

**LIVER FASCIOLISIS AS A RE-EMERGENCY DISEASE**

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**Abstract:** Fascioliasis is a helminthiasis that occurs with damage to the hepatobiliary system and is characterized by a long-term chronic course. Pathogens: *Fasciola hepatica* and *Fasciola gigantica*. At the stage of sexual maturity, fasciola parasitize humans and many herbivores, including sheep, goats, cattle, and less commonly pigs, horses, and dogs. The lifespan of fasciola in humans reaches 10 years or more. According to WHO, about 2.4 million people are currently affected by fascioliasis, and 180 million people live in areas where there is a high risk of contracting it. 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:** Liver fascioliasis, *fasciola hepatica*, parasitic diseases, helminthiasis, fascioliasis.

Fascioliasis is a helminthiasis that occurs with damage to the hepatobiliary system and is characterized by a long-term chronic course. The causative agents are trematodes *Fasciola hepatica* and *Fasciola gigantica*. At the stage of sexual maturity, fasciola parasitize humans and many herbivores, including sheep, goats, cattle, and less commonly pigs, horses, and dogs. The lifespan of fasciolas in humans reaches 10 years or more, in animals – 3–5 years. *Fasciola hepatica* - liver fluke has a flat leaf-shaped body measuring 20-30x8-12 mm. The front part of the body is covered with spines and extended into a proboscis. It contains oral and ventral suckers. The mouth opening on the corresponding sucker leads into the pharynx and further into the esophagus, from which two branches of the intestine with a large number of branching lateral processes extend. Following the abdominal sucker, in the front part of the body there is a rosette-shaped compact uterus, the loops of which are filled with eggs. Next are the branched ovaries and testes. Liver fluke eggs measure 0.13–0.145 mm. They are yellowish-brown in color and have an operculum and a thickened shell at the poles. *Fasciola gigantica* is a giant fluke. Its dimensions are 5–12 mm. The eggs of the giant fluke are brown in color and size 0.15–0.19 mm. Helminth eggs are released into the environment with the feces of infested animals and humans. In water or moist soil, a larva covered with cilia, the miracidium, forms in the egg within 4–6 weeks [3-6]. When it gets into the water, it invades the mollusk or dies if it does not penetrate this intermediate host within 8 hours. In the body of the mollusk, complex development and reproduction of larval generations of the helminth occurs, ending with the formation and release into the water of a larva - a cercaria, which has a tail. The tail of the cercarium soon falls off, and a special secretion is secreted, enveloping the larva and forming a closed capsule around it. The encapsulated larva is called an adoleoscaria. The larva attaches to the underside of aquatic plants. Infection of humans and animals - the final hosts - occurs through water, edible herbs growing in water bodies, on wet or irrigated lands, as well as through greens, vegetables and fruits washed with water contaminated with fasciola larvae. In areas with an abundance of small bodies of standing water, as well as in hot, humid climates, the risk of infection increases [1–4]. Geographical distribution. Fascioliasis is reported in different countries in the form of sporadic cases. This helminthiasis is more widespread in hot countries of Asia, Africa, and Latin America. For example, in Peru, the prevalence of schoolchildren reached 15.6%, and in some villages - 34%. Outbreaks of

fascioliasis affecting hundreds of people have been reported in Chile, Cuba and France. Sporadic cases of fascioliasis have been reported in the Baltic countries, Transcaucasia, and the Central Asian republics. Fascioliasis caused by giant fasciola has been detected in a number of African countries, Vietnam, the Hawaiian Islands and Russia [11-17].

The pathogenesis of fascioliasis lies in the traumatic effect of parasites. During the migration process, young helminths cause mechanical damage to the host tissues. Perhaps their glands secrete proteolytic enzymes that lyse liver cells. Adult parasites also have a mechanical effect, moving along the bile ducts and attaching to their walls with suction cups. The walls of the ducts and spines located on the surface of the front part of the helminth are damaged. Sometimes parasites completely or partially close the lumen of the ducts, slowing down or stopping the flow of bile. Metabolic products of helminths are allergens. The allergic effect of fasciola is most pronounced in the early stages of infection. Changes that occur in the wall of the bile ducts and disruption of the circulation of bile through them create conditions favorable for the development of secondary bacterial flora. In the liver in the early phase of fascioliasis, microabscesses and micronecrosis are formed with infiltration of eosinophils and giant cells. In the late period, expansion of the lumen, thickening of the walls and adenomatous proliferation of the epithelium of the bile ducts are observed, purulent cholangitis sometimes develops, and cases of obliteration of the hepatic duct are known [1, 5].

Clinical picture. The incubation period is 1–8 weeks. The acute stage of the disease begins with malaise, increasing weakness, headache, loss of appetite, sometimes hives, often fever, chills. On examination, the sclera is subicteric. Soon there is pain in the epigastrium and right hypochondrium, nausea and vomiting. The liver enlarges, becomes dense, painful on palpation.

A symptom that is pathognomonic for fascioliasis is a predominant enlargement of the left lobe of the liver, sometimes to the navel and to the left to the spleen, and bulging of the epigastrium. This enlargement of the organ develops acutely and is accompanied by severe pain. After the attack stops, the liver quickly shrinks to normal size. Sometimes the spleen becomes enlarged.

