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Case Report

Platynosomum fastosum-induced Infections in Domestic Shorthair Cats: a Retrospective Study of Seven Cases

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Abstract

The clinical and the pathological findings associated with *Platynosomum fastosum*-induced infections are described in Domestic shorthair cats by the retrospective analyses of archival records. Icterus was the most predominant clinical alteration observed; three cats demonstrated clinical manifestations of hepatic encephalopathy. The laboratory findings of two cases confirmed severe hepatic dysfunction due to elevations in the serum concentrations of alanine aminotransferase, phosphatase alkaline, and gamma-glutamyl transferase. Hypertrophy of bile ducts occurred in all animals. All cats demonstrated adenomatous hyperplasia of biliary epithelium and/or periductal fibrosis associated with intraductal trematode and/or intralesional ova of *P. fastosum*. One cat had pathological alterations consistent with cystic mucinous hyperplasia and gallbladder mucocele associated with intralesional ova of *P. fastosum,* while dilated cystic intra-hepatic structures associated with chronic platynosomiasis occurred in another cat. Platynosomiasis was an incidental finding in one cat.

Key Words: cats, *Platynosomum fastosum*, parasitic cholangitis, pathology.

Introduction

Platynosomum fastosum (also referred to as *P. concinnum*) is the trematode that inhabits the hepatobiliary system of domestic and wild felids (3). Infection due to *P. fastosum* has been described in tropical climates (3) including regions of Brazil (20, 21), the USA (2, 7), Venezuela (14), Mexico (12), Indonesia (23), Malaysia (11), and in some Caribbean Islands (3, 8). In Brazil, cases have been described in the tropical south-eastern States of Minas Gerais (19), and São Paulo (4, 21), and also in the north-eastern state of Bahia (13) but there are no descriptions of disease in the southern regions of the country. This retrospective study presents the clinical manifestations and pathological alterations associated with *P. fastosum* in cats from Londrina, Parana, Brazil.

Material and Methods

This series represents cats that were submitted for routine necropsy at the Laboratory of Veterinary Pathology, Universidade Estadual de Londrina, Paraná, Brazil during 1983 to 2006. Necropsy accession reports were reviewed from which data relative to the sex, age, and breed of cats affected was collected and tabulated. The clinical manifestations as well as the gross and histopathological lesions described in necropsy reports were reviewed and tabulated. Available archival histological slides were re-evaluated; fragments of formalin-fixed paraffin-embedded (FFPE) tissue fragments of the liver were routinely reprocessed for histopathology evaluation when necessary; duplicates of specific FFPE liver tissue sections were stained by the Periodic Acid-Schiff (PAS) and Masson's trichrome

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histochemical methods. When available clinical laboratory information of the infected cats was obtained and tabulated.

Results

During the period evaluated, 368 cats were necropsied, but manifestations of platynosomiasis were only observed in 1.91% (7/368) of cases. The biological data and the main clinical and pathological findings are summarized in Table 1. All parasitized cats were of the Domestic shorthair breed; 5 were females, with 2 male counterparts; all of these cats were aged animals. Icterus of mucous membranes was the most predominant (71.43%; 5/7) clinical manifestation described in contaminated cats. Neurological manifestations consistent with hepatic encephalopathy occurred in 42.86% (3/7) of the parasitized cats. Most (57.14%; 4/7) cats with platynosomiasis were euthanized due to complications associated with hepatic disease; three died spontaneously.

Clinical laboratory data was only available for two cats, and are summarized in Table 2. Haematological alterations were more severe in Cat Nº 1 than Cat Nº 2. In the first cat there was non-regenerative anaemia and leucocytosis with neutrophilia; significant haematological alterations in the second cat were restricted to eosinophilia. Glycosuria and bilirubinemia were also observed in cat Nº 1 (data not shown). However, both cats demonstrated marked elevation of serum biochemical values for alanine aminotransferase (ALT), phosphatase alkaline (PA), and gamma-glutamyl transferase (GGT).

