

CYSTIC LIVER DISEASE IN DOMESTIC FELINE INFECTED WITH *Platynosomum* sp. - A CASE REPORT

DOENÇA HEPÁTICA CÍSTICA EM FELINO DOMÉSTICO INFECTADO POR *Platynosomum* sp. – RELATO DE CASO

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ABSTRACT: Platinosomosis affects felines and it is caused by a trematode of the genus *Platynosomum* sp. The parasitized animals can be symptomatic or asymptomatic and the disease can evolve severely, killing the animal. Due to the similarity between clinical signs and changes of platinosomosis and those of other pathologies caused by the parasite and having in mind that the parasite occasionally appears on the stool, the search for trematodes and eggs in material coming from bile aspirate is a more efficient way to diagnose the parasitic disease, what helps to distinguish it from other liver diseases. Thus, the clinical veterinarian is able to perform the treatment correctly. The aim was to report a case of cystic liver disease related to platinosomosis in a domestic feline which has been undergoing treatment of liver disease for over a year. During the clinical evaluation, the animal was apathetic and with hepatomegaly. A complete blood count (CBC) revealed a discreet lymphopenia. The enzymes alkaline phosphatase and alanine transaminase were above the normal range for the species. The parasitological examination of the biliary material indicated eggs from the trematode *Platynosomum* sp. whereas the histopathology examination of liver masses indicated cystic structures covered by connective tissue and multifocal mononuclear inflammatory infiltrate. In conclusion, liver cysts in felines with chronic liver disease can be associated with cases of diseases caused by the trematode helminths.

KEYWORDS: Biopsy. Cysts. Liver.

INTRODUCTION

Scientific studies suggest that species such as *Platynosomum fastosum*, *P. concinnum* and *P. illiciens* which infect feline bile ducts can be synonyms (SALOMÃO et al., 2005).

Several information about helminths *Platynosomum* sp. life cycle is still unknown (PINTO; MATI; MELO, 2014), but it is known that requires three intermediate hosts. The first is a land snail (*Subulina octona*), the second can be terrestrial isopods, a beetle or other small bug and finally, the last are lizards (*Anolis cristatellus*, *A. equestris*, *A. sagrei*, *A. carolinensis*) or toads (*Bufo marinus*, *B. terrestris*) as obligatory or paraenic hosts. Insect-eating birds can also be involved in the fluke life cycle. Felidae is the definitive host (FERREIRA; ALMEIDA, 2003). Felines becomes infected when they eat the third vertebrate intermediate host which carries metacercariae (SEETHA; CHENG, 1997). However, Pinto, Mati and Melo (2014) found out that isopods can be involved in the direct transmission of the parasite to felines.

The eggs containing miracidia of the parasite which are eliminated with the faeces of the felines to the environment and ingested by a land

snail, the sporocysts containing cercariae are released into the environment and ingested by terrestrial isopods. After the maturation of cercariae to metacercariae, occurs the ingestion of isopods by lizards, where metacercariae are encysted in the gallbladder and common bile duct of these vertebrates, waiting for the definitive host complete the biological cycle. When the lizard is ingested by the feline the metacercariae are released and migrated to the bile duct where they become mature adults (FERREIRA; ALMEIDA, 2003).

According to Bowman (2003), in the infection caused *Platynosomum concinnum*, the prepatent period lasts for 2 or 3 months, and the cat's stool still carries eggs for 1.5 year after the initial exposure.

The organs affected by the infection are the liver, the bile ducts and the gallbladder. Occasionally other organs can be infected, such as the small intestine, the pancreatic ducts and the lungs. The individual reaction to the parasite, the severity of injuries and the time of infection are needed to be considered to differentiating symptomatic from asymptomatic infection in felines (FOLEY, 1994; PIMENTEL et al., 2005). However, infection of high parasite load can be associated

with severe and often fatal diseases (XAVIER et al., 2007).

Biliary tract disorders, as marked fibrosis, cholangitis, cholangiohepatitis and even extrahepatic bile duct obstruction can occur (SALOMÃO et al., 2005). Zen et al. (2005) described a predisposition to tumors development in human beings with trematodes into the bile duct.

