OBSTRUCTIVE BILIARY CIRRHOSIS IN A CAT DUE TO PLATYNOSOMUM FASTOSUM INFECTION

Cirrosis biliar obstructiva en un gato por infección con Platynosomum fastosum

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ABSTRACT

Biliary cirrhosis is rare in domestic animals and has scarcely been reported in cats. Most cases of biliary cirrhosis in cats are related to parasitic infections by Amphimerus pseudofelineus, Metorchis complexus, and Opistorchis pseudofelineus.
A case of obstructive biliary cirrhosis caused by a severe infection by the liver fluke Platynosomum fastosum and its gross and microscopic lesions are describe in a cat.

RESUMEN

La cirrosis biliar es rara en los animales domésticos y escasamente ha sido reportada en gatos. La mayoría de los casos de cirrosis biliar en los gatos están relacionadas con infecciones parasitarias por Amphimerus pseudofelineus, Metorchis complexus y Opistorchis pseudofelineus. Un caso de cirrosis biliar obstructiva causada por una infección severa por Platynosomum fastosum y sus lesiones macroscópicas y microscópicas son descritas en un gato.

INTRODUCTION

Liver cirrhosis is an uncommon disease in domestic animals. Its prevalence is unknown in dogs and cats, although biliary cirrhosis is more common in cats[17]. There are few reports of biliary cirrhosis in the cat[3, 14]. Both related to Amphimerus pseudofelineus, Metorchis complexus, and Opistorchis pseudofelineus infection. Though cases of liver fibrosis and one case of an unclassified liver cirrhosis in cats infected by Platynosomum fastosum have been reported[5, 11], no cases of biliary cirrhosis caused by this parasite had been described previously.

CASE HISTORY

A female, 2 year-old domestic short-hair cat was brought to the University of Zulia Veterinary Hospital presenting ascites, vomiting, jaundice and emaciation. The ascitic fluid had 3.5 g% of protein, pH 8 and 2,700 leucocytes/dl (mostly segmented neutrophils and macrophages). Blood analysis revealed a 30% PCV, 9 g% Hb and a white cell count of 32,400/dl (37% lymphocytes, 47% segmented neutrophils, 8% stab neutrophils, 7% eosinophils and 1% monocytes). A clinical diagnosis of Feline Infectious Peritonitis was made and euthanasia was requested by the owner due to the poor condition of the animal. A necropsy was performed.

PATHOLOGY

At necropsy, the cat revealed typical gross lesions of Feline Infectious Peritonitis in the liver, spleen and mesenteries, which will not be described further because they are not the purpose of this report.

The liver was moderately reduced in size and weight and covered with abundant fibrous tissue which caused adhesions between lobes and the gallbladder. The external surface of the liver showed a fine nodularity, the nodules measuring from less than 1 mm up to 3 mm in diameter and a diffuse yellow-green discolouration. The organ was very firm and fibrous.

The cut surface had the same fine nodularity and the yellow-green discolouration previously described. The nodules were clearly delimited by fibrous tissue, protruding and were yellow-tan in colour. The intrahepatic bile ducts were thickened, with fibrous walls; their lumina contained inspissated bile and numerous parasites. The gallbladder was enlarged (Fig. 1).

The choledocus duct, and the wall of the gallbladder, were thickened and fibrous. They contained greenish, viscous, bile and numerous parasites which clogged the duct. The parasites were identified as Platynosomum

fastosum by parasitological studies.

Microscopically, the liver had a very distinct lobular pattern due to increased fibrous connective tissue in the portal areas and around the nepatic lobules. The fibrosis caused distortion of the shape of hepatic lobules and atrophy and fatty degeneration of the hepatic cells. Bile canaliculi were dilated and bile pigment lacunae were observed in their lumina. A slight, focal infiltration of lymphocytes and plasma cells was seen, mainly at the portal areas (Figs. 2 and 3).

Intrahepatic bile ducts had thickened fibrous walls and adenomatous hyperplasia of the epithelium which occasionally formed finger-like projections; there was also moderate infiltration of mononuclear cells. At he portal areas there was an increase in the number of cholangioles. Sections of parasites, morphologically compatible with Platynosomum fastosum, were observed in the lumen of bile ducts. (Fig. 4).

