Ultrasonography in Hepatobiliary Evaluation of Domestic Cats (*Felis catus*, L., 1758) Infected by *Platynosomum* Looss, 1907

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ABSTRACT

Platynosomum-infected cats are common in tropical and sub-tropical regions of the world. The helminths elicit unspecific clinical signs that are transitory, but, with the evolution of the disease, they generally reappear with chronic mucous diarrhea or jaundice. Due to the absence of specific clinical signs, in vivo diagnosis relies solely in the detection of operculated eggs in feces. A total of 72 cats were examined by abdominal ultrasound and fecal samples and 33% were found to be infected. It was noted that although no individual sonic alteration could be associated with the infection, the frequency of associated alterations of bile ducts and gallbladder (29.1%) or liver, bile ducts, and gallbladder in infected animals (12.5%) was higher than in animals that did not eliminate eggs (20.8% and 2.1% respectively), suggesting that associated sonographic alterations should be an indicator of *Platynosomum* spp. infection.

INTRODUCTION

Although different trematode species of the families Opisthorchiidae and Dicrocoeliidae can be found in the liver of domestic cats,[1] the genus *Platynosomum* Looss, 1907 is the most commonly reported.[2] The literature cites *P. concinnum*, *P. illiciens*, and *P. fastosum* infecting the gallbladder and bile ducts of cats; however, all these species

may be synonymous.[3] According to Travassos, Freitas, and Kohn[4] in Brazil we find the species *P. illiciens* (Braun, 1901) Kossack, 1910, *P. deflectens* (Rudolphi, 1819) Nicoll, 1915, and *P. reficiens* (Braun, 1901) Travassos, 1916, but only the *P. illiciens* parasitizes the bile ducts and gallbladder of domestic cats. In this study, due to the controversy regarding nomenclature and the lack of recent taxonomic studies, we opted for calling the parasites *Platynosomum* spp.

Cats infected with this helminth can be found in tropical and sub-tropical regions all over the world. It has been described in the Antilles,[5] Australia,[6] the Bahamas,[7] Malaysia,[8] New Guinea,[2] Nigeria,[9] Polynesia,[2] Puerto Rico,[10] and in the US in the states of Ohio,[11] Florida,[12] and Hawaii.[13,14] In Brazil, there is a recognized prevalence of 37.2%[15,16] to 45% in the state of Rio de Janeiro and capable of reaching 56.25%.[17] Occurrences of *Platynosomum* spp were also reported in the state of São Paulo[18] with an incidence of 1.07% to 5.6%.[19,20]

The life cycle of this parasite is not well understood, but it is common knowledge that cats acquire them through ingestion of small lizards. The cycle starts with the elimination of eggs in the feces of infected cats. In the environment, the eggs are ingested by slugs or snails and within approximately 15 minutes miracidia emerge and migrate to the connective tissue of the mollusks. During the next 28 days, the miracidia develop to sporocysts I, generating a great number of sporocysts II, which then migrate to the soil through the breathing pores of the mollusks. In the environment, the stage II sporocysts mature and in 30 days they will contain cercariae. At this point of the cycle, they can be ingested by paratenic hosts such as beetles. Lizards or frogs can ingest stage II sporocysts either directly from the environment or by ingesting the infected paratenic hosts. In both events, the metacercariae will be released and remain encysted in the gallbladder of the intermediate host until being ingested by the final host. A few hours after the cat ingests the parasitized intermediary host, the metacercariae migrate through the minor duodenal papillae to the common bile duct. Although rare, some metacercariae can migrate through the minor duodenal papilla and reach the pancreas through the side branches of the pancreatic duct.[21] The predator instinct of the cats ensures that the cycle completes because, even receiving food from their owners, cats keep their habit of hunting.[10,22]

Under experimental conditions, the production of eggs starts 4 to 5 weeks after the cercariae reach the liver. The prepatent period is 8 to 12 weeks. The life expectancy of the adult forms, although unknown, is long as is the period of egg production.[3,23] The pathogenesis of this infection also involves the size and number of parasites. Adult parasites have a flat body of ellipsoid or egg-like shape covered by a thin cuticle, and their size varies between 2.9 to 6.7 mm in length and 0.9 to 1.7 mm in width.[4]

