

Case report

RESOLUTION OF A BILIARY OBSTRUCTION CAUSED BY *PLATYNOSOMUM FASTOSUM* IN A FELINE BY A MODIFIED CHOLECYSTODUODENOSTOMY APPROACH – CASE REPORT

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Diseases of the biliary tract are the most common feline hepatic disorders. Cholangitis is the term used to describe inflammation of the biliary tract in felines and can be associated with parasites. Affected animals must undergo clinical or surgical treatment, or a combination of both. In this paper we presented a 10-year-old male, mongrel feline with an obstruction of the biliary tract caused by a rare infection of *Platynosomum fastosum*.

Key words: cats parasitic disease, Platinoosmosis, surgery

INTRODUCTION

The most common hepatic disorders in cats are diseases of the biliary tract, unlike dogs, which suffer more frequently from problems of the liver parenchyma [1]. Cholangitis refers to inflammation of the biliary tract that can spread to the hepatic parenchyma. It can be neutrophilic, lymphocytic or associated with parasites such as *Platynosomum fastosum* [2,3]. Extra-hepatic infestations of the biliary tract by *P. coccinum*, *P. illiciens* and *P. fastosum* are common [4].

Conventional obstructions of the biliary tract are associated with different causes, which can be divided into extra- or intra-luminal compressions [5]. Malignant neoplasms and inflammatory conditions such as cholangiohepatitis, pancreatitis, or cholelithiasis are the main causes of extra-hepatic obstructions in felines [6,7]. Neoplastic conditions, diaphragmatic ruptures, cholelithiasis [6], choledochal cysts [8], and traumatic avulsion of the common biliary duct [9] may also cause obstructions. Intraluminal obstruction is a rare occurrence in domestic animals and can originate from gallstones, thick bile, or gallbladder and biliary duct tumors [10].

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Here, we report a case of an extra-hepatic biliary obstruction in a feline caused by a *P. fastosum* infection.

CASE PRESENTATION

A 10-year-old male, mongrel feline was brought to the “Governador Laudo Natel” Veterinary Hospital from UNESP Jaboticabal (HVGLN). It had been diagnosed as a hepatitis carrier 6 months ago. The patient was treated with prednisone and B complex formulation to provide hepatic support over a period of 4 months. However, clinical signs, such as anorexia, and prostration, had reappeared with a higher intensity. Biochemical and diagnostic imaging testing was carried out, which confirmed hepatopathy and a biliary obstruction due to gallstones.

During anamnesis, it was reported that the patient had unrestricted street-access, a variable appetite and suffered from recurrent vomiting, though there was a habitual consistency, volume, and color of the feces. The feline presented with mild jaundice at physical examination, a discrete anisocytosis, thrombocytopenia (80000 platelets/mm³), leukocytosis with neutrophilia and eosinophilia (26200 leukocytes/ μ L; 21710 segmented neutrophils/ μ L and 131 eosinophils/ μ L); serum elevation of GGT¹ (45 U/L) and mild icteric serum.

Abdominal ultrasound revealed the presence of a discrete hepatic augmentation and hyperechogenic structures that formed acoustic shadows over left hepatic lobe; it also showed that the gallbladder was not filled and revealed a dilated choledochal duct. These findings led to the diagnosis of cholangiohepatitis and the presence of gallstones.

Treatment was initiated using oral ondansetron (1 mg/kg BID, “Bis in die” or twice a day) and scopolamine (25 mg/kg BID). Despite this, the patient returned 3 days later, presenting with anorexia, adipsia and emesis. Surgical intervention was now suggested and a cholecystoduodenostomy was carried out with the intention of unclogging the biliary tract and improving the patient’s general state.

Through an exploratory laparotomy, it was observed that there was a severe thickening of the gallbladder wall (Figure 1A), which was drained and, consequently, its contents analyzed (Figure 1B). In response to the presence of gallstones in the biliary tract (Figure 1C), an incision was made in the gallbladder to place a retrograde catheter in the choledochal duct (Figure 1D) and throughout the duodenal papilla, thus removing any choleliths which were present by washing with warm sterile saline. This differed from the standard technique, as the duodenal antimesenteric surface was transposed to achieve apposition of the gallbladder, without having to release it from the liver, which could have otherwise caused significant hemorrhage. The area was closed by surrounding the gallbladder and the duodenum with wet and warm sterile dressings. An initial surgical suture was made between the structures using simple interrupted

stitching, with a non-absorbable suture (3-0 Nylon), creating a stoma (Figures 1E and 1F). An omentopexy was performed over the incision area.