Significant gross lesions in most cases were primarily within the hepatobiliary system, and consisted of moderate to severe hypertrophy of bile ducts and icterus. Obstruction of bile ducts due to visible trematodes occurred in 28.57% (2/7) of parasitized cats; one of these also had nutmeg liver. One cat (Nº 3) demonstrated remarkable gross hepatic lesions; the liver was severely enlarged, and pale, with marked, different-sized (0.5 – 3.5 cm), pale, nodular areas that were visible at the capsular surface of the liver (Fig 1A). Two distinct gross lesions were observed in different regions of the sectioned liver of this cat: one was characterized by severe intraductal accumulations of bile pigments with associated bile duct hypertrophy (Fig. 1B), and in other regions the bile ducts were hypertrophic and obliterated due to accumulations of a mucinous secretion (Fig. 1C). Further, there was severe thickening of the wall of the gallbladder (Fig. 1D). Additionally, there was severe pulmonary haemorrhage and oedema in one cat, and cutaneous ulceration of the nose in another.

The principal histopathological alterations associated with *P. fastosum* are summarized in Table 1. Histological alterations in most cases were predominantly portal due to marked increase in fibrous connective tissue resulting in distinct visualization of the hepatic lobules, and were classified as moderate to severe, chronic, intrahepatic cholangitis associated with intraductal trematode (Fig 2 A-B), but in some cases there were several intraductal flukes resulting in ductal obstruction (Fig. 2C). In cat Nº 5, there were areas of cholangitis and cholangiohepatitis associated with intraductal trematode, while in other sections of the liver there was marked adenomatous hyperplasia without visible presence of the adult fluke or ova (Fig. 2 D-E). In addition, to the accumulations of adult trematodes within bile ducts and associated adenomatous biliary proliferation, several, periportal and intrahepatic, dilated cystic-like structures, that were surrounded by a simple epithelium, were observed in cat Nº 4 (Fig. F-G); periportal cystic structures were frequently surrounded by severe accumulations of inflammatory exudate. Cat Nº 3 demonstrated areas of adenomatous hyperplasia of bile duct epithelium, periductal fibrosis associated with few intralesional ova admixed within severe influx of inflammatory cells (Fig. 3A). Additionally, there was severe cystic proliferation of bile ducts epithelium with papillary projections that contained accumulated mucinous exudate (Fig. 3B); proliferation of bile duct tissue within the lumen was readily observed by the Masson's trichrome stain (Fig. 3C), while the mucinous material accumulated within bile ducts was strongly PAS positive (Fig. 3D). Further, most of the hepatic parenchyma of this cat was severely destroyed, and some of these areas contained bacterial colonies admixed with neutrophilic exudate; in other areas there were severe biliary and canalicular cholestasis and portal proliferation of epithelial cells of bile ducts. Further, gross and histopathological manifestations of *P. fastosum* infection were observed in one cat that had a cutaneous nasal squamous cell carcinoma with pulmonary and lymph node involvement.

Table 1. Summary of the clinical findings, pathological alterations, and outcome of Domestic shorthair cats with *Platynosomum fastosum*

Cat	Sex and age	Physical and clinical examination	Principal gross lesions	Principal histopathological findings	Outcome
1	Castrated female , adult	Anorexia Chronic body wasting Dehydration Icteric mucous membranes Intra-abdominal mass Manifestations of hepatic encephalopathy	Bile duct hypertrophy Icterus Nutmeg-liver Obstruction of intrahepatic bile ducts by flukes Serous atrophy of pericardial fat	Adenomatous hyperplasia Centrilobular necrosis Cholangitis with intralesional fluke Cholestasis Periductal fibrosis	Euthanasia
2	Female, 11-yrs	Anorexia Apathy Chronic body wasting Dehydration Icteric mucus membranes Vomit	Bile duct hypertrophy Icterus Obstruction of intrahepatic bile ducts by flukes Serous atrophy of pericardial fat	Bridging fibrosis Cholestasis Periductal fibrosis with intralesional trematode	Euthanasia
3	Female, adult	Anorexia Icteric mucus membranes Manifestations of hepatic encephalopathy	Bile duct hypertrophy Bile stasis Icterus Mucinous biliary obstruction	Adenomatous bile duct hyperplasia with intralesional ova Cystic mucinous hyperplasia of the gallbladder Extrahepatic cholestasis Gallbladder mucocele Massive hepatocellular necrosis Periductal fibrosis	Euthanasia
4	Male, 3-yrs	Ataxia Convulsions	Bile duct hypertrophy Pulmonary haemorrhage and oedema	Cholestasis Intrahepatic and paraportal cystic dilations Parasitic cholangiohepatitis Periductal fibrosis Pulmonary haemorrhage and oedema Status spongiosa of cerebellar white matter	Spontaneous death
5	Male, adult	Anorexia Apathy Dehydration Icteric mucus membranes	Bile duct hypertrophy	Adenomatous hyperplasia t with intralesional parasite Periductal fibrosis	Euthanasia
6	Female, adult	Dehydration Icteric mucus membranes	Bile duct hypertrophy Icterus	Cholestasis Periductal fibrosis with intraductal parasite	Spontaneous death
7	Female, 7-yrs	Anorexia Dehydration Nasal sporotrichosis	Bile duct hypertrophy Enlarged lymph nodes Ulcerative nasal lesion	Nasal squamous cell carcinoma with pulmonary and lymph node metastasis Periductal fibrosis with intraductal trematode	Spontaneous death