Regarding epidemiology, *Platynosomum* sp. is found at Malaysia, Papua New Guinea, Australia, Hawaii, Bahamas, Puerto Rico, Cuba, Brazil, British Guiana and Florida (BARRIGA; CAPUTO; WEISBRODE, 1981). In Brazil, some reports about the presence of the parasite in the states of Pernambuco (FERNANDES; TRAVASSOS, 1976), São Paulo (OGASSAWARA; BENASSI; LARSSON, 1980) and Mato Grosso do Sul (ASSIS; FREIRE; RIBEIRO, 2005). According to Mundim et al. (2004), the parasite *Platynosomum fastosum* (40%) and the nematodes *Ancylostoma braziliense* (38%) and *Physaloptera praeputialis* (34%) prevailed among cats in Uberlândia, Minas Gerais.

The diagnosis includes information on cat's hunting for lizards behavior, the search for trematodes or their eggs in the animal stool or bile, and the search for changes on marker enzymes of liver diseases and hyperbilirubinemia. Fluke eggs on the stool, however, is a sporadic event, and eggs might even not appear if the infection results in a complete biliary obstruction. This way, the search for trematodes and eggs in bile aspirate is a more efficient method for diagnosing the animal (NELSON; COUTO, 2010).

As the stool test is not the most efficient method for diagnosing this parasitic disease, complementary imaging tests are required to evaluate potential liver changes caused by the parasite (LEAL et al., 2011).

The present study aimed to report a case of cystic liver disease associated with platinosomosis in a domestic feline that has been assisted to treatment of liver disease for over a year.

CASE REPORT

The animal assessed was an adult mixed-breed female cat. During the medical history taking, the animal's owner reported that it was a semi-domiciled animal, which lived with three other felines, ate specific cat food, and it has been undergoing treatment for liver disease for over a year, using Ursacol and Hepatovet. Physical examination pointed apathy and hepatomegaly as the only changes, since the animal had normal body

temperature and respiratory rate, normocorated mucous and good nutritional status.

Laboratory and ultrasound exams were requested as complementary exams. A 2 ml (0.07 fl. oz.) blood sample was collected by means of cephalic venipuncture. Half of the sample was mixed into a test tube with 10% ethylenediaminetetraacetic acid (EDTA) to undergo a complete blood count (CBC), and the remaining sample was stored in a blood collection tube to undergo serum biochemistry testing.

The rates of erythron (red blood cell (RBC) count $9.54 \times 10^6/\text{mm}^3$, hematocrit 40.5% and hemoglobin 14.3 g%), platelet count ($472,000/\text{mm}^3$) and white blood cell (WBC) count ($8,500/\text{mm}^3$) are within the physiological parameters for the species (JAIN, 1986), except for the white blood cell differential count, which showed a discreet lymphopenia ($935/\text{mm}^3$).

The serum biochemistry testing showed normal rate of creatinine (1.45 mg/dL), whereas the enzymes alkaline phosphatase (ALP: 600 U/L) and alanine transaminase (ALT: 1090 U/L) rates were above the normal range for the species, according to Kaneko, Harvey and Bruss (1997).

An abdominal ultrasound examination suggested hepatomegaly: swollen liver with homogeneous, hyperechoic parenchyma and coarse echotexture. Also, there were heterogeneous, hypoechoic masses of irregular shape, located on the temporal and middle lobes and measuring around $2,98 \text{ cm}^2$ (1.17 in^2). Finally, the examination showed a distended gallbladder with well-defined contour, anechoic content and regular echoic internal wall.

In addition to the imaging and laboratory tests, an exploratory laparotomy revealed millimeter-lengthed non-ulcerated white nodules with irregular surface and firm consistency, which were spread across the lobes of liver, besides a large nodule – 2 to 3 cm^2 (0.8 to 1 in^2) in diameter – with the same characteristics in the right lobe of liver (Figure 1A). Next, by means of fine-needle biopsy, the content of gallbladder was packed into a specimen cup for later parasitological examination. The biliary material was analyzed through both direct exam and Willis-Mollay method. Both methods detected brown eggs provided with an operculum (FOLEY, 1994) which optical microscopy using 40X objective lens (Figure 1B) described as *Platynosomum* sp. eggs.

A biopsy of the large nodule was performed and a fragment of tissue was packed into a specimen cup which contained 10% formalin solution by volume twice as big as the sample for fixation. The histological section was made using hematoxylin

and eosin (HE) stain (Figure 1C), besides special staining technique by Masson's trichrome (MT) (Figure 1D), as stated by Tolosa et al. (2003). Both staining methods detected cystic structures covered by connective tissue and multifocal mononuclear

inflammatory infiltrate. Special stain colored in blue the collagen fibers around the lesion, confirming the diagnosis by means of optical microscopy with objective at 40x magnification.