The clinical signs displayed by the cats vary, and their seriousness depends on the number of adult parasites, the time of infection,[22] and on the individual reaction to parasite aggression.[10] Under experimental conditions, it was shown that cats with discrete infections (up to 125 parasites) remain clinically asymptomatic while animals with a high number of parasites (more than 1000) show lack of appetite and lethargy.[10] When present, the clinical signs can be observed between the seventh and sixteenth week after infection and include lethargy, weight loss, and abdominal tenderness. Jaundice, anorexia, and enlargement of the liver may be observed although most cats show no recognizable clinical alterations.[10,22-24] Besides being unspecific, the signs are transitory, but, with the evolution of the disease, the unspecific symptoms generally reappear together with chronic mucous diarrhea or jaundice. In this stage of the disease, the animals die in most cases. Jaundice observed in infected cats with high parasite load is related to bile stasis due to presence of high number of parasites in the bile ducts. Hyperplasia or con-

strictive fibrosis of the bile duct may occur, causing obstruction of bile flow to the duodenum.[2] The presence of the parasites in the bile tract can favor secondary bacterial contamination and contribute to the development of cholangitis and liver abscesses and increase the risk of cholangiocarcinoma and pyogenic cholangitis.[25–28]

Due to the absence of specific clinical signs, the diagnosis techniques are of special importance.[10] Conclusive diagnosis in vivo is made through detection of operculated eggs in the feces,[22] although this depends on the technique employed[29] and on the number of samples examined.[17] Ultrasound examination is the method of choice for diagnosing jaundice in humans, and its use in veterinary medicine is increasing.[30] It is, however, a subjective technique because the same sonographic aspect can appear in different diseases and their interpretation is thus dependant on the skills of the operator and on objective evaluation parameters.[31,32]

The objective of this project was to study the hepatobiliary characteristics detectable with ultrasound for obtaining a better knowledge of evaluation parameters of ultrasound findings in cats infected with *Platynosomum* spp.

MATERIAL AND METHODS

The study included domestic cats of more than 6 months of age[33] collected from different parts of urban Rio de Janeiro. These animals were submitted to ultrasonographic and coproparasitological examinations within a maximum interval of 60 days.

All the cats were examined with the free and informed consent of the owners. The lifestyle of the animals was classified as follows: 1) free, living without direct supervision; 2) semi-confined, living under direct supervision and receiving food regularly but with access to the streets; or 3) confined, cats held in a household without having access to the streets. The study was conducted in a double-blind fashion.

For the ultrasound procedures, the cats were sedated intramuscularly with a combination of ketamine chlorhydrate (Vetaset®, Fort Dodge Saúde Animal Ltda., Campinas, Brazil) at the dose of 10 mg/kg and xylazine chlorhydrate (Rompum®, Bayer do Brasil S.A., São Paulo, Brazil) at the dose of 2 mg/kg.[34]

The samples of feces were obtained from the sedated animals after spontaneous defecation or directly collected from the rectum. The samples were transferred to appropriate vials without conserving agent and maintained at 4° C for up to 24 hours, then processed using the Faust's technique.[35]

The ultrasound exams were carried out with portable bidimensional equipment (General Electrics [GE]® Logiq 100 Pro)with multifrequency transducers (1 convex of 5.0–7.5 MHz, 1 linear of 7.5–10.0 MHz). Tricotomy was performed with a shaving machine (Shave Machine Golden A5 Oster® with blade 40). After cleaning the area with a dry paper towel, ultrasound gel was applied (Carbogel®). The best images were selected and recorded in a video graphic printer (Video Graphic Printer Sony® UPP 895 MD) with appropriate ultrasound printing paper (Ultrasound Printing Paper Sony® UPP-110 HG).

The animals were examined in dorsal as well as left and right lateral decubitus position. Images from liver, gallbladder, and bile ducts were taken in longitudinal, transversal and oblique plane.

Ultrasonographic Criteria

The size of the liver was evaluated subjectively based on its position in the abdomen in relation to the transducer. The parameters and criteria for evaluation are presented in Table 1.

Statistical Analysis

For analysis of discrete data frequency was used. For comparison of 2 proportions, the z test was used.[40] Differences were considered significant when $P < 0.05$.