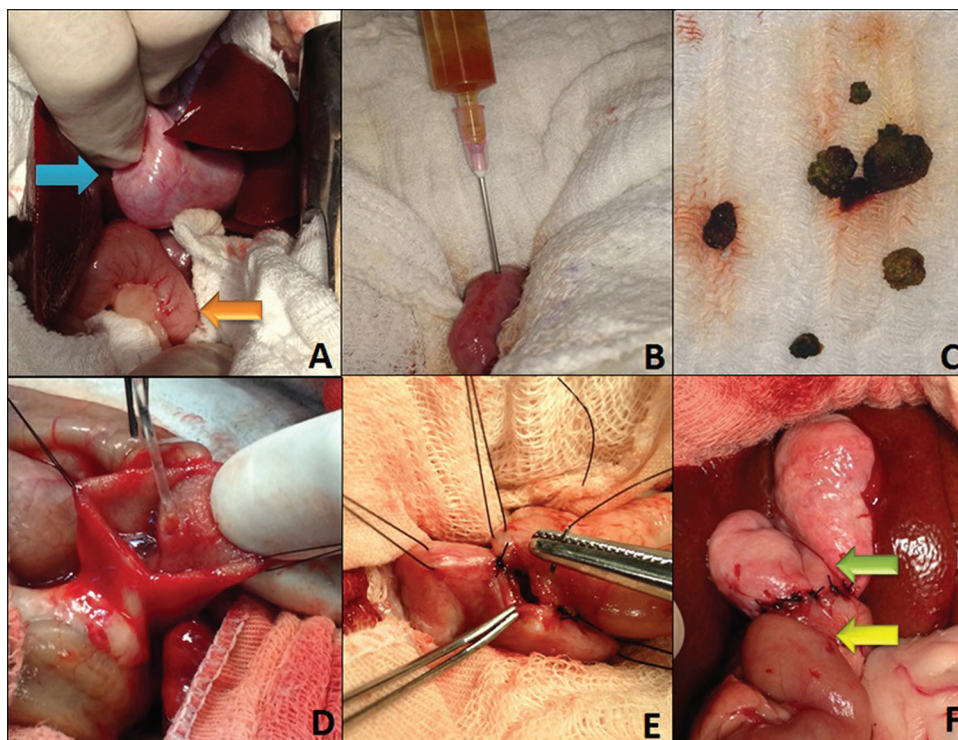


Figure 1: Modified cholecystoduodenostomy in a *Platynosomum fastosum*-carrier feline. **A** – blue arrow shows plethoric gallbladder and the orange arrow indicates the duodenal loop. **B** – cholecystocentesis. **C** – gallstones over the extra-hepatic biliary tract. **D** – duodenal papilla with a catheter after enterotomy. **E** – ligature between incisions of the gallbladder wall and the duodenal antimesenteric surface, simple interrupted stitching using 3-0 Nylon suture. **F** – cholecystoduodenostomy, showing the perfect apposition between the gallbladder (green arrow) and the duodenum (yellow arrow). Note, the gallbladder remained in the appropriate hepatic location due to modified technique. HVGLN's General Surgery Service UNESP - Jaboticabal.

The material collected through the biliary puncture was analyzed microscopically, and *Platynosomum fastosum* eggs were identified (Figure 2), confirming the diagnosis of feline platinosomosis. An antiparasitic medication was prescribed, a single dose of praziquantel (6 mg/kg), which was repeated after one month.

Twelve days after surgery, the patient returned for a follow-up appointment and appeared to be in good general condition, with a normal appetite, absence of vomiting, and firm, but yellowish, stools. The patient was monitored for a year, and maintained good health without any further complications.



Figure 2. A *Platynosomum fastosum* egg seen while collecting bile smear. HVGLN's General Surgery Service, UNESP - Jaboticabal.

DISCUSSION

Felines are considered definitive hosts of *P. fastosum* and they excrete the parasite's eggs through their stools. They can be infected after ingesting frogs, geckos or lizards (intermediary hosts); these in turn are infested by trematodes [11].

