RESEARCH NOTE

FELINE CHOLANGITIS/CHOLANGIOHEPATITIS COMPLEX SECONDARY TO *Platynosomum fastosum* INFECTION IN A CAT

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ABSTRACT:- CARREIRA, V.S.; VIEIRA, R.F.C.; MACHADO, G.F.; LUVIZOTTO, M.C.R. **Feline cholangitis/chonlangiohepatitis complex secondary to *Platynosomum fastosum* infection in a cat.** [Complexo colangite/colangiohepatite felino secundário a parasitose por *Platynosomum fastosum* em um gato]. *Revista Brasileira de Parasitologia Veterinária*, v.17 supl. 1, p. 184-187, 2008. Patologia Veterinária, Universidade Estadual Paulista "Júlio de Mesquita Filho", FOA/Setor de Patologia Veterinária, Hospital Veterinário, Campus Araçatuba, Rua Clóvis Pestana, 793, Dona Amélia, Araçatuba, SP 16050-680, Brasil. E-mail: ruimcl@fmva.unesp.br

Platynosomiasis has been associated with cholangitis/cholangiohepatitis complex in domestic cats. Despite being typically asymptomatic, some individuals may develop severe disease resulting in hepatic cirrhosis. Definitive diagnosis by means of parasite eggs direct visualization is not always possible and complementary tests should be associated. This paper reports a case of a three-year-old female cat with anorexia and jaundice. Ultrasound and biochemistry laboratory findings as well as liver biopsy were not conclusive. Definitive diagnosis of intense *Platynosomum fastosum* infestation was only possible through direct examination of biliary fluid at necropsy, stressing the importance of including platynosomiasis, commonly an underdiagnosed disease, among differential diagnoses of feline liver diseases.

KEY WORDS: Platinosomiasis, *Platynosomum fastosum*, necropsy, feline.

RESUMO

A platinossomíase figura entre os agentes etiológicos envolvidos no complexo colangite/colangiohepatite dos felinos. Apesar de freqüentemente cursar como infestação assintomática, alguns indivíduos desenvolvem quadros severos culminando em cirrose hepática. O diagnóstico definitivo por visualização dos ovos nas fezes nem sempre é possível e exames complementares devem ser associados. O presente trabalho refere um caso de um felino, fêmea, três anos com quadro clínico de anorexia e icterícia. Os achados de ultrassonografia, laboratoriais e biópsia foram inconclusivos. O diagnóstico definitivo de infestação por *Platynosomum fastosum* foi realizado somente à necrópsia por exame direto da bile, salientando a importância da inclusão desta entidade comumente subestimada no diferencial das doenças hepáticas em felinos.

PALAVRAS-CHAVE: Platinossomíase, *Platynossomum fastosum*, necropsia, felinos.

Cholangitis/cholangiohepatitis (CC/CH) complex is characterized by bile ducts and hepatic parenchyma inflammation and is a frequent cause of hepatic disease in felines (DAY, 1995; GAGNE et al., 1996; WEISS et al., 2001; ILHA et al., 2004). The clinical signs seen in affected animals are consistent with hepatic failure: jaundice, cachexia, firm nodular liver, and ascitis. Trematodes are associated with CC/CH (DAY, 1995; XAVIER et al., 2007). Specifically, biliary ducts infection by *Platynosomum fastosum* in felines is considered one of the causes of biliary obstruction and nonsuppurative CC/CH (CENTER, 1996) as well as cystic hepatic disease (XAVIER, 2007).

Platynosomum fastosum eggs are eliminated with the feces of infected cats (definitive host). In the environment they are

