

## Cholecystojejunostomy in a Cat with common Bile Duct Obstruction

Lucinéia Costa Oliveira<sup>1</sup>, Dandara Quelho Rosa<sup>2</sup>, Michelle Lussac Silva<sup>1</sup>, So Yin Nak<sup>1</sup>, Bruna Martins Berutti<sup>1</sup>, Maria Eduarda dos Santos Lopes Fernandes<sup>3</sup>, Diefrey Ribeiro Campos<sup>4</sup> & Ricardo Siqueira da Silva<sup>5</sup>

### ABSTRACT

**Background:** Domestic cats are affected by several hepatic diseases, among which biliary tract disorders are the second most common, behind hepatic lipidosis. The causes of those disorders are controversial, but inflammatory diseases are frequently associated with this comorbidity. The diagnosis is realized by laboratory exams and abdominal ultrasonography. A complete obstruction of the biliary tract is a surgical emergency and the desobstruction or deviation of flow must be carried out as soon as possible. Our objective here is to report the clinical pathology findings and the surgical therapy of a biliary duct obstruction in a cat.

**Case:** A 6-year-old male mixed-breed cat with history of chronic rhinosinusitis was treated at the Veterinary Medical Teaching Hospital of Rio de Janeiro Federal Rural University (UFRRJ), presenting prostration, anorexia and jaundice for 4 days. We request laboratory tests (hemograma and liver and kidney biochemical profile) and the hepatic enzymes showed increased. Due to the alterations related to cholestasis the patient underwent abdominal ultrasonography evaluation, which revealed cholangiohepatitis, thickened gallbladder with a large amount of bile sludge, severe extrahepatic bile duct dilatation and the presence of a duodenal papillary mass with approximate diameter of 0.5 cm. Therefore, a bile sample was collected for culture and antibiogram, which isolated *Enterococcus* sp. Furthermore, guided cytology of the mass was performed, which suggested duct hyperplasia and fibrosis. Because the findings indicated serious mechanical obstruction of the extrahepatic bile ducts caused by the duodenal papillary hyperplasia, and due to the negative response to conservative clinical management, the patient was referred for cholecystojejunostomy to divert the bile flow to the small intestine. Also, during the surgery we collected material from the liver, gallbladder, intestine and pancreas for histopathological analysis and culture and antibiogram testing with the objective to diagnosing alterations compatible with the feline triad. There was bacterial development in all the organs collected except the pancreas, supporting the histopathological results, indicating chronic cholecystitis, mild lymphoplasmacytic enteritis, and chronic pericholangitis of the liver, but no alterations in the pancreas. The post-surgical treatment consisted of antibiotic therapy based on the culture and antibiogram results and administration of corticoids. Finally, an esophagostomy tube was placed for correct alimentary management.

**Discussion:** The total obstruction of biliary tract in cats is a serious disease that demands surgical intervention. The causes are diverse, but it commonly attacks felines with inflammatory disease, as in the present case. During the surgery, we attempted to achieve mechanical clearance through retrograde and normograde pinning with urethral tube with no success. Thus, the technique chosen to divert the gallbladder flow to the small intestine was cholecystojejunostomy because it is easier to manipulate and migrate the jejunum to the gallbladder. There were no complications during or after surgery, and the animal did not present recurrence, showing that the technique was efficient at promoting the cat's welfare even with reserved prognosis. The patient survived for 260 days and according to the necropsy died of hyper accurate cardiac failure not related with the cholecystojejunostomy.

**Keywords:** cholecystojejunostomy, biliary flow diversion, domestic cat.

DOI: 10.22456/1679-9216.129092

Received: 15 May 2023

Accepted: 30 October 2023

Published: 10 December 2023

<sup>1</sup>Programa de Residência em Medicina Veterinária; <sup>2</sup>Departamento de Parasitologia Animal; <sup>3</sup>Departamento de Medicina e Cirurgia Veterinária, Instituto de Veterinária; <sup>4</sup>Departamento de Parasitologia Animal & <sup>5</sup>Departamento de Medicina e Cirurgia Veterinária, Programa de Pós-Graduação em Ciências Veterinárias, Universidade Federal Rural do Rio de Janeiro (UFRRJ), Seropédica, RJ, Brazil. CORRESPONDENCE: L.C. Oliveira [lucineiacoliveira@hotmail.com] & D.Q. Rosa [dandar\_a@hotmail.com]. Instituto de Veterinária - UFRRJ. BR-465, km 7. CEP 28.890-000 Seropédica, RJ, Brazil.

