic eruption on the lips.There are dyspnoe, distinct tachycardia, dull sounds of the heart, arterial hypotension. Diuresis is lowered. In tropical malaria alongside with these symptoms there may be exanthemas, bronchospasms, pains in the abdomen, diarrhea. The stage of heat lasts from 1 up to 12 hr.

Stage of sweating. The body temperature critically decreases to normal indices; its subsiding is accompanied by sweating of a various degree of expressiveness. The state of health of the patient improves, pains disappear. After the attack there is expressed weakness and arterial hypotension. The general duration of malarial paroxysm makes 6-12 hr; in tropical malaria it can be prolonged for about one day and longer.

Typical malarial paroxysms are divided by intervals of the normal body temperature, attacks repeat in a day, in four-day malaria - in 2 days. In the normal body temperature the state of health of patients improves, but asthenization is kept.
The following clinical forms of the disease are distinguished:

Tropical malaria
Three-day malaria
Oval-malaria
Four-day malaria

Complications. Most typical are: cerebral malaria with development of coma, hemoglobin fever, acute renal failure, hemorrhagic syndrome, less often collapse, edema of the lungs, malarial psychoses, etc.

7. A chart of individual students' work

The main tasks	Instructions	Answers
To learn:		
Etiology	To name the main etiologic factors of echinococcosis	A representative of tapeworm (cystoid) –echinococcus
	schistosomiasis	Trematoda (blood schistosome)
	filariasis	Subgroup of filaria

Clinical course	Echinococcosis	Pain in the lumbar area, disuria
	schistosomiasis	Pain in the perineum, disuria, terminal hematuria, hypochromic anemia, allergic manifestations at the site of invasion as papullae, pustules, urticarial rash, fever
	Filariasis	Chiluria, signs of lymphostasis, disuria, in males: hydro- and hematocele, epididimitis, orchitis, funiculitis
Diagnosis		Tests of Buaden and Larosh (Cacconi)
	Specific methods Echinococcosis	Ovoscopy
	schistosomiasis	Miscroscopy of blood and urine (microfilaria)
	filariasis	
	X-ray methods: echinococcosis	Signs of calcinosis sickle-shaped calcifications on the review roentgenogram, grapes clusters and presence of fluid in the cyst cavity – on

		excretory urograms
		Review roentgenogram – signs of calcinosis (linear and ring-shaped calcifications); excretory urogarm – stenoses of the ureters, hydrouretero-nephrosis
		Excretory urogram – signd of necropapillitis
	schistosomiasis	
	Filariasis	Vegetative blisters from the ureter ostium on the side of affection
	<u>Cystoscopy:</u>	
	In echinococcosis	Schistosome tubercles, polupoid growths, “sand” granuloma
	In schistosomiasis	
		Echinococectomy, nephrectomy, resection of the bladder
Treatment	<u>Surgical:</u>	After assessment of results of conservative therapy
	In echinococcosis	Albendazol
	In schistosomiasis	

	<u>Conservative:</u> echinococcosis schistosomiasis Filariasis	Metrifonate (clibarzil) 7.5-10.0 mg/kg a day per os for three days or or prasikvantel (biltracid) 25-35 mg/kg – per os for 5 days . Invermectin

8. Materials for self-control.

8.1. Questions for self-control.

1. What basic parasitic diseases occur in urology?

2. What basic endemic zones of parasitic invasions?

3. Name the basic routes of penetration of parasites into a person's organism.

4. Name the basic clinical symptoms of echinococcosis.

5. Symptoms of bladder echinococcosis.

6. Clinical course and diagnosis of schistosomiasis (bilharziasis).

7. Clinical course, diagnosis and treatment of filariasis.

8.2. Tests for self-control.

Choose the basic symptom of urogenital filariasis:

-hematuria

-pyuria

-oxalaturia

-chiluria

-isostenuria

-cylindruria

The standard answer: chiluria

9. Themes of the students' scientific work on the given theme:

1. Clinical course, diagnosis and treatment of renal echinococcosis.

2. Clinical course, diagnosis and treatment of schistosomiasis of the urinary tract.

3. Clinical course, diagnosis and treatment of three-day and four-day malaria.

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