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ingested by the snail *Sublima octona* (primary intermediate host), releasing miracidia inside the eggs (FERREIRA; ALMEIDA, 2003). Approximately 28 days after ingestion, miracidia turn into sporocyst I, sporocyst II and then migrate to the ground through the mollusks' respiratory pore. In the environment, 30 days later, maturation of sporocyst II into cercaria occurs, and cercaria will be ingested by arthropods (intermediate host II) such as beetles and turn into metacercariae. Infected beetles are then ingested by lizards and frogs (intermediate host III) and metacercarial cysts are formed in the bladder and biliary ducts of these animals. When an infected intermediate host III is ingested by cats, metacercariae migrate to biliary ducts and bladder of these animals, reaching the maturity between eight to 12 days and are then eliminated in feces as fertilized eggs, closing the cycle (SOULSBY, 1982; CENTER, 1996; FERREIRA; ALMEIDA, 2003). *Platynosomum fastosum* infection usually causes mild or none clinical signs, varying according to the severity of infestation, number of adult parasites, time of infection, and individual reaction to the parasite (SALOMÃO et al., 2005). Severe clinical presentation of Platynosomiasis with secondary hepatic encephalopathy has been described (PIMENTEL, 2005). The diagnosis of platynosomiasis is based on history and clinical findings and the definitive diagnosis can be made by the presence of eggs in fecal examination for parasites (FERREIRA; ALMEIDA, 2003). The centrifugation formalin-ether method is the most effective one (BIELSA; GREINER, 1985). Finding parasite eggs in fecal samples is not always possible, especially in cases with complete biliary obstruction (WILLARD; FOSSUM, 2000). Complementary diagnostic imaging may be helpful, findings as dilated biliary ducts and distended gallbladder are usually related with high parasite infection (SALOMÃO et al., 2005). Biochemistry changes in hepatic enzyme profile are only suggestive of disease. Histopathological findings include: diffuse portal congestion, hepatic sinusoid dilation, hepatocyte degeneration, mild centrilobular fatty change, diffuse polymorphonuclear inflammatory infiltrate, and mononuclear cell infiltrate around biliary ducts (FERREIRA et al., 1999; FERREIRA; ALMEIDA, 2003). Ductal obstruction is secondary to hyperplasia of the biliary duct epithelium, periductal inflammation, and fibrosis (FERREIRA et al., 1999).

For ILHA et al. (2004), the cause-effect correlation between biliary extra-hepatic obstruction and CC/CH has not yet been totally accepted in feline medicine. CC/CH therapy of affected patients is determined according to the stage of disease. This disease prognosis is reserved, mostly because it is usually diagnosed at advanced stages (DAY, 1995). *Platynosomum fastosum* regional prevalence data, although limited, should be taken into consideration and platynosomiasis should be considered among differential diagnosis of jaundice, hepatitis, and cirrhosis in feline local population (ILHA et al., 2004).

The purpose of this study was to describe a case of *P. fastosum* infection in a cat.

A 3-year-old female spayed domestic shorthair feline patient arrived at a private practice clinic presenting signs of apathy, anorexia, and jaundice. The physical examination revealed hepatomegaly and jaundice. The ultrasound exam revealed a 4-cm diameter hypoechogenic cystic structure, suggestive of hepatic cyst or neoplastic nodule. Clinical laboratory findings included anemia (packed cell volume, 17%), leukocytosis with neutrophilia, alkaline phosphatase within normal values (ALP: 108 U/L), creatinine (0.6 mg/dL) and increased serum alanine transaminase (ALT: 820 U/L). After therapy with doxycycline, Legalon®, Plasil® and gastric tube feeding, the animal showed relative improvement. Despite treatment, the animal had an unfavorable clinical progress and the referring veterinarian performed an ultrasound-guided biopsy of the liver. Due to a histopathological diagnosis of inflammation, clinical and nutritional support was maintained. Progressive clinical worsening was seen. After a month of therapy without any satisfactory results the owner elected euthanasia. The necropsy showed advanced cachexia and marked jaundice of mucosa. Natural body cavities presented ascitis and hydrothorax. Other relevant findings included peripancreatic steatonecrosis, liver natural surface irregularities with yellow to orange irregular areas (Figure 1-A), firm with evident lobular pattern, and a distended gallbladder full of inspissated content (Figure 1-B). Liver sections showed macroscopic structures suggestive of *P. fastosum* associated with inspissated biliary fluid. The light optic microscopy confirmed these structures were parasites. Relevant histopathological findings were as follows: biliary hyperplasia amid adult parasites (Figure 1-C), periportal fibrosis (Figure 1-D), hemosiderin casts, biliary stasis, and diffuse mixed inflammatory infiltrate with periductal lymphoplasmocytic inflammatory infiltrate (Figure 1-E). Microscopic findings were consistent with chronic cholangiohepatitis secondary to *P. fastosum*.