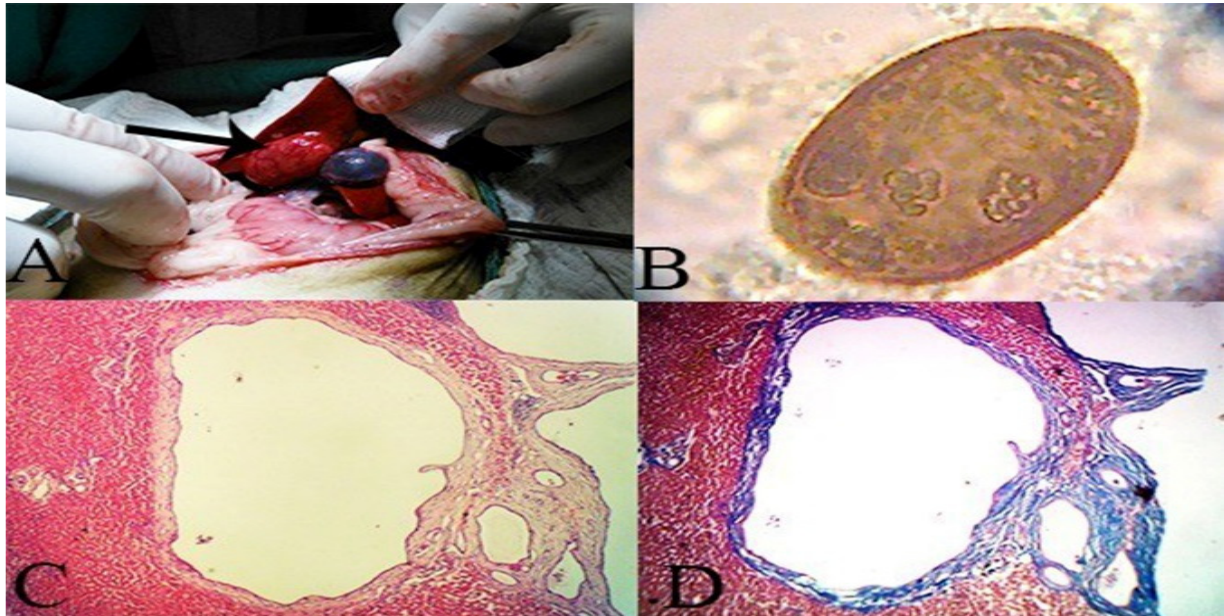


Figure 1. In (A), a 2 to 3 cm² (0.8 to 1 in²) in diameter nodule in the right lobe of liver; (B) *Platynosomum* sp. brown egg provided with a lid (operculum). Objective at 400x magnification; (C) Liver cysts covered by connective tissue and multifocal mononuclear inflammatory infiltrate (HE). Objective at 40x magnification; (D) Collagen fibers around the lesion (MT). Objective at 40x magnification. Feline, mixed-breed animal, adult, 2014.

DISCUSSION

In this report was observed a discreet lymphopenia due to the infection caused by *Platynosomum* sp., an increase of ALT (1090 U/L) and ALP (600 U/L) rates, which suggested hepatocytes lesions and impairment of biliary flow (THRALL, 2015). Hepatomegaly was verified through imaging test and cysts in the liver parenchyma through exploratory laparotomy. The results confirm the study of Xavier et al. (2007), who report laboratorial and imaging changes related to parasitic infection caused by *Platynosomum* sp. in domestic felines, such as discreet lymphopenia (230mm³) and increase on the serum concentrations of the liver enzymes ALT (892 U/L) and ALP (339 U/L).

The abdominal ultrasound showed a thin-walled gallbladder containing anechoic material. It also revealed an enlarged liver with regular boundaries and heterogeneous echogenicity in addition to cavitated lesions, which were distributed diffusely on the parenchyma, suggesting the presence of cysts or abscesses. Hepatomegaly was confirmed through the exploratory laparotomy,

according to DANIEL et al. (2012), who also found hematologic values within the normal range of the species, along with an increase of enzymes ALT (180U/L) and ALP (298U/L) rates, what suggests a case of feline platinosomosis.