Table 2. Summary of laboratory values of Domestic shorthair cats with *Platynosomum fastosum*

Laboratory parameters	**Reference values**	**Cat 1**	**Cat 2**
Haematology			
Hematocrit (%)	24.0-46.0	15.6	40.2
Haemoglobin (g dL^{-1})	8.17-15.26	5.1	13.3
Red blood cells ($x10^6$ mm^{-3})	5.92-11.16	3.61	9.04
MCV (fl)	36.96-54.98	43.2	44.5
MCHC (g dL-1)	26.24-35.91	32.7	33.1
Leucocytes (mm^{-3})	10,570-14,390	38,690	17,830
Segmented neutrophils (mm^{-3})	6,100-9,480	35,208 (91%)	16,760 (94%)
Lymphocytes (mm^{-3})	2,410-3,990	3,482 (9%)	357 (2%)
Eosinophils (mm^{-3})	200-610	0.0	357 (2%)
Monocytes (mm^{-3})	290-470	0.0	357 (2%)
Platelets ($x10^3$ mm^{-3})	200,670-377,000	361,000	378,000
Serum chemistry			
Alanine aminotransferase (ALT)	6-83 (26±16)	236	266.6
Phosphatase alkaline (PA)	25-93	1089	272.7
Gamma-glutamyl transferase (GGT)	1.3-5.1	80.6	23.3

