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Hepaticofasciolas (Fasciolosis) as a Cause of Mechanical Jaundice in a Woman (Case Study)

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ABSTRACT

The article describes a rare obstructive jaundice caused by hepaticofasciola. The clinical picture was characterized by cholestasis, pruritus and acholia. The patient was discharged home in a favorable condition after endoscopic retrograde cholangiography and parasite extraction.

The paper presents a rare clinical observation of biliary fasciolosis in a 35-year-old woman, which caused obstruction of the extrahepatic bile ducts and obstructive jaundice.

Fascioliasis (Fasciolahepatica) is a zoonotic biohelminthiasis characterized by damage to the hepatobiliary system. The main sources of invasion for humans are sheep and cattle. Infection occurs when drinking non-disinfected water from open water bodies, as well as algae [1,3]

Etiology. The main carrier of fasciola hepatica is cattle, goats and sheep. With the excretion of the feces of infected animals, the larvae of the parasite penetrate into the environment. The causative agent of fascioliasis is - Fasciolahepatica - liver fluke, having a length of 20 - 30 mm. Consider the fasciola cycle. Flukes parasitize in the biliary system of cattle and small cattle, pigs, horses, sometimes rats, and also humans, which are the final hosts of the parasite. Fluke eggs are excreted into the environment with feces and enter a freshwater reservoir. At the next stage of development, they need freshwater warm water. The optimum temperature is considered to be 23 °C. If the temperature is low, about 10 °C, the development of parasites stops, and too high, about 30 °C, is detrimental to fluke larvae. [2,4]

Under favorable conditions, after 10-17 days, parasites appear from the eggs that are able to move independently, where larvae - miracidia - hatch from the eggs, which penetrate the body

of the intermediate host - the mollusk (small pond snail). The next stage in development is the intermediate host, the freshwater snail. This phase is mandatory for this microorganism and lasts approximately 1-2 months.

After many metamorphoses, mobile circaria emerge from the small pond snail, which drop their tail and turn into a spherical shape - adolescaria. The latter are attached to aquatic plants or to the surface film of water. Then it attaches itself to the leaves and stems of plants, or simply sits on the surface of the water and waits for its prey.

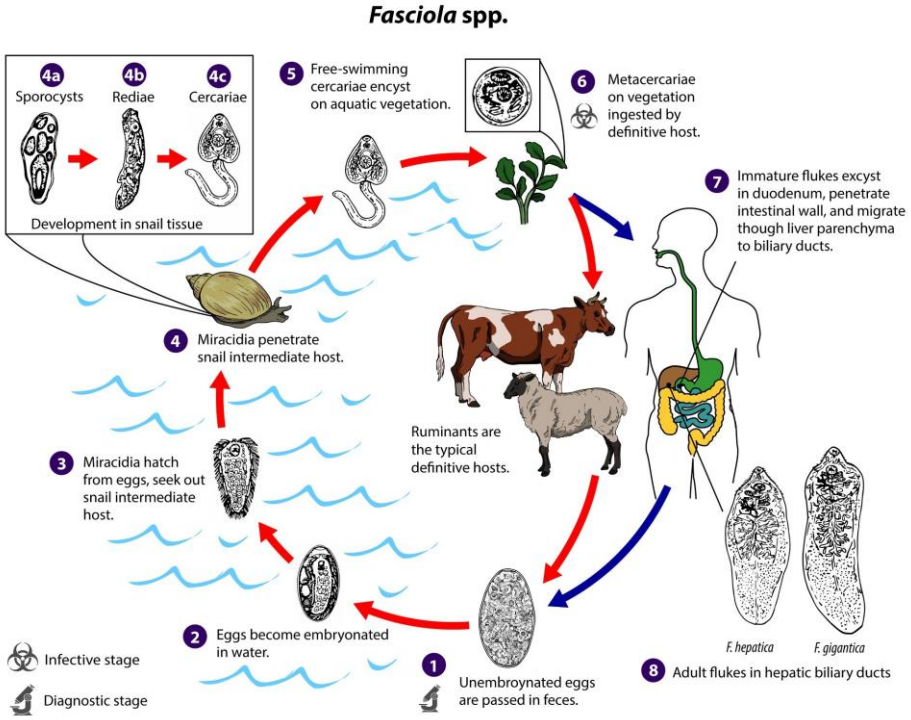

Figure 1. The cycle of development of hepaticofasciol in nature and in the human body