## INTRODUCTION

Hepatic disease has a major importance in domestic feline medicine. Gallbladder diseases have high incidence, just behind hepatic lipidosis [2,7]. Among the disorders of the biliary tract, obstruction of the bile ducts is the most common, and in the majority of cases is caused by an inflammatory disease [4,11].

Biliary tract surgery in cats is complex and can result in fatal complications [6]. The mortality rate is high, so surgery is only recommended in cases of total obstruction [13]. In the long run, felines submitted to cholecystoenterostomy may develop cholecystitis, intestinal stenosis, chronic vomiting, diarrhea after exocrine pancreatic insufficiency and ascending cholangiohepatitis [1,10].

Our objective is to report the surgical therapy of common bile duct obstruction caused by duodenal papillary hyperplasia in a domestic cat, offering welfare to the patient, which survived for 260 days and died of hyper accurate cardiac failure.

## CASE

A 6-year-old neutered male mixed breed cat, with serology negative for feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) with history of chronic rhinosinusitis, presenting prostration, anorexia and icterus for 4 days was treated at Veterinary Medical Teaching Hospital of Rio de Janeiro Federal Rural University (UFRRJ). According to the owners, the cat was submitted to broad-spectrum antibiotic therapy once to twice a year due to chronic rhinosinusitis, and almost one month previously, before the icterus, treatment began with famciclovir<sup>1</sup> (Penvir®), which was interrupted because the patient started to present vomiting and hyporexia.

During the physical exam, the patient was prostrate and was found to suffer from icterus and dehydration. The cardiac frequency was 238 beats per min, and there were 44 lung movements per min, both with no auscultate alterations. Rectal temperature was 38.1°C, systolic pressure was 120 mmHg, and moderate pain was noted on abdominal palpation. Due to the hepatic alterations related to cholestasis, we requested laboratory tests (hemogram, liver and kidney biochemical profile and urinalysis) and abdominal ultrasonography. The hemogram indicated normocytic and normochromic anemia, monocytosis, relative eosinopenia, hypoproteinemia and icterus plasma. The biochemical exam showed increases of alanine

aminotransferase (ALT), aspartate aminotransferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (AF), globulins, total cholesterol, triglycerides, fructosamine, total bilirubin (TB), direct bilirubin (DB), and indirect bilirubin (IB), Table 1 (day -8). Urinalysis revealed orange coloration, hematuria, proteinuria, hyalinus, granulated cylinders, degenerated crystals, transitional epithelial cells and bilirubinuria.

The ultrasound<sup>2</sup> evaluation (Siemens P500) evidenced thickened and irregular gallbladder walls, with a lot of bile sludge, distended common biliary duct (approximately 5 mm) and tortuous until the duodenal papilla, where it was possible to see a round mass with approximate diameter of 0.5 cm, which was probably obstructing the biliary duct. Moreover, the liver presented low echogenicity associated with the biliary duct alterations, suggesting cholangiohepatitis. There were no significant alterations in intestine and pancreas. Also, cystitis and chronic kidney alterations on both sides were identified. Because of these alterations, we performed guided cholecystocentesis to collect bile for culture and antibiogram analysis, and guided cytology of the duodenal papillary mass.

Until the culture and antibiogram results were available, treatment started with the following drugs: amoxicillin with clavulanate<sup>3</sup> [amoxicillin with clavulanate - 20 mg/kg, PO, BID, 5 days]; prednisolone<sup>4</sup> [Predsim® - 2 mg/kg, PO, SID, 5 days]; S-adenosyl-methionine<sup>5</sup> [SAME - 90 mg/cat, PO, SID, 5 days]; silibinin<sup>5</sup> [silibinin - 30 mg/kg, PO, SID, 5 days]; and maropitant<sup>6</sup> [Cerenia® - 1 mg/kg, SC, SID, 5 days]. However, no clinical improvement was observed. After 3 days, the culture and antibiogram results were ready, indicating *Enterococcus* sp. resistant to amoxicillin with clavulanate, so the therapy was changed to ceftriaxone<sup>7</sup> [Triaxin® - 25 mg/kg, IV, BID] and metronidazole<sup>8</sup> [Nidazofarma® - 7.5mg/kg, IV, BID].

