



Case Report

Polycystic Liver Disease Associated with *Platynosomum fastosum* Infection in a Cat

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Abstract

A 10-year-old cat was admitted to the Jardim da Saúde Veterinary Center with a history of anorexia, prostration and jaundice. On physical examination, it was observed an abdominal enlargement. The ultrasound revealed polycystic liver disease and biliary duct obstruction. During cholecistoduodenostomy, several cystic structures were observed within the liver. Bile cytology was performed revealing the presence of *Platynosomum fastosum* eggs. Findings were consistent with *Platynosomum fastosum* infection associated with polycystic liver disease. Although uncommonly mentioned, infection by *Platynosomum fastosum* should be placed as a differential diagnosis in polycystic liver disease in cats, always taking into account the geographic location and the hunting habits of the cat.

Key Words: liver fluke, *Platynosomum sp.*, polycystic disease, cat.

Introduction

Platynosomum fastosum (*Dicrocoelidae* family), also known as (syn.) *Dicrocoelium concinum*, *Platynosomum concinum* and *Platynosomum plancipidus*, is a feline liver fluke, commonly associated with cholangitis and cholangiohepatitis, seen in both domestic and wild cats (4, 6, 12). It has been reported in many countries, such as Bahamas, Nigeria, Puerto Rico, Malaysia and Brazil. In the latter, it has been already reported in regions of São Paulo, Rio de Janeiro, Bahia, Amazonas and Minas Gerais. The prevalence in Brazil ranges from 1.91% to 45% according to the location (4, 6, 12, 14). In the USA (Hawaii and Florida), its prevalence reaches up to 70%, where it is referred to as “lizard poisoning” (2, 8, 13).

P. fastosum is the most common liver parasite of domestic cats and it is usually located in the biliary ducts

and gallbladder (4, 6). Moreover, it is the most common genus identified in subtropical and tropical climates worldwide. The parasite lifecycle includes three intermediate hosts; the primary intermediate host, the snail *Sublima octona*, ingests the eggs taken from the environment, which form the sporocyst containing cercariae; these are subsequently eliminated to the environment. The second intermediate host, usually arthropods, such as beetles, ingest the sporocyst containing cercariae, leading to the generation of metacercariae. The isopoda is ingested by the third intermediate host, usually a lizard, gecko or toad, with the metacercariae forming cysts in the gallbladder and biliary ducts of these animals (5, 11, 14). Cats acquire the parasite by ingesting intermediate hosts. The metacercariae then migrate from the intestine to the liver via biliary ducts, becoming patent adults in 8 to 10 weeks time. Eggs can then be found in the feces and, most consistently, in biliary aspirates (13).

The severity of the associated disease seems to be influenced by the parasite load, time of infection, severity of infestation and individual responses (10, 13). Clinical findings can vary, from an asymptomatic cat to hepatic and post hepatic jaundice, depression, anorexia, vomiting, diarrhea, weight loss, hepatomegaly, abdominal distention, gallbladder and biliary duct distention, ascites and hepatic failure (4, 6, 11, 13, 14). Laboratorial findings include high levels of liver enzymes (high concentrations of ALP, AST, ALT and GGT) and hyperbilirubinaemia. Ultrasonography generally reveals typical changes of biliary tract disease, such as tortuosity and dilation of the bile ducts and distended gallbladder (10, 13).

The parasite is usually located in the biliary ducts and gallbladder associated with cholangitis and cholangiohepatitis; however, it is rarely reported associated with polycystic liver disease. Here, we report the clinical, diagnostic and histopathological features, treatment and outcome of a cat with polycystic liver disease associated with *Platynosomum* spp. infection.

Case report

A 3kg, 10-year-old neutered male domestic shorthair cat was admitted to the Jardim da Saúde Veterinary Center (São Paulo State, São Paulo – Brazil) with a history of anorexia, inappetence, weight loss and marked jaundice. The animal progressively developed jaundice and increase of abdominal size for one month before admission. It was an indoor cat with restricted outdoor access and exclusively fed with commercially cat dry food; according to the owner, the cat was frequently engaged in hunting geckos.