Pathogenesis and pathomorphology. Adoleskaria that enter the human body penetrate through the portal vein system or through the peritoneum, Glisson's capsule into the liver, then into the bile ducts, where they reach puberty in 3–4 months. Migration of larvae is accompanied by toxic-allergic reactions and liver damage. Parasitization of adults leads to the development of proliferative cholangitis, fibrosis of the walls of the bile ducts and gallbladder, sometimes obstruction of the ducts and the addition of a secondary bacterial infection.

When the fluke enters the human or animal body by ingestion, it reaches the digestive tract, where its protective shell dissolves. Next, the parasite breaks through the intestinal wall and enters the abdominal cavity, where the process of its migration to the liver begins. Reaching this organ, hepatic fasciola tries to penetrate the bile ducts. [4]

There is another way to penetrate to the habitat - the fluke enters the blood from the intestines and reaches the liver through the bloodstream (hematogenous method). Migration of the parasite can last several months. After sticking to the wall of the liver of an infected person for 2 months, the fluke forms a hermaphroditic reproductive system. Then, after about 4 months, fasciola begins to lay eggs. The disease in humans can take a very long time, about 5-10 years, in some cases even more . Human infection with fasciola hepatica occurs in the process of eating untreated water and unwashed plants, by ingesting parasite eggs while swimming in open water, or in the process of using contaminated water to wash fruits and vegetables. Therefore, residents of settlements remote from cities, who often use untreated water from reservoirs, are most likely

to be infected with this helminth. Fluke cannot be infected from infected people, since the parasite must go through certain stages of development in the body of snails, and only after that the helminth is dangerous to humans.

Let's take our example. Patient S. M., born in 1988, was admitted to the emergency department of the Bukhara branch of the RRCEM with complaints of pain in the right iliac region, nausea, vomiting, yellowness of the skin, and weakness. From the anamnesis he has been ill for 7 days. Conducted instrumental and laboratory studies in the admissions and surgical department.

clinical picture. The incubation period is 1–8 weeks. There are acute and chronic stages of fascioliasis. The disease begins acutely with fever, sweating, weakness. There are urticarial rashes on the skin, symptoms of asthmatic bronchitis, pain in the epigastrium and right hypochondrium, often jaundice, enlarged liver and spleen. Possible myocarditis. X-ray examination reveals "flying" infiltrates, pneumonia. Blood tests reveal leukocytosis, hypereosinophilia (up to 85%). Acute phenomena gradually subside, and the disease passes into the chronic stage, and in some patients the acute stage is absent, and the chronic stage is the first manifestation of invasion. In the chronic stage, allergic phenomena (itchy rashes, eosinophilia up to 20%) persist, but signs of biliary tract damage come to the fore: pain and heaviness in the right hypochondrium, nausea, transient jaundice. When a secondary bacterial infection is attached, the pain becomes intense, takes on a paroxysmal character, fever, jaundice, and hyperbilirubinemia appear. The activity of alkaline phosphatase increases. Possible purulent cholangitis, liver abscesses. Chronic fasciolosis can occur with predominant manifestations of gastroduodenitis. The duration of the invasion is 5–7 years, but even after the release of the organism from the parasite, the biliary tract damage can persist.