As stated by Macgavin and Zachary (2010), congenital liver cysts commonly present thin wall covered by a single layer of biliary epithelium. They are filled with material of fluid and light aspect, tend to be unique and usually are accidentally detected. The authors still state that most of the choledochal cysts originate from abnormal development of intrahepatic bile ducts and can be found in animals of all ages. Thus, it is necessary to discriminate liver cysts from parasitic cysts.

Literature reports on cystic liver disease associated with severe infection caused by *Platynosomum fastosum* (ROBINSON; EHRENFORD, 1962, JEKINS et al., 1988). According to Daniel et al. (2012), the trematode usually inhabits the bile ducts and the gallbladder and is associated with cholangitis and cholangiohepatitis conditions. However, it is seldom related to polycystic liver disease.

According to Nelson and Couto (2010), congenital cysts are ordinarily multiple and often are part of polycystic diseases in various organs, including the kidneys. Congenital cystic content tends to be light, whereas acquired cyst may be small or large and its content may be light, bloody or bilious. It can be secondary to trauma, inflammation, neoplasia and hepatic cystadenomas, however it is rarely caused by trematodes. The feline showed creatinine levels within the normal range for the species (1.45 mg/dl), what makes it possible to discard congenital polycystic disease.

Considering a possible medical history of platinosomosis, Xavier et al. (2007) used exploratory laparotomy to notice a diffuse distribution of small cysts all over the liver, including two lesions of 3 cm (1 in) in diameter and one of 10 cm (4 in) in diameter. The analysis of the biliary material confirmed transudate and the presence of adult parasites which were identified as *Platynosomum fastosum*.

Histopathological exam revealed periportal fibrosis and lymphocytic inflammatory infiltrate mostly periductal, along with cystic structures of different sizes, which were found surrounded by varying amounts of fibrous tissue. Thus, to distinguish platinosomosis from other liver diseases, the clinical and anatomopathological

findings similar to those found in this report are essential: hepatomegaly, cysts of different sizes on the liver, *Platynosomum* sp. eggs in the biliary material, as well as cystic structures covered by connective tissue and multifocal mononuclear inflammatory infiltrate.

For postoperative treatment was prescribed 0,1mg/kg of non-steroidal anti-inflammatory SID during two days, 20mg/kg of amoxicillin + potassium clavulanate BID for ten days, 3mg/kg of tramadol TID for four days and 2mg/kg of ranitidine BID for ten days. After that, the animal got into a three day treatment for platinosomosis, where it were administered subcutaneous injections of praziquantel 20mg/kg, as suggested by Nelson and Couto (2010). On return to veterinary consultation the feline showed a significant improvement of his general state of health, the results of laboratory exams returned to normal and parasitological examination was negative.

CONCLUSION

The presence of liver cysts in felines may be related to the parasitism caused by trematode helminths. As a result, a different diagnostic procedure must be performed to detect the platinosomosis in cases of chronic liver disease.

RESUMO: A platinosomose afeta felinos e é ocasionada por um trematódeo do gênero *Platynosomum* sp. Os animais parasitados podem ser sintomáticos ou assintomáticos e a doença pode evoluir de forma grave levando o animal a óbito. Devido à similaridade dos sinais clínicos e alterações ocasionadas pelo parasita com outras patologias hepáticas e a liberação do parasita nas fezes ocorrer de forma esporádica, a pesquisa de trematódeos e ovos em material proveniente de aspirados de bile é uma forma de diagnóstico mais eficiente da parasitose, o que auxiliará na diferenciação de outras hepatopatias, e com isso o clínico veterinário poderá realizar o tratamento de forma correta. Dessa forma, objetivou-se relatar um caso de doença hepática cística associado a platinosomose em felino doméstico assistido para tratamento de hepatopatia há mais de um ano. Na avaliação clínica, o animal se apresentava apático e com hepatomegalia. Foi realizado hemograma onde se evidenciou uma discreta linfopenia e as enzimas fosfatase alcalina e alanina aminotransferase estavam acima dos limites de normalidade para espécie. No exame parasitológico de conteúdo biliar foi observado ovos do trematódeo *Platynosomum* sp. enquanto que no exame histopatológico de massas hepáticas foram observadas estruturas císticas recobertas por tecido conjuntivo e infiltrado inflamatório mononuclear multifocal. Conclui-se que a presença de cistos hepáticos em felinos com hepatopatia crônica pode estar associada a quadros de parasitismo por helmintos trematódeos.

PALAVRAS-CHAVES: Biópsia. Cistos. Fígado.

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