Fasciola hepatica as a cause of jaundice after chewing khat: a case report

L.C.J. de Bree*, A.G.L. Bodelier, G.P. Verburg

Department of Internal Medicine and Gastroenterology, Amphia Hospital, Breda, the Netherlands,
*corresponding author: tel: +31 (0)76 5955000, fax: +31(0)76 5952410, e-mail: cdebree@amphia.nl

ABSTRACT

Fasciola hepatica is a worldwide distributed zoonotic trematode incidentally infecting humans. Although often asymptomatic, fascioliasis can cause a wide spectrum of disease. The diagnosis can be established by stool examination detecting ova of the parasite, although serological testing has a higher sensitivity and specificity in the acute phase of disease. This case presents a 24-year-old Somalian man admitted with jaundice and abdominal discomfort after fascioliasis. The patient was treated successfully with a single dose of triclabendazole.

KEYWORDS

Fasciola hepatica, fascioliasis, jaundice, parasitosis, khat

CASE

A 24-year-old Somalian man was admitted because of jaundice. He had a one-month history of pruritus and longer existing intermittent upper abdominal discomfort. He was known for a bilateral panuveitis caused by tuberculosis for which he had completed tuberculostatic treatment. He lived in the Netherlands and his last visit to Somalia was four years ago. Physical examination revealed jaundice and right hypogastric tenderness. The liver and spleen were not enlarged. Laboratory findings revealed a total white blood count of 10.0 x 10⁹/l (normal values 5.0-10.0 x 10⁹/l) with marked eosinophilia of 5.0 x 10⁹/l (0-0.5 x 10⁹/l), as well as elevated liver tests with a total and conjugated bilirubin of 157 μmol/l (<20 μmol/l), alanine aminotransferase 68 U/l (<35 U/l), alkaline phosphatase 394 U/l (<115 U/l), gamma glutamyltranspeptidase 46 U/l (<55 U/l) and lactate dehydrogenase 394 U/l (<248 U/l). Abdominal ultrasound was completely normal. Serological tests for hepatitis A, B and C, Epstein-Barr virus, cytomegalovirus, human immunodeficiency virus, strongyloides and schistosoma infection were negative, as well as markers for autoimmune hepatitis. Stool samples sent for microscopic examination did not show any parasites. MRCP revealed a dilated common bile duct with distal tapering (figure 1). Serum Fas2 ELISA, detecting IgG-antibodies of the liver fluke Fasciola hepatica, was positive at a titre of 1: 350 (negative <1:32). In new stool samples, ova of the liver fluke were detected (figure 2). The patient admitted chewing freshly imported khat leaves, a psychoactive drug traditionally used and cultivated in the Horn of Africa, which was most likely the source of infection. Patient was treated successfully with a single dose of triclabendazole 950 mg orally.

DISCUSSION

Fascioliasis is a zoonotic foodborne disease caused by the trematode Fasciola hepatica. This parasite, causing liver rot in sheep and cattle, incidentally infects humans after ingestion of contaminated water, fish or uncooked aquatic vegetation. Although the source of fascioliasis in this case could not be proven, it is very likely attributed to chewing freshly imported khat leaves. Anecdotal evidence of fascioliasis after chewing khat has been reported before.1,2

As one of the ‘neglected’ tropical diseases, F. hepatica has a substantial impact on global public health, with an estimated 2.4-17 million people infected worldwide.3 Nowadays F. hepatica has a worldwide distribution including Europe, where a watercress-related outbreak in France was reported.4 However, highest prevalences are reported in Andean countries, Northern Africa and parts of the Middle East.3,6

After ingestion, metacercariae excyst and migrate in 4-6 weeks through the intestinal wall and Glisson’s capsule to the liver where they cause parenchymatous destruction, and migrate to the biliary ducts. Matured liver flukes release eggs via the biliary system into the small bowel.
Subsequently the eggs become embryonated and upon reaching water they release miracidia, which are capable of invading freshwater snails as their intermediate hosts. Subsequently cercariae are released and transformed to metacercariae on aquatic vegetation.

Although often asymptomatic, *F. hepatica* can cause a wide spectrum of disease depending on the degree of fluke burden and disease stage. The acute hepatic phase is usually mild and is sometimes accompanied by fever, abdominal pain and hepatomegaly. Biliary colics, epigastric pain and jaundice can be seen in the chronic biliary phase, which can last from a matter of months to up to ten years. Complications such as cholangitis, pancreatitis and cirrhosis can occur. Marked eosinophilia is the most common laboratory finding.

Detection of eggs in faeces is considered to be the gold standard for diagnosing fascioliasis, although eggs are often undetectable during the acute phase of the disease. The diagnosis can also be established serologically by Fas2 ELISA, which is able to detect IgG-antibodies against Fas2 antigen as early as ten days after infection. Therefore it can be used in the initial phase of disease, having a sensitivity of 92.5% and a specificity of 70.8-92.9%. In the acute phase, characteristic findings on imaging studies are multiple subcapsular small nodular or branching lesions in the liver. In the chronic phase, bile duct dilatation, gallbladder or bile duct oedema and even parasites can be seen. In any phase of disease, successful treatment can be achieved with triclabendazole, in a single dose of 10 mg/kg. As in many other countries, in the Netherlands triclabendazole is only available via veterinary pharmacies. Cure rates of more than 90% are reported.

**CONCLUSION**

As illustrated in this case, diagnosing fascioliasis can be challenging in non-highly endemic countries. However, *F. hepatica* infection should be considered in patients with abdominal pain, jaundice and eosinophilia. We recommend serological testing above stool sample microscopy in early stages. Special attention should be given to food, drugs and travel history.

**REFERENCES**