Despite being considered a key hepatic parasite among domestic felines, *P. fastosum* infestations are often underdiagnosed due to nonspecific clinical signs [10]; in the present case, the patient initially presented with broad and vague symptoms and the chronicity of the disease increased over time and unspecific symptoms of biliary compromise were noted. Some authors have also reported diverse clinical signs from patients with *P. fastosum* infections, including mild inappetence and weight loss, leading to anemia, ascites, jaundice and even death [12,13].

In a disease prevalence study in northeastern Brazil, samples of 148 euthanized cats were collected at a zoonoses center. In 60 necropsied animals (42.6%), eggs of *P. fastosum* were seen in the bile [14]. Another study carried out by Andrade et al. [15] in which 348 cats were necropsied in one year, 11 (3.16%) of them were diagnosed with the presence of *P. fastosum*. Of these 11 cats, three had their death due to cholangiosarcoma, and in two there were the presence of metastases. Cholangiosarcoma is a tumor not commonly found, but under constant inflammatory conditions of the biliary tree due

to the migration of the parasite, the neoplastic process can settle, generating greater complications for the host [16,15].

In this case there was an evident, clear disease progression with a worsening of the clinical signs depending on the location of the parasite over the biliary ducts and gallbladder. This in turn led to a biliary flow obstruction and the formation of gallstones, consequently causing hepatic and bile duct inflammatory disturbances. Biliary obstructions can be multifactorial, with different causes as gross presence of parasites, consequent inflammation with bile duct hyperplasia or fibrosis, and the formation of choleliths [17-19].

The observed symptomatology and history in this case, including the general hepatic compromise and disease progression, the patient's street-access, GGT augmentation, and eosinophilia [20] were suggestive of a *P. fastosum* infestation, which was confirmed by the detection of eggs in the biliary ducts.

As mentioned above, a definitive diagnosis of *P. fastosum* infection is made through the detection of operculated parasitic eggs in the stool [21]. However, despite the possibility of detecting eggs through a fecal examination, this test is not completely effective in confirming an infestation: only 25% of infested cats are identified using this method and serial repetitions are often necessary [13,22], together with clinical signs, laboratory findings, and diagnostic imaging exams [9,10].

In the present case, the biliary flow obstruction was associated with a blockage caused by parasitic egg release, limiting the accuracy of the test. Analysis of the bile collected through ultrasound-guided puncture in order to diagnose this parasitic disease has been previously reported in literature [3], confirming the diagnostic strategy used in this case.

Hyperechogenic structures form acoustic shadows are seen during ultrasound examinations and these suggest the presence of gallstones, which are probably formed during a previous inflammatory process, but could also be an indication of local parasites, as previously cited [13]. In the current case, an ultrasound examination was critical in confirming a diagnosis of cholangiohepatitis and the presence of gallstones, revealing the patient's biliary obstruction.

Soldan and Marques [3] noted that the efficacy of clinical treatment depends on the extent and degree of damage caused by the parasites in the liver, biliary ducts, and gallbladder, until the patient is diagnosed and treated [3]. Failure to diagnose the patient at the initial stage of the disease explains the sustained parasitic and clinical nature of the disease, rendering the therapeutic treatment approach ineffective and necessitating a mandatory surgical intervention through cholecystoduodenostomy.

Possible complications of extra-hepatic biliary tract surgeries include hemorrhage, suture dehiscence and biliary peritonitis, biliary duct obstruction, stenosis of biliary-enteric anastomosis, ascendant cholangiohepatitis, recurrent cholelithiasis, and an alteration of gastrointestinal physiology [23,24]. None of these complications were

observed post-surgery, with the patient achieving complete reversion of the critical clinical condition after cholecystoduodenostomy. Two weeks after surgery, the patient was presented in optimal clinical health, with a return of appetite and absence of pain, jaundice, and vomiting. One month post-surgery, the patient recovered completely, thus confirming that the surgical treatment was successful.