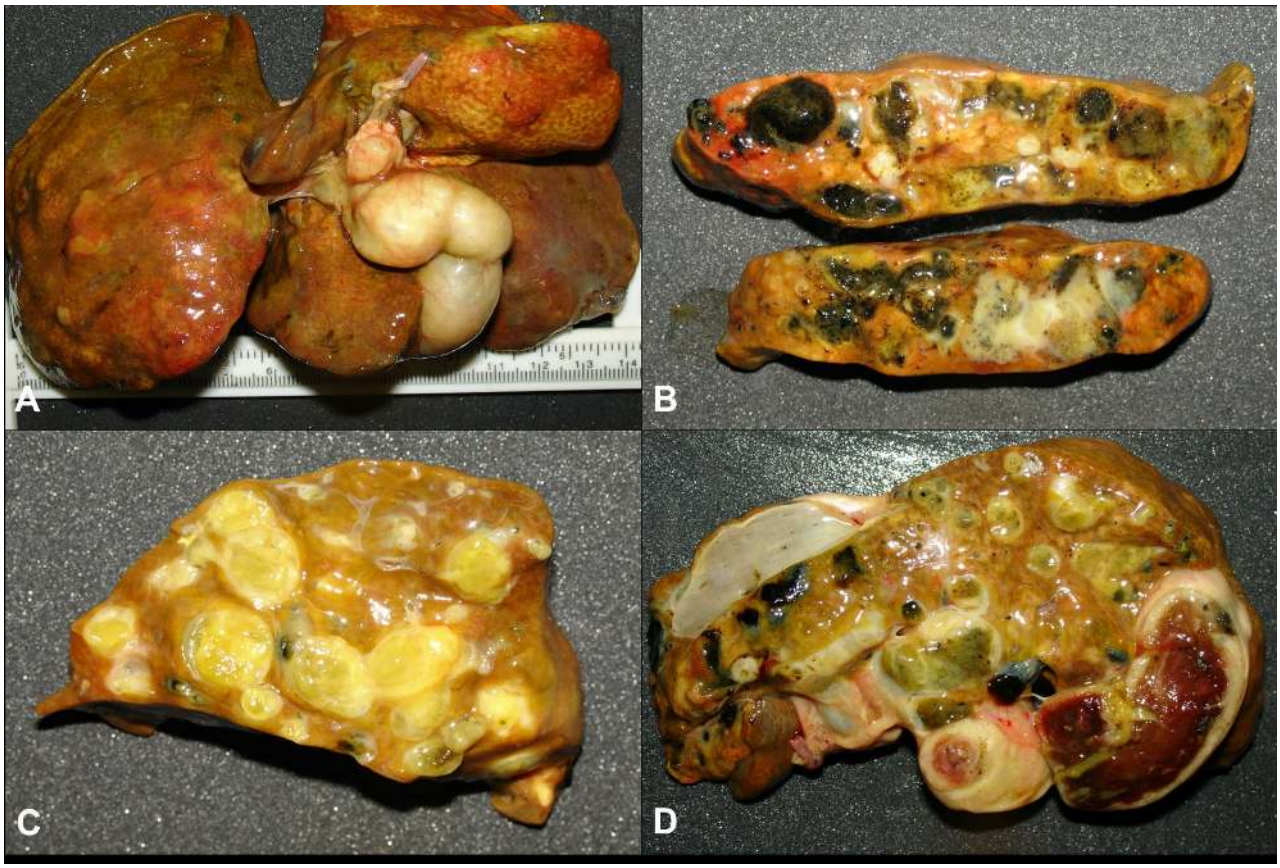


Figure 1. Pathological manifestations of *Platynosomum fastosum* infection in a Domestic shorthair cat. Observe the severely enlarged and pale liver with several nodular formations at the capsular surface and marked thickening of the gallbladder (A). Severe accumulations of bile pigment (B) and a mucinous-like secretion (C) are shown within intrahepatic bile ducts at the sectioned surfaces of the liver. The severely thickened wall of the gallbladder and areas of mucinous intraductal accumulations are shown at the sectioned surface of the liver (D).

