Capillaria hepatica infection: a rare differential for peripheral eosinophilia and an imaging dilemma for abdominal lymphadenopathy

Rajaram Sharma, Amit K. Dey, Kartik Mittal, Puneeth Kumar, Priya Hira

Department of Radiology, Seth GS Medical College and KEM Hospital, Acharya Donde Marg, Mumbai – 400012, India

Corresponding author: Amit K. Dey; e-mail: amit5kem@gmail.com

ABSTRACT. Capillaria hepatica which accidentally infects humans is a zoonotic parasite of mammalian liver, primarily rodents and causes hepatic capillariasis. The diagnosis is difficult because of the non-specific nature of clinical symptoms, leading to misdiagnosis and can be confirmed only through liver biopsy or on autopsy results. This paper is written with an objective to report a new case of hepatic capillariasis as a rare differential for peripheral eosinophilia and an imaging dilemma for abdominal lymphadenopathy.

Key words: Capillaria hepatica, liver biopsy, treatment, CT scan, child, India

Introduction

Capillaria hepatica which accidentally infects humans [1] is a zoonotic parasite of mammalian liver, primarily rodents [2]. It is a nematode of the family Trichocephalidea, class Tricuroidea and was discovered by Bancroft in 1893 [3]. The diagnosis is difficult and can be confirmed only through liver biopsy or on autopsy. There is formation of granulomatous inflammation along with necrosis [1]. Majority of cases reported were in children, may be due to frequent soil-hand mouth contact [4]. This paper is written with an objective to report a new case of hepatic capillariasis in a pediatric patient in India confirmed by liver biopsy.

Case presentation

A 3-year-old boy came with the chief complaints of fever (moderate to high grade) since two months and upper abdominal distension since three weeks. History of pica was present. No history of cough, lymphadenopathy or Koch’s contact was present. Child was vegetarian and belonged to lower socioeconomic strata. Pallor and mild hepatomegaly was seen on physical examination. On routine blood investigations 68 percent eosinophilia was reported. WBC count was 44300, polymorphs 23%, lymphocytes 8%. Chest x-ray was normal. Patient was treated with albendazole for three days but eosinophilia was persistent even after that. Patient was further investigated with IGE levels and LFT which were normal. Subsequently, ultrasonography was done which showed mild hepatomegaly, however no focal lesion was seen. So bone marrow biopsy was done in view of persistent eosinophilia which revealed reactive pattern of eosinophilia with no underlying bone marrow pathology. A CT scan of abdomen was done which showed diffuse sub-centimetric hypodense lesions in liver and non-necrotic retro and para aortic and portal lymphadenopathy measuring 3–4cm raising suspicion of lymphoma (Fig. 1a–d). Finally liver biopsy was done due to inconclusive diagnosis which showed necrotizing granulomatous inflammation of liver parenchyma secondary to parasitic infection, special stains highlighting the pathognomic eggs of Capillaria hepatica (Fig. 2). The child was treated simultaneously with albendazole and steroid for two weeks and Diethylcarbamazine for 21 days. On follow up the
child recovered and eosinophilia levels were normal.

Discussion

Capillaria hepatica has monoxenic life cycle [1]. Both, the adult and larva form remain in the liver of the host organism [5]. Capillaria hepatica is usually found in the liver of rats, mice, dogs, cats, pigs, monkeys and rabbit, whereas humans are infected accidentally [1].

The eggs are 54–65 µm in length and 29–33 µm in width, bioperculated, elliptical in shape, double enveloped external envelope is thinner than internal having sagittal striae between them. The eggs reach the soil either by decay of the infected host carcass, or through the feces of a predator that has fed upon the infected host [1]. The infection is acquired either by ingesting the food or soil contaminated [1,6] with embryonic egg or non-embryonic eggs called as genuine and spurious infection, respectively [1]. While the later results in gastrointestinal symptoms [5] the former may pass through the intestinal wall and via portal vein and may lodge in liver or other organs where they grow into adult form [7]. The mature form mates producing millions of egg in liver and the organism dies in around 40–60 days [1].

The larva that hatches from embryonated eggs moves to the intestine and subsequently to the liver where they mature and mate. It also moves to other organs like the lungs and the spleen where it doesn’t mature and dies almost immediately. Symptoms of hepatic capillariosis range from mild to severe with varied symptoms. Apart from the classic triad of persistent fever, hepatomegaly, eosinophilia others are respiratory alterations, vomiting, splenomegaly, pneumonitis, extreme weakness, constipation, abdominal distension, and sometimes ascites and malnutrition [1]. In earlier stages immature worms
without eggs are seen in infected host followed by, adult worms and eggs along with granulomatous inflammation in advanced cases. In the end stages eggs are seen with inflammation and fibrosis of liver parenchyma without adult worms [8].

*Capillaria hepatica* may be associated with lymphadenopathy which can be detected on CT scan [8]. Because it has no specific clinical symptoms and is very rare therefore it’s a diagnostic dilemma and frequent misdiagnosis have been made [12]. A definitive diagnosis in the case of hepatic capillariasis can be made only by obtaining a liver biopsy but then biopsy is a traumatic diagnostic approach [13]. Since no immunodiagnostic method is currently available and the detection of eggs in the feces only characterizes a spurious infection it is a diagnostic difficulty [1]. One of the diagnostic difficulties is false positive serologic test with other conditions like toxocariasis [1]. The infection of larva migrans, diffuse infection by *Strongyloides stercoralis*, ascaridiasis of the liver, amebic hepatitis, infectious hepatitis, pyogenic hepatitis produces similar clinical condition and hence need to be differentiated clinically [1,9]. Anti-nematoid drugs like thiabendazole or albendazole for 3 months is the treatment of choice. Ivermectin can produce similar clinical condition and hence need to be differentiated clinically [1,9]. Anti-nematoid drugs like thiabendazole or albendazole for 3 months is the treatment of choice. Ivermectin can develop. Severity of infection decides prognosis [10,11]. Clinical manifestation of varying intensity can develop. Severity of infection decides prognosis and can range from mild to severe and even fatal. Hepatomegaly may be present even after treatment which gradually subsides over a period of months to year [1].

Considering the high prevalence of *C. hepatica* infection of rats in large cities and in areas with unhygienic and poor socioeconomic conditions rodent control in zoos and houses and improving the socioeconomic conditions are important methods of prevention. Also because it is more common in less than 5 years of age population due to frequent soil hand mouth contact preventing such practices can help in prevention of the disease. Animal Disease Surveillance Plan also needs to be carried out to prevent outbreaks [1,12,14].

**Conclusions**

In India abdominal lymphadenopathy is mostly due to tuberculosis and lymphoma. However, parasitic infection like *Capillaria hepatica* should also be kept in mind. A complete clinical, biochemical and radiological co-relation may help to clinic this rare diagnosis of parasitic infection and rule out other differentials of abdominal lymphadenopathy. Although it is a diagnostic dilemma though traumatic still liver biopsy remains the gold standard for diagnosis of this rare entity. Preventive measures need to be taken for stopping outbreaks.

**References**


Received 17 January 2015
Accepted 23 February 2015