After 5 days, results of the cytology suggested that the mass in the duodenal papilla was ductal hyperplasia/fibrosis. Due to the negative response to the clinical treatment of the total bile duct obstruction, the patient was submitted to exploratory celiotomy and surgical desobstruction of the duodenal papilla.

The animal remained fasting for 8 h. The pre-anesthesia medication was methadone<sup>9</sup> [Mytedom® - 0,3 mg/kg, i.m.]. After 15 min, with venous access, anesthesia was induced with propofol<sup>9</sup> [Propovan® - 6 mg/kg, i.v.]. The patient was intubated and anesthesia was

maintained with isoflurane<sup>9</sup> (Isoforine®). The patient was in dorsal decubitus and after trichotomy, antisepsis and sterile drape placement, the surgery started with a midline celiotomy from sternum to umbilicus.

It was possible to observe distension of the bile duct and gallbladder and a blood clot on the cranial face (Figure 1A). The surgery technique initially planned was retrograde desobstruction with urethral tube #4<sup>10</sup>. To do this, an enterotomy was performed in the proximal duodenum, 3 cm caudal to the pylorus. After locating the duodenal papilla, which was hyperplastic, an attempt was made to des obstruct it, but was unsuccessful (Figure 1B). Before trying normograde desobstruction, the gallbladder was punctured with a fine needle to collect bile, which was delivered for culture and antibiogram analysis (Figure 1C), and a gallbladder wall fragment was resected for biopsy. After that, the team flushed and emptied the gallbladder to try normograde desobstruction, but it did not work either. Given the impossibility of mechanical desobstruction, we chose to divert the biliary flow to the jejunum by performing a cholecystojejunostomy.

The cholecystojejunostomy started with a linear incision in the antimesenteric border of the jejunum, with approximate length of 4 cm. The gallbladder, incised previously, was mobilized to the jejunum, without tension (Figure 2A). The suture was made with poliglecaprone 3-0<sup>11</sup> thread, in simple continuous pattern between the gallbladder seromucosa and jejunum seromucosa throughout the extension (Figure 2B), followed by cholecystojejunostomy omentalization.

The duodenum enterorrhaphy was performed with nylon 3-0 thread<sup>12</sup>, with a simple interrupted pattern, but beforehand we collected 2 fragments, 1 for culture and antibiogram and the other for histopathological analysis (Figure 3A). Moreover, fragments from liver and pancreas (Figure 3B) were collected for the same purpose.

An abdominal flush was performed with 1000 mL of warm saline solution<sup>10</sup> and then the celiorrhaphy was carried out: the musculature was sutured with nylon 2-0<sup>12</sup> thread in a cruciate pattern. In turn, the subcutaneous suture was performed with poliglecaprone 3-0<sup>11</sup> in a continuous mattress pattern, and in the skin with a simple pattern.

An esophagostomy tube<sup>12</sup> was placed after the surgery because the patient was anorectic. The procedure was successful and the patient remained assisted until total anesthetic recovery.

Immediately after the surgery, the cat was medicated with ceftriaxone<sup>7</sup> [Triaxin® - 25 mg/kg, i.v.] and metronidazole<sup>8</sup> [Nidazofarma® - 7.5mg/kg, i.v.], meloxicam<sup>13</sup> [Maxicam® - 0.05 mg/kg, i.m.] and methadone<sup>9</sup> [Mytedom® - 0.25 mg/kg, i.m.].

The postoperative regimen consisted of ceftriaxone<sup>7</sup> [Triaxin® - 40 mg/kg, s.c, BID, for 15 days], metronidazole [Nidazofarma® - 7.5 mg/kg, via esophagus tube, BID, for 15 days], meloxicam<sup>13</sup> [Maxicam® - 0.03 mg/kg, s.c, SID, for 10 days], omeprazole<sup>5</sup> [omeprazole - 1 mg/kg, via esophagus tube, BID, for 30 days], methadone<sup>9</sup> [Mytedom® - 0.25 mg/kg, i.m., TID, for 4 days], maropitant<sup>6</sup> [Cerenia® - 0.1 mL/kg, s.c., SID, for 5 days] and vitamin K<sup>14</sup> [Monovin K® - 1 mg/kg, s.c., SID for 30 days]. The cat was submitted to fasting for 8 h after surgery, followed by feeding with pasty hypercaloric food.