RESULTS

Seventy two animals (33 males and 39 females) were included in the study. Thirty-three percent of them (24/72) were

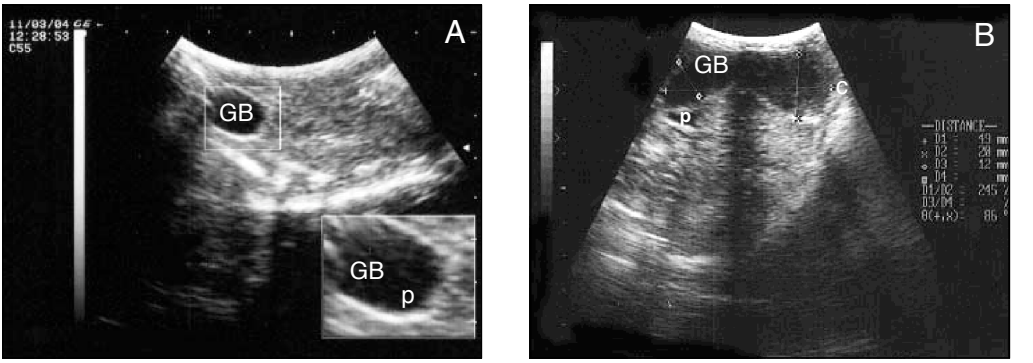

Figure 1. Sonographic images of gallbladders (GB) of *Platynosomum* spp. infected cats. A) Moderate distension, hyperechoic walls (p), and anechoic content; B) Accentuated distension, hyperechoic walls, and echogenic content (c).

Table 1. Parameters and Criteria for Evaluating the Ultrasound Exam of the Liver, Gallbladder, and Bile Ducts of Domestic Cats (*Felis catus*, L., 1758).

Structure	Parameters	Normal	Changed
Liver	Dimensions	Liver contained in the costal arch	Enlarged Reduced
	Shape	Regular	Irregular
	Echogenicity	Intermediate	Hyperechoic Hipoechoic
	Texture	Homogeneous	Heterogeneous
	Hepatic vessels	Gradual reduction of diameter	Homogeneous and more apparent dilation of vessels
Gallbladder	Distension	Related to ingesta	Accentuated
	Form	Pyriform, oval or round	—
	Walls	Not visualized	>2 mm
	Content	Anechoic	Echogenic
Bile ducts	Diameter	Not visualized <4 mm	>4 mm at the maximum point of distension
	Course	Regular	Tortuous
	Periductal Echogenicity	NWM	Hyperechoic

NWM = not worth mentioning.

infected with *Platynosomum* spp. The number of infected females was higher (38.5%; 15/39) than that of infected males (27.3%; 9/33), although the difference was not significant. The lifestyle of the studied animals influenced the prevalence of the infection. There was significant difference among the free-living (42%; 21/50) and confined cats (7.1%; 1/14). The frequency of infection of the semi-confined cats (28.6%; 2/7) showed no significant difference in comparison to free-living and confined cats (Table 2).

The ultrasound findings of the liver, gallbladder (Figure 1), and bile ducts (Figure 2) occurred with similar frequency in animals that eliminated and those that did not eliminate eggs of *Platynosomum* spp. (Figure 3, Table 3).

The frequency of associated alterations of bile ducts and gallbladder (29.1%) or liver, bile ducts, and gallbladder in infected animals (12.5%) was higher than in animals that did not eliminate eggs (20.8% and 2.1% respectively), although the differences were not significant (Table 4).

Table 2. Distribution of Domestic Cats Infected by *Platynosomum* spp. (+)* According to Sex and Lifestyle (urban region of Rio de Janeiro).

	Lifestyle							
	Free		Semi-confined		Confined		Total	
Sex	+/total	%	+/total	%	+/total	%	+/total	%
Male	8/21	38	1/4	25	0/8	0	9/33	27.3
Female	13/29	44.8	1/3	33.3	1/6	16.6	15/39†	38.5
Total	21/50	42[a]	2/7	28.5	1/14	7.1[b]	24/72†	33.3

Different letters in columns significant at a level of % ($z = 2112$; $P = 0.035$).

*Coproparasitological exam.[35]

†The lifestyle of one of the animals was not reported by the responsible caretaker (female that did not shed eggs of *Platynosomum* spp.).