DISCUSSION

Hepatic cirrhosis is a chronic inflammation of the liver characterized by fibrosis, degeneration, necrosis and regeneration of hepatocytes and infiltration of mononuclear cells[7, 15]. Biliary cirrhosis is a type of liver cirrhosis related to obstruction of intrahepatic or extrahepatic bile ducts caused by congenital duct aplasia, chronic inflammation, cholelithiasis, pressure of tumours or parasitic infections.

Infection of cats by the liver fluke Platynosomum fastosum is common and widespread in tropical areas and has been reported from several countries[1, 2, 4, 5, 6, 8, 9, 10, 11]. The lesions reported were confined to the bile ducts and consisted of adenomatous hyperplasia of biliary epithelium, fibrous thickening and infiltration of mononuclear cells and eosinophils around parasitized ducts[12, 13, 16].

Liver fibrosis of cats infected by Platynosomum fastosum has been reported by Hitt[5] and by Powell[11]. The latter observed a nodular appearance of the liver which revealed, histologically, a marked hepatic cirrhosis but he did not classify the type of cirrhosis observed, though presumably it was a biliary cirrhosis, according to the pathogenesis of parasitic infection of the bile ducts. Othe researchers have found cholangiocellular carcinomas in parasitized livers[2].

Parasitic infections of the liver can cause biliary cirrhosis due to a hindrance of the bile flow by obstruction of the bile ducts, either by chronic inflammation or physical blockage by parasites. Both pathogenetic mechanisms were present in our case. Opistorchis pseudofelineus, Metorchis complexus[3], and Amphimerus pseudofelineus[14] have also been reported as responsible for biliary cirrhosis in cats.

The liver lesions observed by us satisfy the criteria for biliary cirrhosis due to biliary obstruction by a severe, chronic, Platynosomum fastosum infection. The laboratory findings were related to the Feline Infectious Peritonitis, which was also present. The abdominal fluid was an inflammatory exudate rather than an oedema caused by circulatory failure of the portal system.

The relationship between the fluke infection and the Feline Infectious Peritonitis, both present in the same animal, is unknown. It has been observed in 2 out of 5 cases of Platynosomum fastosum infections studied by us, which may suggest a predisposing relationship.

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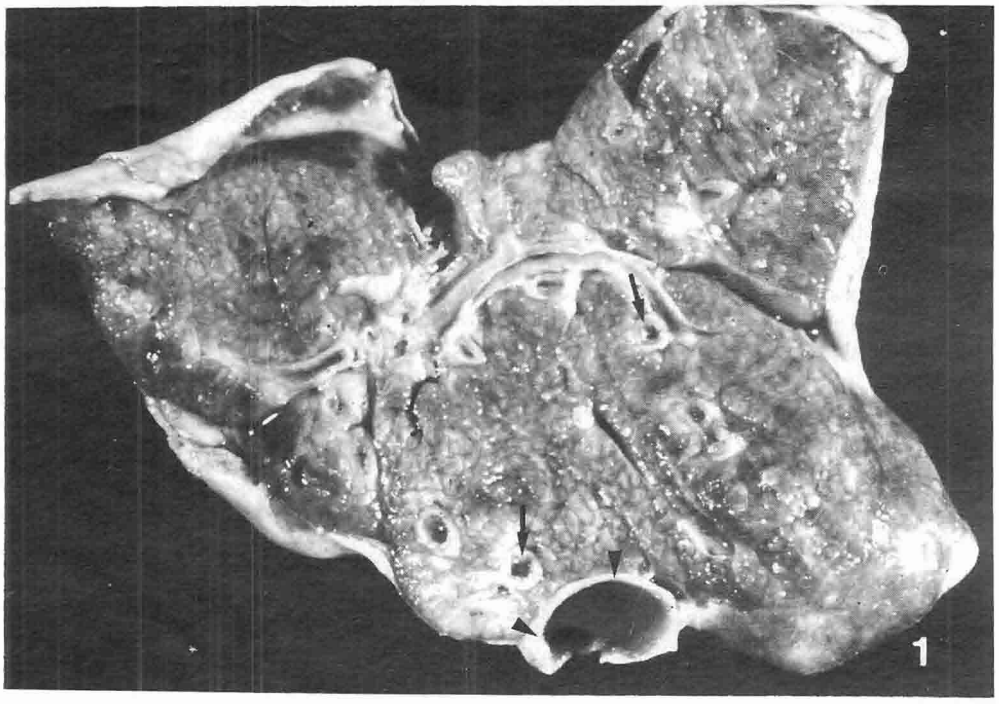

FIG. 1. Cut-surface of liver showing a fine nodularity of the parenchyma and dilated, thickened, cholangioles (arrows) and gallbladder (arrowheads).