On clinical examination, body temperature, heart and respiratory rates were within normal reference range. The cat was found to be dehydrated (7% estimated

dehydration), lethargic and profoundly icteric. An enlarged abdomen was noticed on palpation, accompanied by the presence of hepatomegaly and tubular structures which could not be differentiated by palpation. The body condition score was considered to be 3/9.

Routine hematology was performed, revealing a complete blood count (CBC) with low packed cell volume (24%; reference range 25 – 45%); white blood cell count showed lymphopenia. Serum biochemistry findings included an increased activity of the liver enzymes alanine aminotransferase (ALT: 180UI/L; reference range 10 – 88 UI/L), alkaline phosphatase (ALP: 298UI/L; reference range 10 – 80UI/L), gamma-glutamyl transferase (GGT: 11; reference range until 5UI/L) and aspartate aminotransferase (AST: 125UI/L; reference range 10 – 88UI/L). Bilirubin levels were above normal reference range (total bilirubin 15.6mg/dL; reference range 0.1 – 0.7 mg/dL), serum protein levels were 7.2 g/dL (reference range 5.4 – 7.8 mg/dL), and albumin levels were low (2.0g/dL; reference range 2.1 – 3.3 g/dL).

Abdominal ultrasound showed a marked increase in liver size, despite its regular boundaries. Furthermore, numerous cystic structures in the liver were observed which were very tortuous and diffusely spread within the parenchyma; these lesions suggested severe intrahepatic biliary duct dilation (Figure 1A). The gallbladder had thickened walls, echogenic content and sludging of bile (Figure 1B).

With a possible diagnosis of biliary duct obstruction, the cat was rehydrated and stabilized for an exploratory laparotomy. During this procedure, hepatomegaly with numerous cystic structures was confirmed (Figure 2A). Biliary ducts were dilated, as well as the common biliary duct. The biliary content which was collected for analysis was discolored (white bile syndrome).

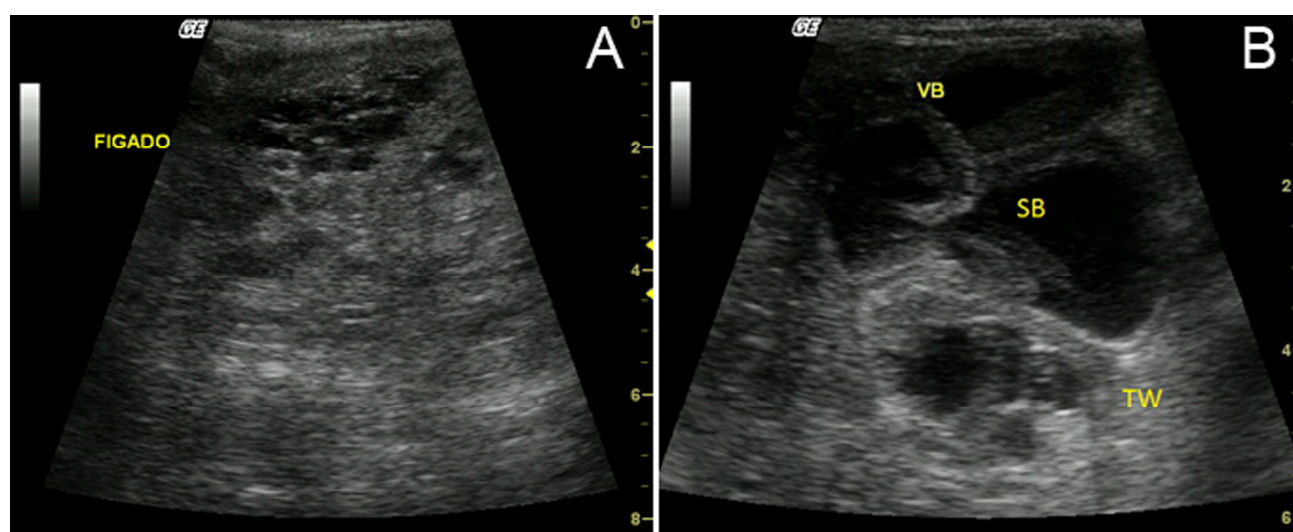


Figure 1. Chronic cholangitis associated with infestation by *Platynosomum fastosum* in a domestic cat. (A) Ultrasound image of the cat liver with multiple cystic structures. (B) Ultrasound image of the gallbladder (VB) with thickened walls (TW), dilation and sludging of bile (SB).