On the day after surgery, the patient remained stable and after 3 days the improvement in jaundice was noticeable (Figure 4B) compared to the surgery day (Figure 4A). On the same day, a new hemogram and biochemistry profile were performed. The hemogram showed leukocytosis without left shift, neutrophilia, lymphopenia and monocytosis, while in the biochemistry profile, the serum levels of all the hepatic enzymes analyzed decreased in comparison to the pre-surgical exam: ALT, AST, GGT, AF, globulins, TB, DB and IB, Table 1 (Day +3). On the 5<sup>th</sup> day, tramadol<sup>15</sup> [tramadol - 2 mg/kg, s.c, SID, for 7 days], dipyrone<sup>16</sup> [Dipimed® - 25 mg/kg, via esophageal tube, BID, for 7 days], ondansetron<sup>17</sup> [Vonau Vet® - 0.5 mg/kg, via esophageal tube, BID, for 7 days], and marbofloxacin<sup>18</sup> [Marbopet® - 4.2 mg/kg, via esophageal tube, SID, for 30 days] were added to the protocol.

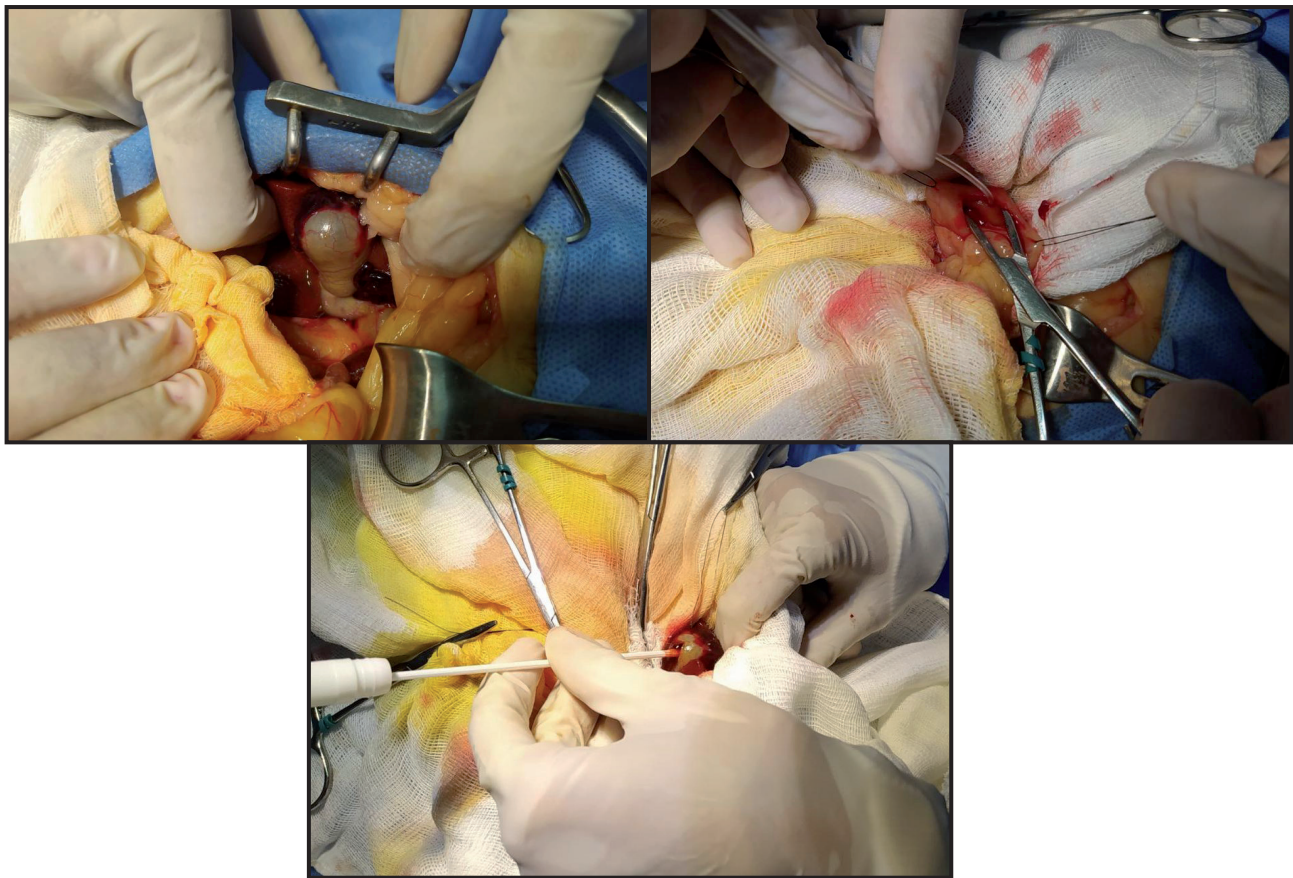
The laboratory exams were repeated 7 days after the surgery. The hemogram showed hypochromic macrocytic anemia, leukocytosis without left shift, neutrophilia, relative lymphopenia and monocytosis. In the hepatic biochemistry profile, serum levels of ALT, AST and AF increased, while GGT decreased. The globulins maintained high levels; TB and IB increased while DB decreased compared to the previous biochemistry profile, as shown in Table 1 (Day +7). Due to the laboratory results, SAME [S-adenosylmethionine - 90 mg/cat, v.o., SID] was prescribed again.