Platynosomum fastosum infection is usually asymptomatic and animals harbor parasites without any important clinical abnormalities. Host immune status determines the severity of parasitic infection and the time for the animal to develop chronic liver inflammation leading to cholangitis/cholangiohepatitis and invariably biliary obstruction with related clinical consequences. Fecal examination for diagnosis has low sensitivity due low occurrence of eggs in feces, and it is of poor value in severe cases with biliary obstruction as eggs do not reach the small intestine. Ultrasound and laboratory findings such as increase of GGT, ALP and serum ALT, associated with direct bilirubin increase and jaundice may be associated to the extent of parasite infestation. In routine diagnostic experience, felines with platynosomiasis are frequently suspect to have hemoparasitosis (mainly by *Mycoplasma haemofelis*), hepatic lipidosis or hepatic neoplasia. Data on the prevalence of platynosomiasis in Brazil is scarce. The parasite cycle is complex and includes three intermediate hosts, but it must be considered as a differential diagnosis of feline jaundice.

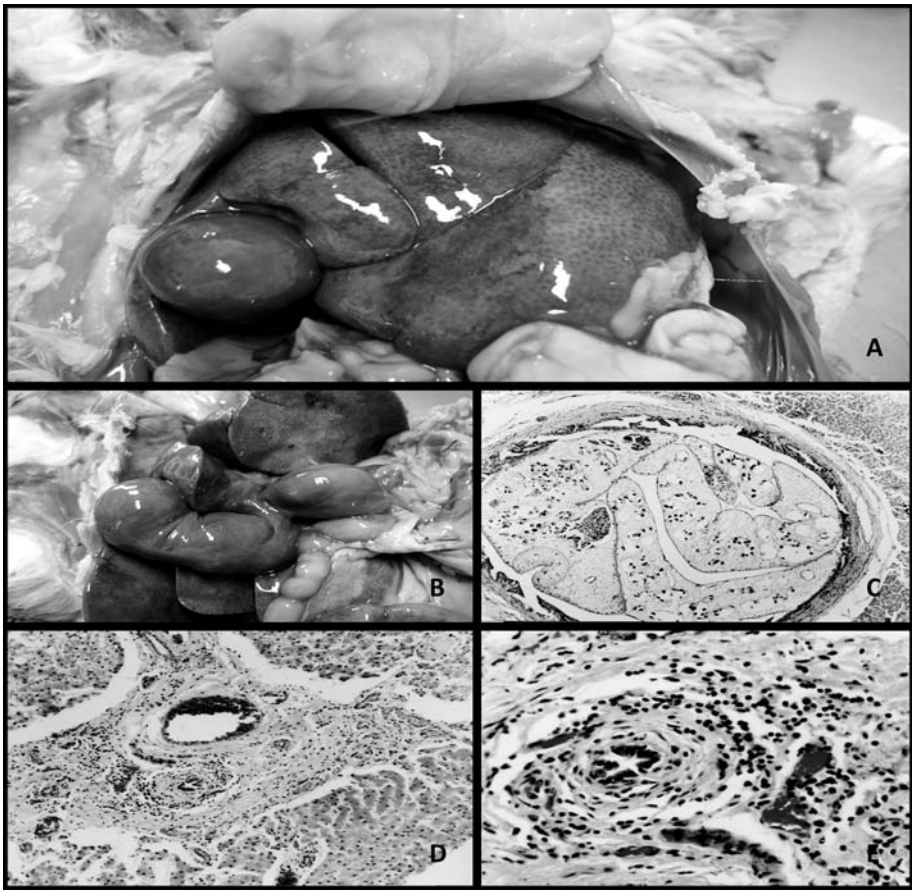

Figure 1A: Liver exhibiting accentuated hepatomegaly within round boundaries, accentuated increased of consistency, capsular thickening with irregular surface. Distended gallbladder full of bile; B: Liver visceral side evidencing a distended gallbladder and cystic duct; C: *Platynosomum fastosum* inside the biliary duct, accentuated proliferation of epithelium with associated fibrosis (H.E., Obj.10x); D: Extensive periportal fibrosis (H.E., Obj.20x); E: Prominent periductal inflammation, predominantly mononuclear infiltrate, and periductal fibrosis associated (H.E., Obj.40x).

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