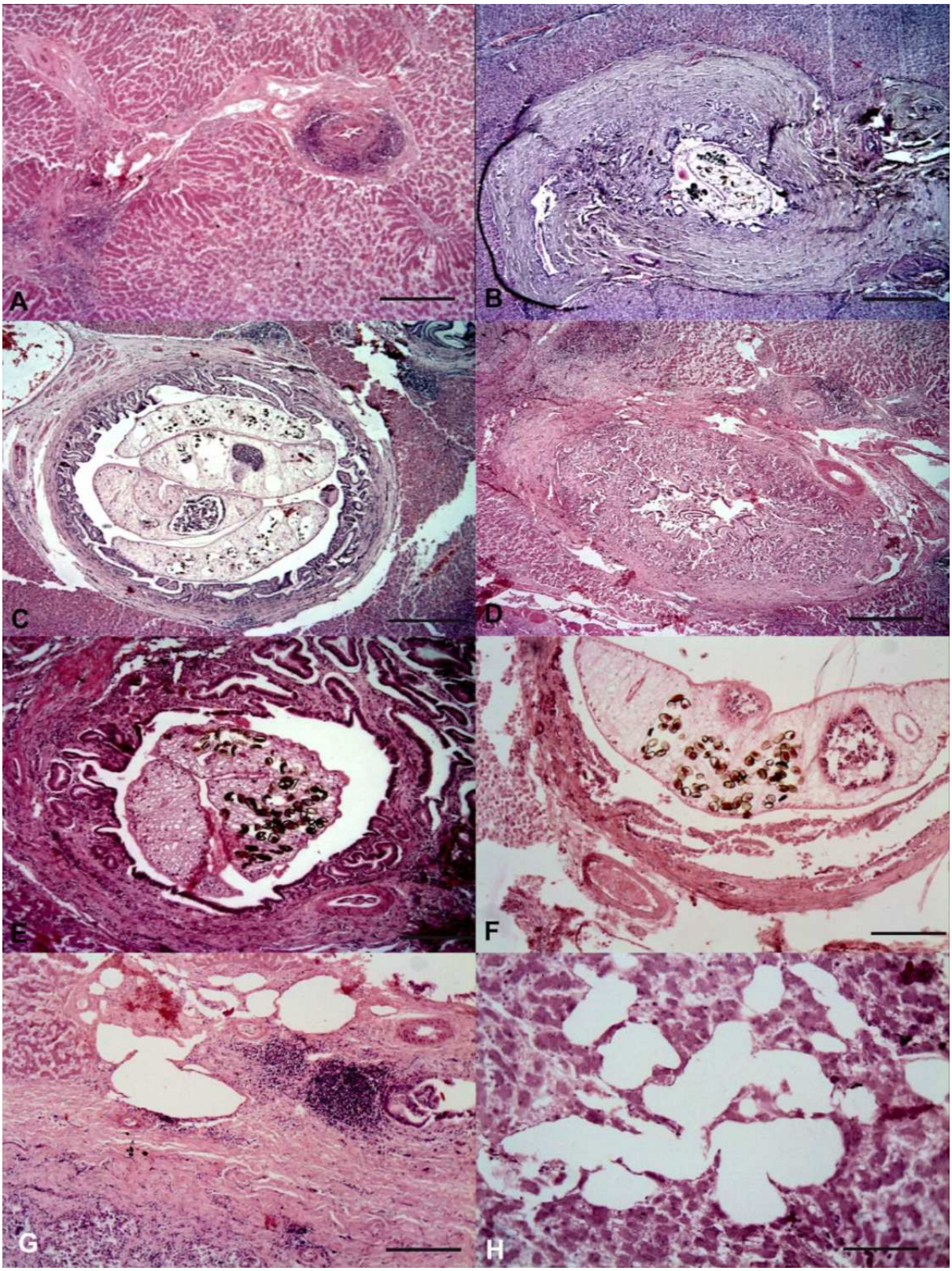


Figure 2. Histopathological manifestations of platynosomiasis in Domestic shorthair cats. There is severe periductal fibrosis with intraductal trematode (A). Observe periductal fibrosis with intraductal trematode and manifestation of adenomatous hyperplasia of bile duct epithelium (B). Several examples of the adult fluke are packed within a proliferated bile duct (C). There is adenomatous bile duct hyperplasia without intraductal trematode (D) and demonstration of parasitic cholangitis (E) within the same liver. Observe periductal fibrosis with intraductal fluke (F), portal (G) and intrahepatic (H) cystic formations within the liver of the same cat. (A-H; Hematoxylin and Eosin stain; Bar, A, G-H, 200 µm; B-F, 100 µm)