Diagnosis and differential diagnosis. In the acute period of the disease, fever, toxic-allergic symptoms, hypereosinophilia suggest helminthic invasion. The diagnosis can be confirmed by immunological tests (RNGA, RIF, ELISA). After 3 - 4 months. after infection, fasciol eggs can be found in the duodenal contents. Differential diagnosis is carried out with other clinically similar helminthiases (opisthorchiasis), with typhoid fever, cholecystitis and cholangitis of another etiology, hepatitis.

Patient X., aged 35, was admitted to the emergency department of the Bukhara branch of the RRCEM with complaints of recurrent pain in the epigastric region and in the right hypochondrium, with periodic icterus of the sclera, occasionally acholic stool. The general condition upon admission is closer to moderate severity. Consciousness is clear. On examination, the reaction is adequate. Appetite saved. Skin, visible mucous membranes and sclera are icteric. Heart tones are clear, A/D 120/80 mm., pulse - 86 beats/min Hg. Art., BH 20 per min. The abdomen is of the usual shape, symmetrical, evenly participates in the act of breathing, is soft on palpation, accessible, moderately painful on deep palpation in the right hypochondrium. The liver protrudes from under the edge of the costal arch +2.0 cm, the surface is smooth, the edges are sharp, of normal consistency. The spleen is not palpable. There is no free fluid in the abdominal cavity. Symptom Pasternatsky negative on both sides. The stool is regular, of normal color and consistency. Urination adequate, painless.

Analyzes: HB-85g / l, erythrocytes - 2.6 million; CPU-0.9. Platelets - 205 109/l; leukocytes 4-109/l; ESR-16mm.

Biochemical blood test: total protein-64g/l, glucose - 5.1 mmol/l, urea - 3.0 mmol/l, bilirubin - 58.0 µmol/l, ALT-233 U/l, AST-140 U/l, fibrinogen - 3.3 g/l.

Urinalysis: no protein, relative density -1024., color - light yellow, pH-6.4.

Ultrasound of the gallbladder 96-34 mm, wall thickness 3-4 mm, in the cavity there are stones up to 3-4 mm in size. OZHP 7 mm. Moderate choledochoectasia. Intrahepatic ducts are not dilated.

Preliminary diagnosis: GSD. Choledocholithiasis? complications: obstructive jaundice.

Treatment. The patient, for the purpose of diagnosis and treatment, underwent FGDS, ERCP, EPST.

Fig.2. Hepatica fasciola parasite (up to 5 cm long)

In the operating room under an image intensifier tube with an X-ray machine using a FUGINON video duodenoscope , after preliminary sedation and local anesthesia of the oropharynx with a solution of lidocaine, duodenoscopy was performed. There is bile in the duodenum, the papilla is normal without pathology. Next, a standard conjugation of the BDS was performed and the CBD was contrasted, the latter up to 13-15 mm wide, using a string papillotome, papillotomy was performed over 10 mm. When auditing the CBD with a Dormia basket, no stones were found, live hepaticofasciola was extracted into the 12th duodenum. The parasite is extracted through the mouth. Parasite up to 5 cm long, up to 0.5 cm wide. In the postoperative period, the disease clinic, bilirubin decreased to normal levels, and the patient was discharged home in a satisfactory condition.

In the acute stage, detoxification and desensitizing therapy was used. After the symptoms subsided, antihelminthic therapy with albendazole or praziquantel was prescribed.

Forecast. In general, it is favorable for life, but with intensive invasion, long-term lesions of the biliary tract are possible. Prevention is aimed at combating fascioliasis in animals, protecting water bodies from fecal pollution. Refusal to use non-disinfected water from open reservoirs for drinking and washing vegetables.

Thus. Fascioliasis is a rare, but occurring pathology for the countries of Central Asia. This clinical example, which was hidden under the clinic, characteristic of the calculous nature of the breast, is of great interest due to its rarity and once again proves the high efficiency of endoscopic interventions in the diagnosis and treatment of biliary tract obstructions.

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