

HEPATIC FASCIOLIASIS

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ABSTRACT

A case of hepatic fascioliasis without peripheral eosinophilia is presented. CT scan revealed a cluster of small abscesses in subhepatic area of the liver with tract like fashion. The adjacent subhepatic cyst was noted as a complication of the disease.

INTRODUCTION

Fascioliasis, caused by the trematode *Fasciola hepatica* or less common by the *Fasciola gigantica*, is a common parasitic disease in cattle and sheep. Humans are accidental hosts during the life cycle of the parasites.^{1,2} They are infected by ingesting contaminated watercress,³ water,⁴ raw fish⁵ or infected raw sheep or cattle livers.³

In human fascioliasis, two stages of disease have been distinguished; an acute phase, which coincides with hepatic invasion, and a chronic phase caused by persistence of adult flukes in the bile ducts.² Triad of fever, hepatomegaly and high peripheral eosinophilia was known as characteristic of the disease. Ultrasonographic and CT findings of the disease had been reported.⁶⁻⁹ However, radiologic findings of complicated cases were seldom demonstrated.

We report a case of hepatic fascioliasis with no peripheral eosinophilia and a large subhepatic cyst.

CASE REPORT

A 48-year-old man was admitted to the hospital because of severe epigastric pain and

syncope for 2 hours. He had experienced abdominal discomfort for 1 year, anorexia and weight loss for 4 kgs in 2 months. Physical examination revealed fever (T 38-39 degree celsius), and no jaundice. During the examination, mild hepatomegaly and tenderness at epigastrium and right upper abdomen were noted. Laboratory findings showed Hct 47.5%, mild leukocytosis (WBC 11,900/mm,³ N 80, L16, M3, B1). Urine and stool examination were within normal limit. Liver enzymes were slightly elevated; alkaline phosphatase 104 (20-90) U/L, alkaline Am. trans 37 (6-36) U/L, G.Glutamyl trans 67 (5-38). Lipase and amylase were within normal limit. HIV antibody and AFP were negative. CEA was 3-31 mg/ml. Ultrasound revealed multiple small hypoechoic lesions in the left lobe of liver and a large cystic mass between left lobe of liver and the pancreatic head region (Fig.1).

CT scan showed a cluster of small, low attenuation lesions in the periphery of the medial segment of the left lobe. They were arranged in linear fashion. A large cystic mass about 7.5 X 5 X 5 cm. is noted containing few septations protruding from the subhepatic area to the peripancreatic region (Fig. 2). Inflammatory thickening

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of the surrounding fat and the gallbladder was noted (Fig. 3). Explore-laparotomy was performed, a large hepatic abscess was found about 8 cm. in diameter, protruding from the liver surface (segment IV) with a liver fluke 1 X 1.5 cm in this abscess. Intraoperative cholangiography was normal. Draining of the abscesses and cholecystectomy were performed. Pus culture showed no growth after 3 days. Histologic analysis showed a parasitic cyst, Fasciola specie in the liver tissue.

Gall Bladder specimen showed evidence of cholecystitis.

After medical treatment with Albendazole, improvement was seen clinically. Sonography obtained at 3 weeks later showed multiple small hypoechoic areas in the medial segment of left lobe with mild hepatomegaly (Fig. 4).

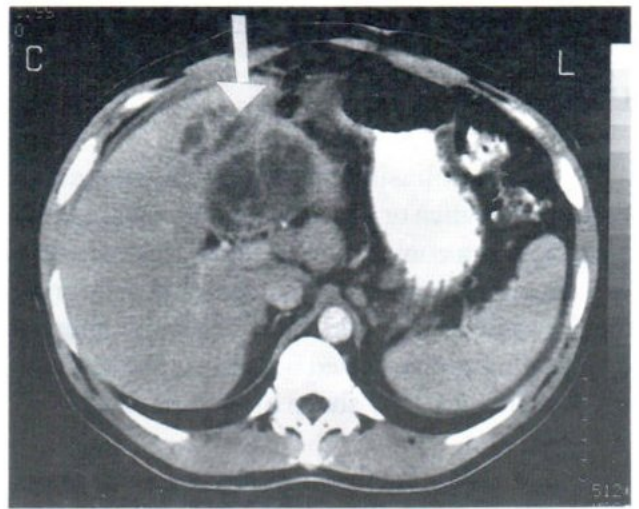
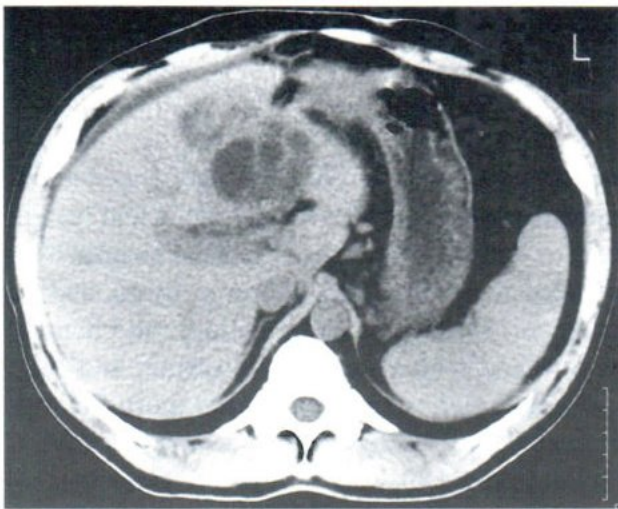