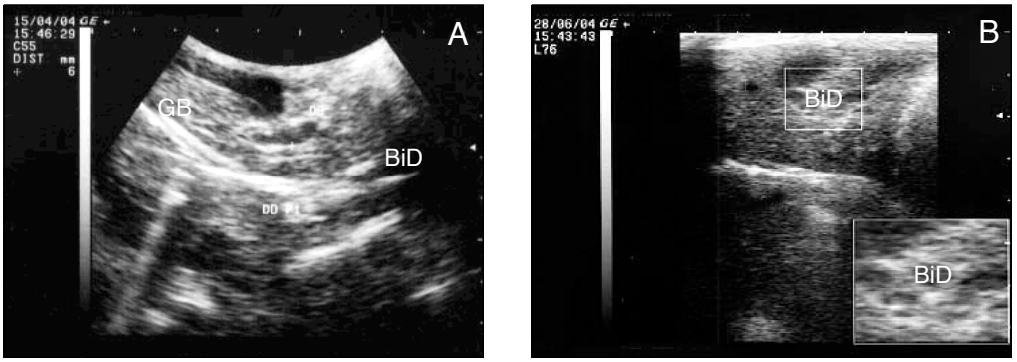

Figure 2. Images of bile ducts (BiD) of *Platynosomum* spp. infected cats. A) Distended, measuring 0.6 cm; B) Distended and tortuous with bullet-like shape (in detail, right inferior corner).

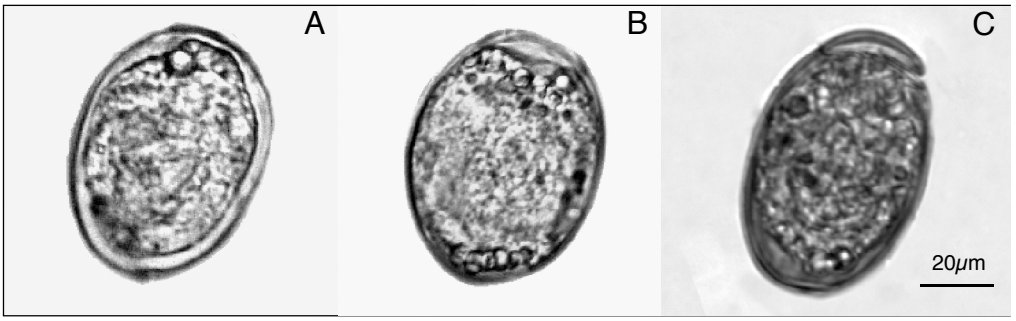

Figure 3. Photomicrographs of eggs of the genus *Platynosomum* found in the feces of cats using Faust's technique. (A) Egg with poorly visible operculum; (B) Egg with well–defined operculum; (C) Egg with disrupted operculum.

DISCUSSION

The free-living cats were more frequently infected than the confined cats, suggesting that a constant and abundant source of food together with restricted access to prey reduced the risk of *Platynosomum* spp. infection. The fact that no difference in infection frequency was observed between confined and semi-confined cats may be attributed to the sample size of semi-confined cats.

The number of females eliminating eggs was not higher than that of males although it is to be expected that females get infected more often while teaching their offspring to hunt.[41] However, it is known that urban females form colonies where the provision of food is constant and abundant, while males either join the female colonies or transit among them.[42,43] Thus, the free-roaming females living in urban centers only eat prey as a complement and mostly hunt to teach

Table 3. Number of Domestic Cats Eliminating Eggs of *Platynosomum* spp. (PL) and with Alterations to the Liver, Gallbladder, and Bile Ducts Detected by Ultrasound (urban region of Rio de Janeiro, May 2003-September 2004).

	Liver					Gallbladder							Bile ducts					
	Dimensions			Echo		Walls			Distension		Content		Diameter		Course		EP	
PL	N	Au (%)	N	Ai (%)	N	Hi (%)	Es (%)	Hi + Es (%)	N	Ac (%)	An	Ec (%)	N	Au (%)	N	Tor (%)	N	Hi (%)
+	24	3(12.5)	23	1 (4.1)	12	11 (45.8)	0	1 (4.1)	21	3 (12.5)	19	5 (6.9)	19	5 (20.8)	11	13 (54.1)	21	3 (12.5)
-	48	5 (10.4)	44	4 (8.3)	32	15 (31.2)	1 (2)	0	48	0	46	2 (4.1)	41	7 (14.5)	30	18 (37.5)	45	3 (6.2)
Total	72	8 (11.1)	67	5 (6.9)	44	26 (36.1)	1 (1.3)	1 (1.3)	69	3 (4.1)	65	7 (9.7)	60	12 (16.6)	41	31 (43)	66	6 (8.3)

(+) Cats eliminating eggs of *Platynosomum spp.*; (-) cats not eliminating eggs of *Platynosomum spp.*; N = without alterations; Au = enlarged; Ai = abnormal; Hi = hyperechoic; Es = thickened; Ac = accentuated; An = anechoic; Ec = echogenic; Tor = tortuous; Ep = periductal echogenicity.

their young, rarely eating the prey but generally offering it to the kittens.[41] Consequently, it is possible that the first infection with *Platynosomum* spp. is acquired by the young animals while learning to hunt.