Cholecystoduodenostomy and hepatic biopsy were performed. During the latter, it was possible to observe that the cystic structures were cavitory (Figure 2B). Moreover, a yellow-colored, and 0.8 cm long adult fluke emerged from the liver parenchyma (Figure 2C). Bile analysis revealed 100 – 300 mg/dL of protein, presence of *Platynosomum fastosum* eggs (Figure 2D) and live parasites (Figure 2E). Bile culture was negative for bacteria. Further analysis of the liver showed the presence of several cystic structures formed by the dilation of large

bile ducts, occasionally filled with an amorphous substance and mucin. These ducts showed intense periductal fibrosis, lymphoplasmacytic inflammatory infiltrate with moderate amounts of eosinophils. In the liver tissue adjacent to cystic lesions, it was found congestion, degeneration of hepatocytes and sinusoidal dilation, predominantly in the centrilobular regions. The portal spaces were marked by the presence of periductal inflammatory infiltrate, with various regions of destruction and loss of bile ducts (Figure 3).

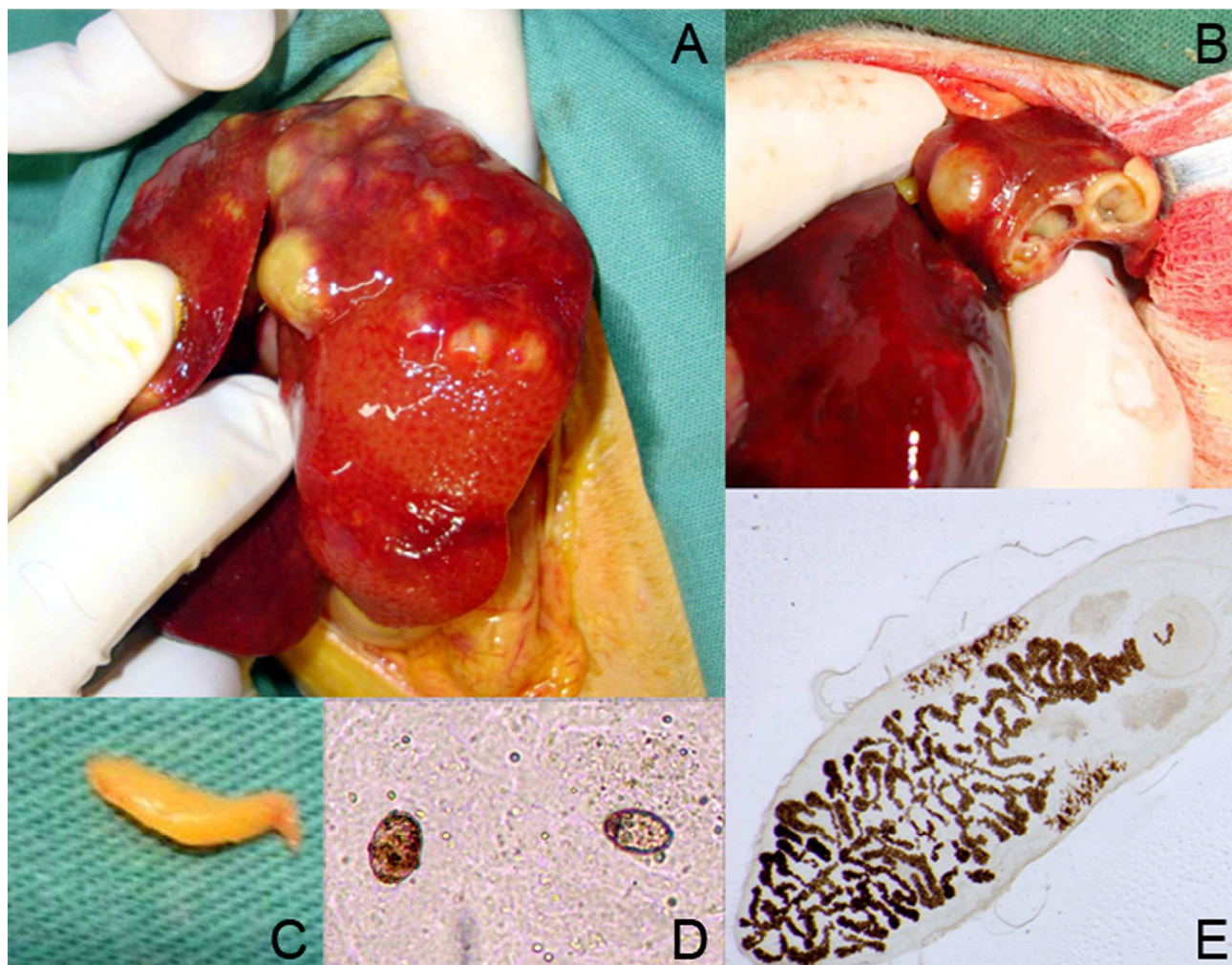


Figure 2. Chronic cholangitis associated with infestation by *Platynosomum fastosum* in a domestic cat. (A) Hepatic cystic structures observed during exploratory laparotomy. (B) Cavitory liver structures observed during liver biopsy. (C) Adult fluke obtained during hepatic biopsy. (D) *Platynosomum fastosum* eggs in bile cytology (100 X original). (E) *Platynosomum fastosum* in bile cytology.

The cat was treated with amoxicillin associated with clavulanic acid (12,5mg/kg twice a day, for ten days), silymarin (15mg/kg twice a day, for thirty days), tramadol (three days, 2mg/kg twice a day) and praziquantel (20mg/kg once a day, for seven days). It recovered well from surgery, with no complications. Chronic treatment with silymarin and S-Adenosyl methionine was then prescribed, since the damage caused by the parasites

(observed during histopathological exam) seemed to be irreversible. Thirty days after the surgery, the cat showed signs of hepatic insufficiency, ascites and symptoms compatible with hepatic encephalopathy (ptyalism, depression). The cat was euthanized by owner request. Necropsy was not performed (the owner did not give permission to perform a necropsy).