A critical part of the cholecystoenterostomy procedure is obtaining a sufficiently large orifice to allow drainage of intestinal reflux from the biliary tract to the gut. It has been suggested that a 2.5-4 cm aperture length minimizes the possibility of a recurrent stenosis and cholangitis, associated with the insufficient drainage of the biliary tract and intestinal reflux [25]. In this case, the surgical incision mirrored that in previous reports [7,9,25-27]. A cholecystoduodenostomy is the procedure of choice in dogs and cats, because their bile is in contact with the duodenum. Another surgical option is a cholecystojejunostomy, but this physiological approach may cause multiple problems associated with lipid malabsorption and the proper contact of gastric fluids in the duodenum, in response to the absence of bile, leading to an absence of pH neutralization [28]. A modification of the technique used in this case, maintaining the gallbladder at the hepatic bed, could avoid the occurrence of trans-operative hemorrhages, as the modified technique reduced the amount of hepatic parenchyma handling in order to release the gallbladder, which was part of the original technique [25]. This minimizes the post-surgical complications caused by bleeding.

The success of this modified approach was due to the appropriate selection of surgical approximation along with the clinical treatment using anti-parasitic drugs. Mehler and Bennet [27] described the possibility of using a hepatic biopsy to identify inflammatory conditions caused by obstructive processes, but this was not performed in the present case [27].

Greater attention is needed to successfully diagnose hepatic diseases in cats. Despite being a rare condition, the presence of *P. fastosum* should be taken into consideration in cases when the patient exhibits signs of hepatic disturbance, independent of historical reports of lizard ingestion. In the present report, the modified cholecystoduodenostomy technique was considered appropriate and successful, and it is an important alternative to the deviation of biliary flow, without the need for excessive handling of the hepatic parenchyma.

Authors' contributions

FA, MG, SL, CA, DL, MP carried out the molecular genetic studies, participated in the sequence alignment and drafted the manuscript. MG and MP conceived of the study, and participated in its design and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

1. Center SA: Disease of the gallbladder and biliary tree. Vet Clin North Am Small Anim Prac 2009, 39:543 – 598.
2. German A: Colangite felina. Veterinary Focus 2009, 19:41–46.
3. Soldan MH, Marques SMT: Platinosomose: Abordagem na clínica felina. Revista FZVA 2011, 18.
4. Travassos L, et al. Trematódeos do Brasil. Memórias do Instituto Oswaldo Cruz 1969, 67:140-141.
5. Nelson RW, Couto CG: Hepatobiliary disease in the cat. In: Small Animal Internal Medicine. California: Elsevier, 2014. 520–540.
6. Mayhew PD, Holt DE, Mclear RC, Washabau RJ: Pathogenesis and outcome of extrahepatic biliary obstruction in cats. J Small Anim Pract 2002, 43:247–253.
7. Buote NJ, Mitchell SL, Penninck D, Freeman LM, Webster CR: Cholecystoenterostomy for treatment of extrahepatic biliary tract obstruction in cats: 22 cases (1994 – 2003). J Am Vet Med Assoc 2006, 228:1376–1382.
8. Best EJ, Bush DJ, Dye : Suspected choledochal cyst in a domestic shorthair cat. Journal of Feline Medicine and Surgery, v. 12, n. 10, p. 814 – 817, 2010.
9. Bacon NJ, White RA: Extrahepatic biliary tract surgery in the cat: a case series and review. J Small Anim Prac 2003, 44:231–235.
10. Lehner C, Mcanulty J: Management of extrahepatic biliary obstruction: a role for temporary percutaneous biliary drainage. Compend Contin Educ Vet 2010, 32:E1-E10.
11. Ferreira AMR, Almeida ECP, Labarthe NV: Liver fluke infection (*Platynosomum concinnum*) in Brazilian cats; prevalence and pathology. Feline Practice 1999, 27:19-22.
12. Castro L, Albuquerque G: Ocorrência de *Platynosomumilliciens* em felinos selvagens mantidos em cativeiro no estado da Bahia, Brasil. Rev Bras Parasitol Vet 2008, 17:239–241.
13. Azevedo FD, Da Veiga CCP, Scott FB, Correia TR, Fernandes JI, Verocai GG: Avaliação radiográfica e ultrassonográfica do fígado e da vesícula biliar em gatos domésticos (*Felis catus domesticus*) parasitados por *platynosomumilliciens* (Braun, 1901) Kossak, 1910. Revista Brasileira de Medicina Veterinária 2013, 35:283-288.
14. Braga, RR, Teixeira, AC, Oliveira, JAA, Cavalcanti, LPG: Prevalence of *Platynosomum fastosum* infection in free roaming cats in northeastern Brazil: Fluke burden and grading of lesions. Veterinary parasitology, 2016, 227, 20-25.
15. Andrade, RLFS, Dantas, AFM, Pimentel, LA, Galiza, GJN, Carvalho, FKL, Costa, VMM, Riet-Correa, F: *Platynosomum fastosum*-induced cholangiocarcinomas in cats. Veterinary parasitology, 2012, 190(1), 277-280.
16. Sripa, B, Kaewkes, S, Sithithaworn, P, Mairiang, E, Laha, T, Smout, M, Pairojkul, C, Bhudhisawasdi, V, Tesana, S, Thinkamrop, B, Bethony, JM, Loukas, A, Brindley, PJ: *Liver flukes induces cholangiocarcinoma*. PLoS. Med. 2007, 4, 1148–1155.