When analyzing the ultrasonographic parameters in a hepatobiliary system in isolation (liver, gallbladder, or bile ducts) and relating them to the elimination of eggs of the parasite, it can be noted that none of the alterations was more frequent in infected cats than in cats not eliminating eggs (Table 3). The changes on the liver such as enlargement and echogenicity of the hepatic parenchyma detected in the ultrasound exam were only observed in animals that did not eliminate eggs.

These alterations normally reflect histological alterations like fibrosis and vacuolar degeneration that can be observed in the ultrasound exam in an advanced stage of liver disease, including disease caused by parasites of the genus *Platynosomum*.[44] Thus, in the course of platynosomiasis, lesions probably appear in the ultrasound exam in an advanced stage of the disease and may be dependent on the parasite load.

Platynosomum spp. infection may have influenced the sonographic aspect of the gallbladder walls of the cats, given that hyperechogenicity was more frequent among infected cats. This aspect, however, was also observed among animals that did not eliminate eggs of the parasite, suggesting that hyperechogenicity: 1) can indicate an unspecific inflammatory process; 2) can be an artifact of the technique; or 3) can indicate individual alterations.[38,39] Thus, hyperechogenicity may be due to a series of factors, among them inflammation caused by parasites of the genus *Platynosomum*. Another alteration of the bile ducts was echogenic content that, when observed, was more frequent in infected cats (71.4%). This alteration possibly is a consequence of a long fasting period,[39,45] and perhaps appeared more frequently among infected cats due to the inflammatory process or to partial bile duct obstruction caused by the parasites.

Table 4. Distribution of Domestic Cats Infected by *Platynosomum* spp. (PL) and Presenting Ultrasonographic Alterations of the Liver (L), Gallbladder (GB), and Bile Ducts (BiD) (urban region of Rio de Janeiro, May 2003-September 2004).

		L	GB	BiD	L/GB	L/BiD	GB/BiD	L/GB/BiD	NWM	Total
+	n	0	3	5	0	1	7	3	5	24
	%	0	12.5	20.8	0	4.2	29.1	12.5	20.8	100
-	n	3	7	12	0	1	10	1	14	48
	%	6.2	14.5	25	0	2.1	20.8	2.1	29.1	100
Total	n	3	10	17	0	2	17	4	19	72
	%	4.1	13.8	23.6	0	2.7	23.6	5.5	26.3	100

(+) Cats that eliminated *Platynosomum* spp.; (-) cats that did not eliminate eggs of *Platynosomum* spp.; NWM = not worth mentioning.

Furthermore, 3 animals presented with accentuated distension of the gallbladder together with extrahepatic biliary obstruction, not noted in any of the animals of the other group. This emphasizes the possibility of platynosomiasis being involved in the obstruction of the bile ducts, especially in animals developing ductal fibrosis as a consequence of the presence of adult forms of the parasite.[22]

The ultrasound evaluation of the bile ducts showed that this structure presented alterations more frequently (55.5%; 40/72), independently of parasitosis. Considering only the infected animals, alterations in the bile ducts were observed in 67% (16/24), emphasizing the importance of examining this structure when platynosomiasis is suspected, because the bile ducts can enlarge or present tortuosity as a consequence of chronic inflammation or obstruction.[33,45]

The joint assessment of alterations of the gallbladder and bile ducts or liver, gallbladder, and bile ducts (Table 4) made clear that, as already suggested, frequencies were higher in infected animals than in animals that did not eliminate eggs.[44] It has to be pointed out that when comparisons were made by means of necropsies for diagnosing the infection, none of the uninfected cats presented lesions in all 3 hepatobiliary structures and that 89%, if presenting alterations, only presented them in 1 structure.[44] Thus, although the difference was not significant, associated sonographic alterations of bile ducts and gallbladder or liver, bile ducts, and gallbladder should be an indicator for serial coproparasitological exams as the elimination of eggs of *Platynosomum* spp. is weak and intermittent.[29]

Moreover, the low sensitivity of the coproparasitological exams using only one sample[17] may at least in part explain the 12 cats that did not eliminate eggs of *Platynosomum* spp., but presented with 2 or 3 structures with sonographic alterations. Besides, the lesions associated with low parasite loads (<10 adult forms) may not appear in the ultrasound exams[44] and some of the 5 parasitized animals that did not present lesions possibly hosted few parasite forms.

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