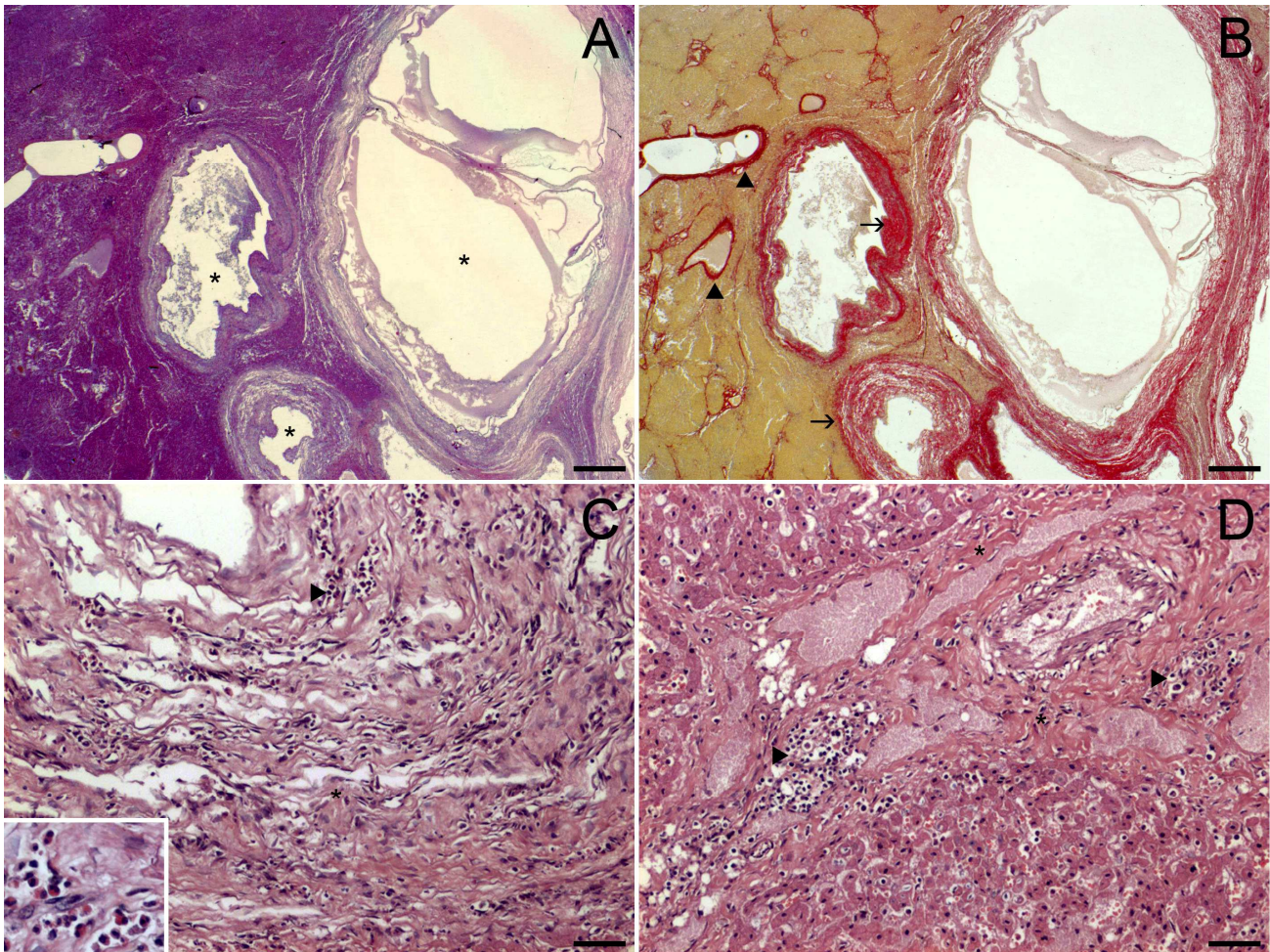


Figure 3. Chronic cholangitis associated with infestation by *Platynosomum fastosum* in a domestic cat. (A) presence of cystic structures (*) in liver tissue, formed by the dilation of large bile ducts (HE stain; scale bar = 200 µm); (B) Intense deposition of collagen fibers (red) around the bile ducts (arrows) and vascular spaces (arrowheads) (Picrosirius stain; scale bar = 200 µm); (C) Marked lymphoplasmocytic inflammatory infiltrate (arrowheads) around the ducts, with some eosinophils (inset) (HE stain; scale bar = 20 µm); (D) Portal area, located in the adjacent liver tissue, demonstrating venous congestion, fibrosis (*) and inflammation / destruction of bile ducts (arrowheads) (HE stain; scale bar = 20 µm).

Discussion

This report describes a case of a *Platynosomum fastosum* infection associated with polycystic liver disease leading to hepatic failure in a cat. Considering the clinical signs and history and taking into account the available peer reviewed literature on this matter, congenital polycystic disease was initially discarded; then, neoplastic (mainly biliary carcinoma), traumatic and inflammatory causes were the most important differential diagnosis. The symptoms and characteristics of ultrasonography, associated with geographical features and especially the cat's hunting habit led us to consider *Platynosomum* spp infection as the most probable diagnosis. However, definitive diagnosis was only obtained by bile cytology and liver histopathology.