17. Ferreira AMR, Almeida ECP. Platinosomose. In: Souza HJM. Coletâneas em Medicina e Cirurgia Felina. Rio de Janeiro: LF Livros de Veterinária Ltda. 2003, 385-393.
18. Bunch SE. Doenças Hepatobiliares no Gato. In: Nelson RW, Couto CG. Medicina Interna de Pequenos Animais. 3ed. São Paulo: Elsevier, 2006, 500–502.
19. Gorgozinho KB, Souja HJM, Neves A, Oliveira AP: Obstrução do ducto biliar comum em gato (*Feliscatus*) parasitado por *Platynosomum concinnum*. Revista Brasileira de Medicina Veterinária 2007, 29:33-36.
20. Basu AK, Charles RA: A review of the cat liver fluke *Platynosomum fastosum* Kossack, 1910 (Trematoda: Dicrocoeliidae). Vet parasitol 2014, 200:1-7.
21. Foley RH: *Platynosomum concinnum* infection in cats. The Compendium on Continuing Education for the Practicing Veterinarian 1994, 16:1271-1277.
22. Carreira VS, Vieira RFC, Machado GF, Luvizotto MCR: Feline Cholangitis/Cholangiohepatitis complex secondary to *Platynosomum fastosum* infection in a cat. Revista Brasileira de Medicina Veterinária 2008, 17:184-187.
23. Sato M, Shibata C, Kikuchi D, Ikezawa F, Imoto H, Sasaki I: Effects of biliary and pancreatic juice diversion into the ileum on gastrointestinal motility and gut hormone secretion in conscious dogs. Surgery 2010, 148:1012–1019.
24. Mehler SJ: Complications surgery of the extrahepatic biliary in pet. Vet Clin North Am Small Anim Prac 2011, 41:949 – 967.
25. Radlinsky MG: Cirurgia do Sistema Biliar Extra-hepático. In: Fossum TW. Cirurgia de Pequenos animais. 4 ed. Roca: São Paulo, 2015, 618-632.
26. Martin RA, Lanz OI, Tobia KM.: Fígado e Sistema Biliar. In: Slatter. Manual de Cirurgia de Pequenos Animais. 3 ed. Manole. Barueri – SP, 2007, 708 – 726.
27. Mehler SJ, Bennett RA. Canine extrahepatic biliary tract disease and surgery. Compend Contin Educ Vet 2006, 20:302-314.

TERAPIJA BILIJARNE OBSTRUKCIJE MAČKE IZAZVANE SA *PLATYNOSOMUM FASTOSUM*, MODIFIKOVANOM METODOM HOLECISTODUODENOSTOME – PRIKAZ SLUČAJA

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Kod mačaka koje boluju od hepatičkih poremećaja, česta su oboljenja žučnih kanala. Holangitis je termin koji se odnosi na sve inflamatorne procese u žučnim kanalićima kod mačaka, a koji može da bude i parazitske etiologije. Obolele životinje moraju da se podvrgnu kliničkom tretmanu ili hirurškom zahvatu, ili se primenjuju oba tretmana. U radu je opisan slučaj obstrukcije bilijarnog trakta kod 10 godina starog mongel mačora, izazvan sa *Platynosomum fastosum*, što se relativno retko sreće u kliničkoj praksi.