Fig.1 CT scan of the liver revealed a cluster of small, low density areas in subhepatic region of left lobe with tract like fashion (arrow). Minimal free fluid was seen at anterior aspect of the liver (arrowhead).

A. unenhanced

B. contrast enhanced study

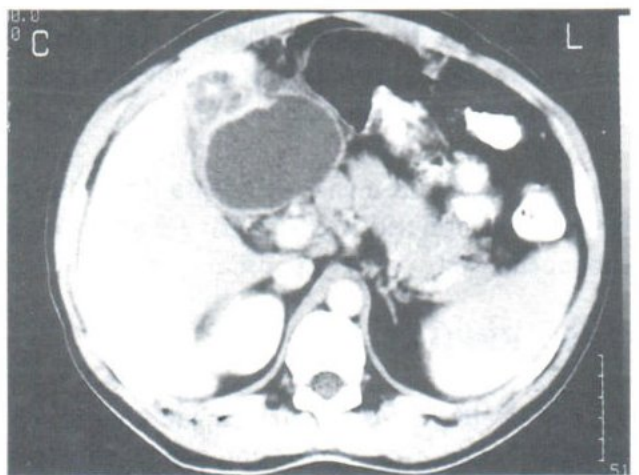
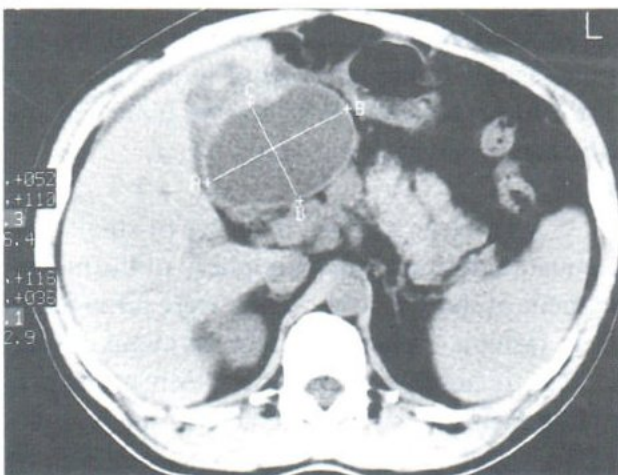


Fig. 2 Large cystic mass containing few septations protruded from the subhepatic area to pancreatic head region

A. unenhanced

B. contrast - enhanced study

DISCUSSION

Human fascioliasis results from the ingestion of metacercaria contaminated plants and water. The clinical course has two stages. The first stage or acute stage is the stage of hepatic invasion. When the metacercaria hatches in the digestive tract, the immature fluke penetrates the intestinal wall and enters the abdominal cavity. It then penetrates Glisson's capsule and enters the

liver parenchyma. This stage usually lasts 1-3 months, is characterized by fever, pain in the right upper abdomen, hepatomegaly and eosinophilia. No eggs or flukes are detected, but the results of serologic tests are usually positive.⁷ In the second or chronic stage begins after the immature flukes migrate throughout the liver until they reach the bile ducts. The immature flukes