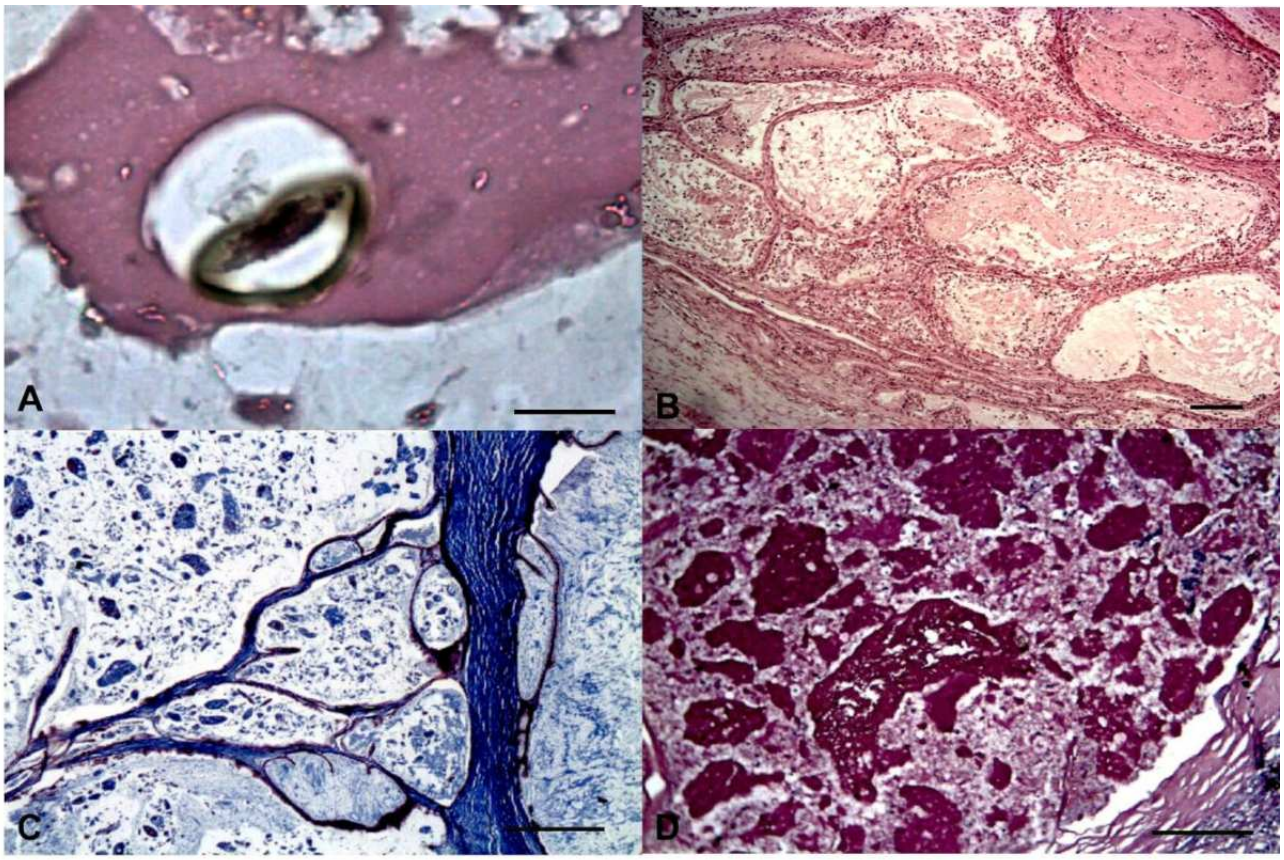


Figure 3. Atypical histopathological findings associated with *Platynosomum fastosum* in a Domestic shorthair cat. There is an ovum consistent with that of *P. fastosum* (A) and cystic proliferation of bile duct epithelium containing mucinous exudate (B). Observe the proliferated biliary epithelium (C) and the PAS positive reaction of accumulated mucinous exudate (D). (A-B; Hematoxylin and Eosin stain; C, Masson's trichrome stain; D, Periodic Acid-Schiff; Bar: A, 20 µm; B-C, 200 µm; D, 50 µm).

Discussion

A diagnosis of parasitic cholangitis associated with *P. fastosum* was made based on characteristic gross findings in few cases, but histopathological demonstration of parasitism was more efficient in confirming the diagnosis of platynosomiasis. A histopathological diagnosis of *P. fastosum* infection was achieved due to the finding of combinations of the following features: the presence of intraductal trematode, intralesional ova, periductal fibrosis, and/or adenomatous hyperplasia of bile duct epithelium. The pathological and parasitological findings of these cats are consistent with previous descriptions of lesions induced by the liver fluke, *P. fastosum,* of cats (3, 6, 7). The frequent reports of platynosomiasis in domestic cats (4, 19, 21), wild felids (5), and in primates (15) from different geographical regions of Brazil, might suggest that this is an endemic disease but poorly reported or underdiagnosed.

Most clinical manifestations of *P. fastosum* infection described in these cats have been previously reported (3, 16). Nevertheless, icterus was the predominant clinical feature of platynosomiasis, occurring in 71.43% (5/7) of the infected cats from this study. Icterus in platynosomiasis is associated with the obstruction of biliary secretion (13, 14), which is a frequent manifestation of chronic *P. fastosum* infection (20). However, the neurological manifestations associated with hepatic encephalopathy as observed in three cats from this study have not been previously described in this parasitism. In two cases there was severe and extensive destruction of the hepatic parenchyma, either due to centrilobular necrosis with cholestasis (Cat 1), or to massive hepatocellular necrosis, cystic mucinous hyperplasia, and cholestasis (Cat 3), probably resulting in hepatic failure and consequent hepatic encephalopathy due to hyperammonemia (16). Alternatively, platynosomiasis is frequently an asymptomatic (11), or subclinical infection (17, 23), and the manifestation of disease is only observed during necropsy and/or by histopathology (6), as was the case of the cat with the cutaneous squamous cell carcinoma.

During this study female cats (71.42%; 5/7) were super-represented relative to males; similar results have been described in association with this parasitism (8, 12). Although this predominance might reflect the number of female relative to male cats submitted for necropsy, it has been suggested that female cats are more likely to be infected by *P. fastosum* than males due to their maternal habits of hunting lizards to feed

their offspring (12). This predator habit was described by the owners of two cats from this study; nevertheless, all of the cats from this study, and in other cases of platynosomiasis (2, 8, 20) were Domestic shorthairs. This breed of cats is more likely to roam the streets and hunt, thereby increasing their risk of eating one of the intermediate hosts of this trematode and become infected.

