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Current State of the Problem of Liver Faciolessis

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Abstract
According to various estimates, from 2 to 17 million people suffer from fascioliasis in the world, up to 180 million people are at risk of infection. Fascioliasis is classified as a new or re-emerging disease that is common in different latitudes of the world. For these reasons, WHO classifies fascioliasis as an important human disease that deserves international attention. The development cycle of the pathogen and the geographic distribution of the disease are described. The pathogenesis of fascioliasis and the clinical picture of the disease are presented. Diagnosis and differential diagnosis of fascioliasis, treatment are described.

Keywords: Parasitic diseases, fasciola hepatica, liver fascioliasis, helminthiases, fascioliasis.

Introduction

Fascioliasis, a parasitic disease caused by the trematode Fasciola hepatica, has a significant pathological impact on human health. According to various estimates, from 2 to 17 million people suffer from fascioliasis in the world, up to 180 million people are at risk of infection. Fascioliasis is classified as a new or re-emerging disease that is common in different latitudes of the world. For these reasons, WHO classifies fascioliasis as an important human disease that deserves international attention [1-6].

The causative agents of liver fascioliasis are Fasciola hepatica - a liver fluke measuring 20-30 x 8-12 mm and Fasciola gigantica - a giant fluke measuring 25-76 mm x 5-12 mm, which belong to the genus Fasciola, family Fasciolidae. F. hepatica has a leaf-shaped body with a cone-shaped head end. F. gigantica has a longer, tapered body. The eggs are yellowish-brown, measuring 105-150 x 63-90 µm in F. hepatica and 150-190 x 75-90 µm in F. gigantica [1, 2, 11]. Sexually mature fasciolae usually live in the large bile ducts and gall bladder of herbivorous mammals, where they lay eggs that are released into the environment with feces. In freshwater bodies of water, the eggs hatch into larvae - miracidia - which penetrate freshwater mollusks, where the development and reproduction of the parasite occurs. Cercariae emerge from the mollusk and attach to plants, various objects, or water film, turning into adolescaria. The definitive host swallows adolescaria, the larvae excyst in the intestine, pass through the intestinal wall, abdominal cavity, capsule and liver parenchyma into the biliary tract. Some parasites migrate to the liver and bile ducts hematogenously through the portal vein system. Sexually mature fascioli develop 3-4 months after

infection, after which fertilization occurs and egg laying begins. The lifespan of fasciolae is about 5 years, some individuals live up to 10 years or more [3-6, 11].

The definitive host of F. hepatica is mainly sheep, F. gigantica - cattle, less often - other domestic and wild herbivores (goats, horses, donkeys, deer, camels, llamas, rodents, lagomorphs), as well as pigs, monkeys, etc. Various freshwater mollusks serve as intermediate hosts [16].

A person usually acts as an accidental host. The disease is more often recorded in areas with widespread grazing livestock. F. hepatica is found in many temperate countries in Europe, Asia, America, Africa, Australia and Oceania. F. gigantica is found mainly in the hot climates of Africa, the Middle East, Transcaucasia, Central, South and Southeast Asia, where the ranges of the two species often overlap, and hybrid forms of parasites are identified. F. gigantica is believed to infect humans relatively rarely. A person becomes infected by eating aquatic plants and plants growing in water meadows, as well as vegetables and herbs that were irrigated with contaminated water, by drinking (including by swallowing while swimming) and by using water from contaminated reservoirs for household needs. Fasciola infestation in regions with warm climates occurs year-round; in other temperate countries, usually in summer and autumn, and clinical symptoms appear in winter [12-15].

Familial cases of fascioliasis are often reported [2, 11]. Fasciola larvae, once in the small intestine, excyst within the next hour. Within two hours after infection, fasciolae penetrate the intestinal wall and over the next 72 hours are found in the abdominal cavity on the way to the liver. Penetration through the capsule (usually the left lobe) of the liver takes 4-6 days, advancement through the hepatic parenchyma - 5-6 weeks. During migration, liver tissue, blood vessels, and bile ducts are damaged. Necrosis, degeneration of hepatocytes, hemorrhages, infiltration of lymphoid, macrophage elements, plasma cells, eosinophils, thrombosis of small vessels and fibrosis of the surrounding liver tissue are noted. The intestinal flora carried by fasciolas can cause the development of microabscesses. 7 weeks after infection, fasciolae reach the bile ducts, where they transform into sexually mature forms. 12 weeks after invasion, fasciola eggs begin to be detected in bile and feces. Sometimes immature fascioli can be found in unusual places, such as the lungs, pancreas, lymph nodes, thymus, eyes, etc.; Fetal damage has been described in pregnant animals [17, 18].

The presence of adult fascioli in the bile ducts is accompanied by the development of a chronic inflammatory process, hypertrophy of the epithelium and fibrosis of the walls of the ducts. If there are accumulations of fascioli in the bile ducts, they become blocked and the outflow of bile is disrupted. Pathological impulses that occur as a result of irritation of biliary tract receptors by parasites lead to disruption of motility and functioning of the gastrointestinal tract (GIT). Sensitization of the body by fasciolae and the products of their metabolism causes the development of allergic reactions [11]. The incubation period ranges from 1 week to 2-3 months. There are a migratory (liver) stage of the disease (acute fascioliasis) and a biliary stage (chronic fascioliasis) [9, 11].