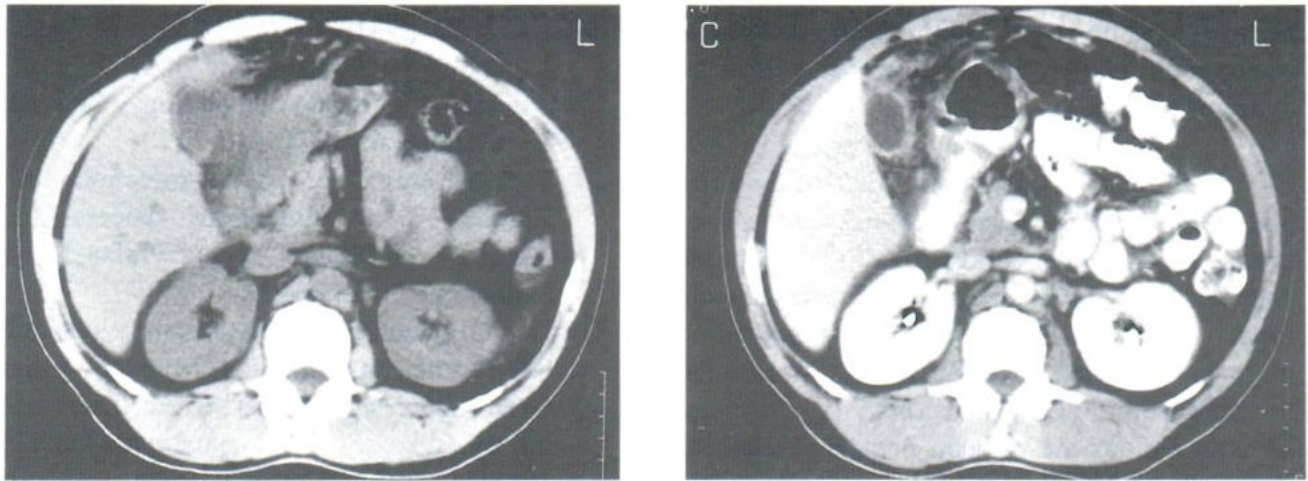


Fig. 3 Inflammatory thickening of the surrounding fat and the gallbladder (arrowhead)
 A. unenhanced
 B. contrast enhanced study

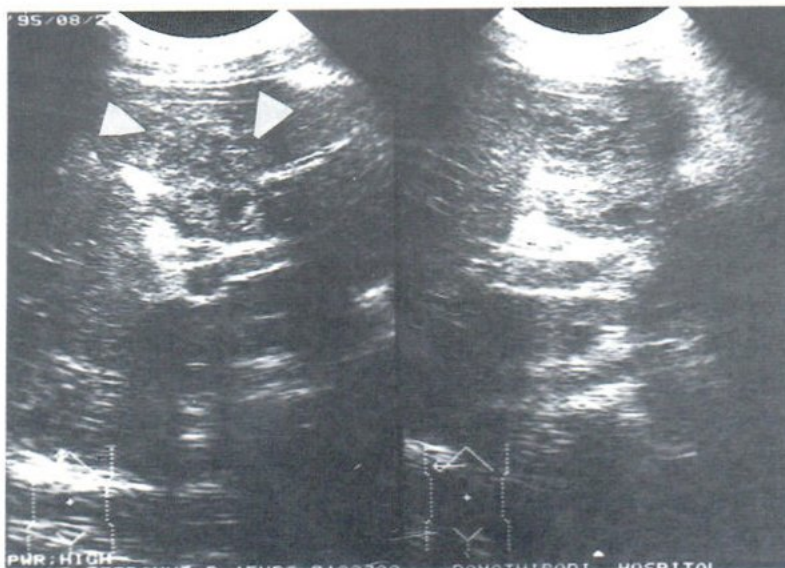


Fig. 4 Sonogram taken 3 weeks later showed ill defined hypoechoic areas in the left lobe of the liver (arrowheads).

mature in the bile ducts after 2 months and produce eggs and metabolites which irritated the bile duct mucosa. The symptoms are caused by the obstruction of the bile ducts such as recurrent episode of biliary colic, angiocholitis and cholecystitis.^{1,8-10} The diagnosis is enabled by identification of flukes or eggs in the bile fluid or feces.

Complications of hepatic fascioliasis are invasion of the gallbladder, hepatic subcapsular hemorrhage, hemobilia and ectopic migration causing pulmonary infiltration, lung mass or hydropneumothorax.¹¹

The CT characteristics of hepatic fascioliasis^{8,12} are 1). clusters of microabscesses arranged in tract-like fashion. The tortuous channels have also been described at surgery and laparoscopy¹³ and probably correspond to migratory tracts left by the flukes. 2). Subcapsular location of the hepatic lesions. If peripheral tortuous lesions are present, hepatic fascioliasis should be the primary consideration. 3). Very slow evolution of the lesion on follow up examination.

In our patient, he had clinical symptom of fever, right upper abdominal pain and mild abnormal liver biochemical test. The initial diagnosis was difficult because of no peripheral eosinophilia. CT scan showed clusters of small abscesses with nodular and tract-like fashion, located at subcapsular area of the liver, corresponded to previous reports.⁶⁻⁹ Small perihepatic fluid could be secondary from the penetration of Glisson's capsule by the liver flukes. The large cystic mass with thin enhancing wall containing internal septations suggested complicated cyst or abscess. This finding occurred because the immature flukes were trapped in the immediate subcapsular tissue of the liver where they died leaving a cavity filled with necrotic debris. Therefore, most of the hepatic lesions were confined to a depth less than 2 cm, beneath the capsule.¹⁴ However, the CT findings may be

impossible to differentiate from the cluster appearance of small pyogenic abscesses.¹⁵

In summary, hepatic fascioliasis may be presented without peripheral eosinophilia. CT characteristics of cluster of microabscesses arranged in tract like fashion and subcapsular location may be helpful in the diagnosis. Perihepatic cysts may be the complications of the disease.

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