When examining blood in the early phase of the disease, as a rule, pronounced eosinophilic leukocytosis is observed with an increase in the number of leukocytes to  $18\text{--}20 \times 10^9/\text{l}$  and eosinophils (sometimes up to 85%). Over time, the acute phenomena of fascioliasis subside, fever (usually of the intermittent type) lasts from 2–5 to 14–21 days, then decreases, and the invasion enters the chronic stage. Dyspeptic symptoms and abdominal pain come to the fore. The pain sometimes becomes paroxysmal in nature, similar to gallstone colic, and is accompanied by an enlargement of the gallbladder, an increase in body temperature to  $38^\circ\text{C}$  and above, sometimes with the subsequent development of mild jaundice. The duration of attacks varies widely - from several hours to 7-8 days. They are separated from each other by periods when there is no pain, or they are insignificant. The liver usually protrudes from under the costal arch by 2–5 cm, but may not increase in size. Its consistency is dense, the surface is smooth. A blood test reveals a normal white blood cell count or moderate leukocytosis. Eosinophilia is usually mild (7–10%). In the acute phase of fascioliasis, signs of allergy usually appear most prominently, and in a later period - manifestations of cholangitis, sometimes purulent, biliary dyskinesia, and sometimes hepatitis. It should be taken into account that in some cases, fascioli have a perverted localization; they were found in the subcutaneous tissue, lungs, in the eye, in the area of the greater curvature of the stomach, in the abscess of the appendix, in the portal vein, in the abdominal cavity. As a result of a long unrecognized course of the disease, severe liver dysfunction, diarrhea, macrocytic anemia, exhaustion and even death can occur [9–13].

The diagnosis of fascioliasis in the acute stage can only be presumptive, since the parasite has not yet matured and does not release eggs. Epidemiological history is important. Parasite eggs appear in feces only 3–4 months after infection. At an early stage, fascioliasis is differentiated from

trichinosis, opisthorchiasis, and diseases of the blood system. Enzyme immunoassay reactions are used to detect specific antibodies against fasciola (enzyme-labeled antibody reactions) using a specific component of mature fasciola antigens, the sensitivity of which reaches 97%. Fasciola eggs are also found in duodenal contents [14]. We must not forget that when eating the liver of infected livestock, human infection does not occur, but transient eggs from the animal can be found in feces. These eggs pass through the human gastrointestinal tract, being damaged by the action of gastric juice, enzymes and partially changing in the process. In doubtful cases, the study should be repeated after a few days to make sure that they are not dealing with a true invasion, but with the transient passage of eggs [8].

Differential diagnosis in the acute phase of the disease is carried out with viral hepatitis, leptospirosis, cholelithiasis, obstructive jaundice of other etiologies. The main signs of acute fascioliasis are the presence of jaundice only during fever, severe pain (it is not typical for viral hepatitis), leukocytosis, hypereosinophilia, significantly accelerated ESR, severe manifestations of allergies, which are very rare in viral hepatitis and obstructive jaundice [19, 20].

The drugs triclabendazole (250 mg tablets) and oxamniquine (250 mg capsules and syrup: 250 mg of the drug in 5 ml of syrup) are used abroad. These drugs are prescribed at a dose of 10 mg/kg (in severe cases 20 mg/kg) in 2 doses with a 12-hour break for one day. Specific treatment is also carried out with praziquantel (and its analogs) at a dose of 60 mg/kg per day in 3 doses for 2–3 days. Bithionol is also used at a dose of 30–50 mg/kg every other day. The course is 10–15 doses. In addition to specific therapy, pathogenetic and symptomatic treatment is carried out [16].

In the early phase of fascioliasis, antihistamines are prescribed for allergic phenomena. In the late stage, tubage of the bile ducts is performed with or without a probe. In the latter case, the patient drinks on an empty stomach 30 ml of a 33% solution of magnesium sulfate, sorbitol or egg yolk and lies with a heating pad on the right side for 2 hours. Tubage is prescribed 1-2 times a week for several months. For pain, antispasmodics are used. Choleric agents and vitamins are indicated in complex treatment. In case of secondary bacterial infection, there is a need to prescribe antibiotics [7–13]. The prognosis for timely recognition and vigorous treatment of fascioliasis is usually favorable. In severe cases, especially with the addition of a secondary infection, the prognosis worsens [8].

Prevention of fascioliasis involves avoiding drinking unboiled water from stagnant or slow-flowing sources. Water should be passed through filters and boiled. As a last resort, the water is filtered through canvas. Aquatic plants from stagnant bodies of water should be consumed only after heat treatment or scalding with boiling water. Great importance in the fight against fascioliasis is attached to the veterinary service, which carries out a complex set of measures (identification and treatment of sick animals, destruction of shellfish, sanitary improvement of livestock farms, deworming of manure, determination of safe places for grazing animals, etc.).

Thus, carriage of fasciolae is much more common than expected, and because of this, fascioliasis is far from being fully detected.

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