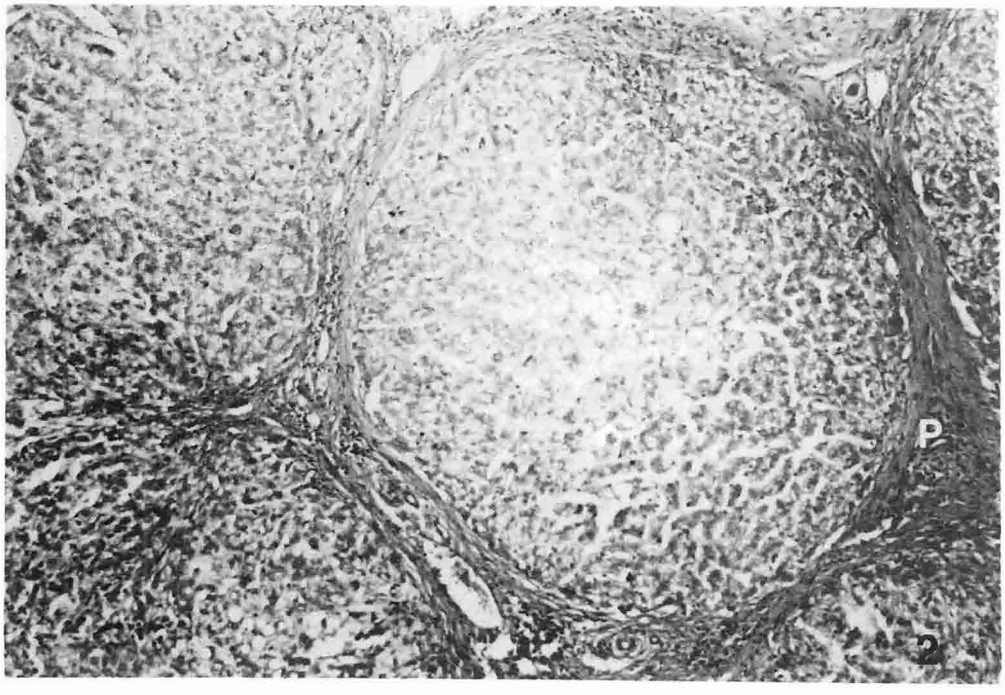

FIG. 2 Increased fibrous tickening of portal areas (P) and around hepatic lobules. Haematoxilin and Eosin, x 115.