The laboratory findings observed in two cats from these series, characterized by elevated serum levels of ALT, GGT, and PA, have been previously described in platynosomiasis of cats (4, 7, 21), and primates (15). However, the anaemia observed in this report and in other studies (2, 4), seems not to be frequent manifestations of this parasitism, and was not a haematological alteration described even in experimentally induced platynosomiasis (17). Nevertheless, the association, if any, with *P. fastosum* infection in this case remains obscure, but the cat did not demonstrate any clinical finding or pathological alterations that could have been directly associated with anaemia.

The most remarkable pathological manifestations of parasitism were observed in cat Nº 3. In the liver of this animal, there were areas of adenomatous hyperplasia of the bile duct associated with few intralesional ova; however, the gross and histopathological lesions of other parts of the liver are consistent with cystic mucinous hyperplasia of the gallbladder (10, 18), which probably proceeded into gallbladder mucocele (16, 18), characterized by the severely thickened gallbladder wall with accumulated mucinous secretion (10), resulting in severe extrahepatic cholestasis. Further, the PAS-positive reaction produced by the mucinous exudate in this cat, was also described in dogs with cystic mucinous hyperplasia (10), and might suggest the proteinaceous nature of this secretion. Cystic mucinous hyperplasia is an incidental necropsy finding frequently described in dogs (16, 18) and sheep (16), but the incidence of this condition in cats is uncertain. Although the etiology of cystic mucinous hyperplasia is unknown, mucosal hyperplasia of the large bile ducts is frequently observed in chronic cholangiohepatitis due to fluke infestation (16); this might be the cause of cystic mucinous hyperplasia herein described, and in similar platynosomiasis-induced cholestatic diseases of cats (7, 14).

Another salient histological alteration observed during this study was the finding of dilated cyst-like areas in cat Nº 4. Cystic hepatic lesions have been previously associated with platynosomiasis in domestic cats (21), but in these cases cystic formations were also observed grossly; the cysts of this cat were only observed histologically. Hepatic cysts in cats are either of congenital or acquired origins and are normally lined by a single layer of biliary epithelium (18), as was observed in this cat. Congenital cystic formations are frequently associated with polycystic kidney disease (18); whereas acquired cysts due to *P. fastosum* are probably induced by chronic inflammatory reactions and consequent biliary obstruction (21), and might have been the cause of these cystic formations of this cat.

Most cats evaluated during this study demonstrated either the intralesional adult trematode or few ova associated with adenomatous bile duct hyperplasia and/or periductal fibrosis, which are probably the principal and characteristic histopathological lesions associated with platynosomiasis (6, 8, 17). However, in one cat there were areas of typical histological characterization of parasitism without the concomitant trematode or ova; similar alterations have recently been described in cats (88.89%; 8/9) infected with *P. fastosum* from Grand Cayman (8). Further, the finding of the adult fluke or ova associated with this parasitism is not frequently observed during histological evaluations (6). Additionally, the histopathological patterns of feline platynosomiasis in endemic regions can be compared to the histopathological features associated with bovine eurytrematosis, where gross characterization of infection does not correlate with histopathological manifestations of disease (1, 9). Consequently, it was proposed that adenomatous hyperplasia and periductal fibrosis, with or without concomitant intralesional manifestations of the adult fluke or ova, in cats from endemic geographical regions of this disease, are diagnostic features of this parasitism, and histopathological manifestation of infection can be classified as active or chronic resolving platynosomiasis (8). Accordingly, active parasitism contains the characteristic histopathological manifestations induced by *P. fastosum* with histological confirmation of intraductal fluke or ova, while resolving platynosomiasis demonstrates typical histological features of parasitism without intralesional ova or the adult intraductal trematode.

In conclusion, platynosomiasis was diagnosed in seven Domestic shorthair cats based on combinations of pathological and parasitological features which are consistent with this disease. Bile duct hypertrophy and adenomatous epithelial hyperplasia might represent the principal pathological alterations associated with parasitism by *P. fastosum* in cats from endemic areas.

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