P. fastosum is the most common liver parasite of domestic cats and it is usually located in the biliary ducts and gallbladder; however, it is rarely reported to be

associated with polycystic liver disease (6, 14). Most cystic lesions in the feline liver are of bile duct origin, derived from ductular epithelium, being either congenital or acquired (3, 13). Congenital disease is usually polycystic and often present as a part of a polycystic disease of several organs, including kidneys. Persian and Persian crosses are at increased risk. The acquired disease is commonly solitary, with blood or bilirubin cyst content, and it can be induced by trauma, inflammation or neoplasia (3). In this case, what probably happened was a high number of *Platynosomum fastosum* producing a chronic inflammation and resulting in an acquired polycystic liver disease.

The diagnosis of platynosomiasis can be made based on history, clinical findings, detecting eggs on stool or bile, and liver histopathology (5, 13). The fecal examination is a specific, but not extremely sensitive test, because egg production is limited in number (8). The formalin-ether centrifugation method is the most effective

procedure for coprologic diagnosis (1, 2). The diagnosis is supported by ultrasonographic findings of bile duct enlargement and tortuosity. However, other causes of cholangitis (lymphocytic or neutrophilic cholangitis) can also result in similar findings (11). In this case we found eggs of *P. fastosum* in bile content as well as live parasites inside multiple cystic structures. Additionally, the histopathological findings were similar to describe in current literature (4, 6, 14), including diffuse portal congestion, hepatocyte degeneration, hepatic sinusoid dilation, polymorphonuclear inflammatory infiltrate and great collection of mononuclear cells around biliary ducts, as well as hyperplasia, increase in number of biliary ducts and proliferation of periductal connective tissues.

Ultrasonography generally reveals typical changes of biliary tract disease, such as tortuosity and dilation of the bile ducts and distended gallbladder (10, 13). However, these alterations are rarely reported to be associated with fluke infestation acquired polycystic disease of the biliary system, which makes this report quite unique. To our knowledge, there are few reports of cystic liver disease associated with severe infestation by *Platynosomum fastosum* in the literature, two of them in Brazil (6, 7, 9, 14). Concerning treatment efficacy, protocols remain still controversial. Praziquantel (20 – 30mg/kg PO q 24h for 5 days) is the most commonly used, being reported to be more efficient than Fenbendazole; which is recommended to be used at 50mg/kg PO q 12h for 5 days (8). However, it lacks controlled studies with naturally infected cats submitted to either treatment.

In this case, the chronic infection associated with bile duct fibrosis and subsequent intra and extra hepatic biliary obstruction resulted in the progressive destruction

of liver parenchyma, leading to liver failure. Intrahepatic ducts obstructions progressed to the multiple cystic formations and chronic fibrosis of bile ducts were likely to be the cause of extra hepatic biliary duct obstruction. Ferreira (4) and Salomão (10) mention a fluke induced obstruction of bile ducts, especially in cases where adult flukes are found

Due to the chronicity of the process, severe destruction of hepatic parenchyma and inefficient response to treatment, together with the poor condition of the animal, euthanasia was performed. Although not always mentioned, *P. fastosum* must be included among the differential diagnosis of cats with signs of hepatobiliary disease. Infection by *Platynosomum* spp also should be placed as a differential diagnosis in acquired polycystic liver disease in cats, always taking into account the geographic location and the hunting habits of the animal.

Conclusion

This report describes a case of a *Platynosomum fastosum* infection associated with polycystic liver disease leading to hepatic failure in a cat. Considering the clinical signs and history and taking into account the available peer reviewed literature on this matter, congenital polycystic disease was initially discarded; then, neoplastic (mainly biliary carcinoma), traumatic and inflammatory causes were the most important differential diagnosis. The symptoms and characteristics of ultrasonography, associated with geographical features and especially the cat's hunting habit led us to consider *Platynosomum* spp. infection as the most probable diagnosis. However, definitive diagnosis was only obtained by bile cytology and liver histopathology.

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