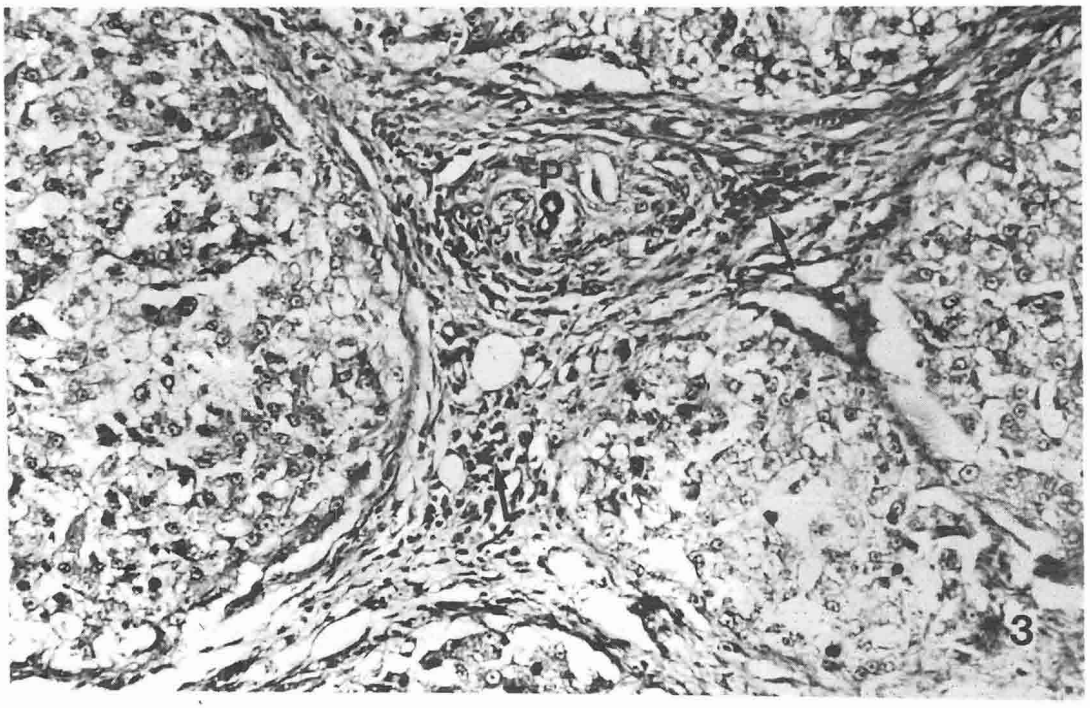

FIG. 3. Portal area (P) showing fibrous thickening and a slight infiltration of mononuclear cells (arrow). Hepatocytes are disorganized and show vacuolar changes. Haematoxilin and Eosin, x 290.

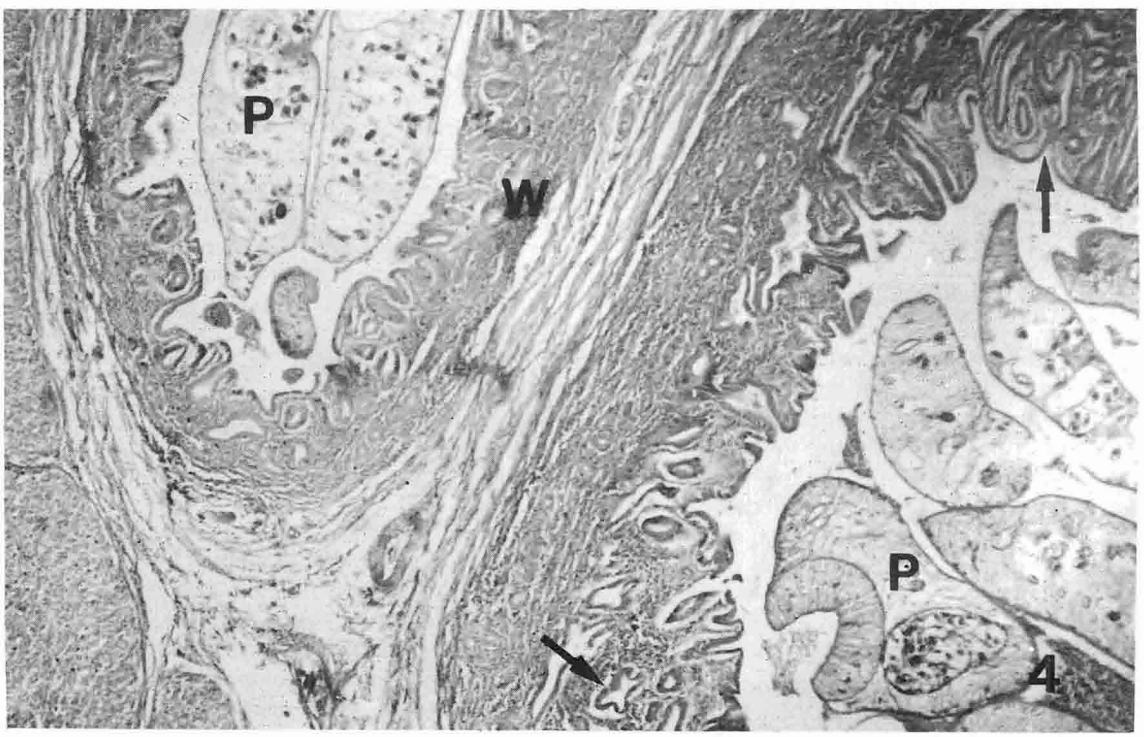

FIG. 4. Numerous parasites (P) in intrahepatic bile ducts which show adenomatous hyperplasia of the epithelium (arrows) and tickened walls (W). Haematoxilin and Eosin, x 46.