Acute fascioliasis usually begins with an increase in body temperature. The duration of fever can be up to several months. There is weakness, headache, pain in the right hypochondrium and epigastric region, sometimes without a specific localization, loss of appetite, bloating, nausea, diarrhea, and less often - vomiting and constipation. The liver is enlarged, not compacted, painful

on palpation. A number of patients have splenomegaly. Ascites may occur, the pathogenesis of which is associated with damage to the intestinal walls, liver capsule and irritation of the peritoneum during the migration of fascioli. Toxic-allergic reactions can manifest as polymorphic exanthema (maculopapular, urticarial); broncho-obstructive syndrome with shortness of breath, cough, hemoptysis, which in some cases serve as the first manifestation of invasion; myocarditis. The duration of the acute stage is 2-4 months. In endemic areas, repeated invasions superimpose the chronic phase of the disease. Chronic fascioliasis develops several months or years after infection and is manifested by symptoms of damage to the biliary tract (cholangitis and cholecystitis). Patients complain of poor health, loss of appetite, intolerance to fatty foods, nausea, vomiting, pain in the epigastrium, right hypochondrium, including an attack of “biliary colic,” enlargement and hardening of the liver. Blistering symptoms are positive. Some patients experience ascites [9-13].

Jaundice occurs when the biliary tract is obstructed by fascioli. The addition of a secondary bacterial infection leads to the development of purulent cholangitis and cholecystitis. With ectopic localization, fascioli can be found in the subcutaneous tissue, abdominal wall, appendix, spleen, pancreas, skeletal muscles, lymph nodes, heart, blood vessels, and epididymis [7, 8].

When it enters the brain, headache, convulsions and focal symptoms are noted. Invasion of the lungs and pleural cavities is accompanied by coughing and the appearance of blood in the sputum. When auscultating the lungs, dry or moist rales can be heard, especially often in the lower parts of the right lung, as well as pleural friction noise. Chest x-ray reveals pulmonary infiltrates (Loeffler's syndrome) and pleural effusion. There are reports of cases of spontaneous pneumothorax. When localized in the Eustachian tubes or middle ear, pain and hearing impairment are noted. Eye damage manifests itself as monocular blindness [11].

Fascioliasis can be complicated by biliary obstruction, bleeding from ulcers in the biliary tract, biliary cirrhosis, subcapsular hematomas of the liver, thrombosis of the hepatic, superior vena cava, mesenteric, myocardial and other veins, as well as pulmonary embolism [14, 15].

A clinical blood test reveals eosinophilic leukocytosis and signs of anemia; ESR accelerated. A biochemical blood test may reveal elevated levels of alkaline phosphatase, GGTP, and conjugated bilirubin. The content of serum gamma globulins, IgG, IgM and IgE increases [9, 11]. Parasite eggs appear in feces 3-4 months after infection. Enrichment methods (flotation and sedimentation) should be used. It is necessary to take into account the possibility of detecting fasciola eggs in the feces of persons who have eaten liver infected with parasites. This is the so-called “false” fascioliasis, in which the eggs of the parasite transit through the gastrointestinal tract of the subject. To exclude parasitic infestation, it is necessary to conduct repeated examinations (after 3-5 days) of the stool of such patients, from whose diet liver and offal are excluded [9, 11].

Using ELISA, RIF, RNGA, Western blot, antibodies to the secretory-excretory or recombinant antigen of fasciolae are detected, which appear 1-2 weeks after infection. Antibody levels usually decline 6-12 months after successful treatment. Fasciola antigens can be detected in blood serum and feces by ELISA 1-3 weeks after infection and disappear 2-4 weeks after cure [2, 4, 9, 11].

The drug of choice for the treatment of fascioliasis is triclabendazole: 10 mg/kg once or 10 mg/kg twice a day for massive invasion. The effectiveness of the latest treatment regimen is almost 100%. Triclabendazole is not registered in our republic. Praziquantel therapy is not effective [2, 15].

The basis for the prevention of fascioliasis is a set of sanitary and veterinary measures aimed at combating mollusks, preventing infection and deworming livestock. Prevention of fascioliasis in humans includes a ban on drinking unboiled and unfiltered water from stagnant bodies of water; heat treatment or the use of vinegar and potassium permanganate for washing plants growing in damp places and garden greens watered with water from polluted reservoirs [9-15].

Conclusion

Thus, to date, at least eight trematodes have been described that affect the human liver, bile and pancreatic ducts. Infection occurs through food through various foods, drinking water, or accidental ingestion. After emerging from the membranes, the parasites ultimately penetrate the bile and pancreatic ducts, where inflammatory changes develop that underlie the clinical manifestations of hepatic trematodes, along with allergic reactions to helminths and their metabolic products. Laboratory diagnostics are based on the coprooscopic method; serological and molecular genetic tests exist or are being developed. In most cases, treatment is with praziquantel, with the exception of fascioliasis, for which triclabendazole should be prescribed. Prevention includes sanitization of sources of invasion, prevention of fecal pollution of the environment, control of intermediate hosts, culinary processing of food products - factors for transmission of invasion, adherence to personal hygiene rules, etc.

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