After 8 days of the surgery, the patient started to eat pasty food spontaneously. An ultrasound review was performed and focal peritonitis was identified in



**Table 1.** Biochemistry profile before surgery (Day -8), 3 days, 8 days, 11 days, 18 days and 153 days after surgery (Day 0).

Parameter	Day -8	Day +3	Day +7	Day +11	Day +18	Day +153
ALT (U/L)	1115	211	295	715	410	298
AST (U/L)	535	125	168	369	139	107
GGT (U/L)	15	9	8	5	3	1
AF (U/L)	425	262	369	245	92	43
TB (mg/dL)	13.88	4.66	5.55	1.77	2.27	0.16
DB (mg/dL)	7.24	1.94	0.81	0.46	0.38	0.1
BI (mg/dL)	6.64	2.72	4.74	1.31	1.89	0.06



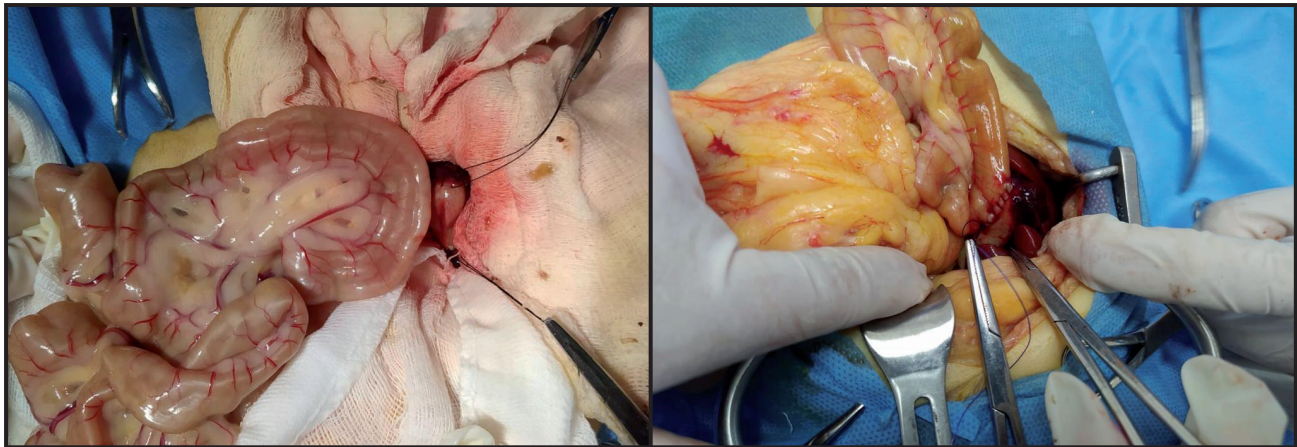
**Figure 1.** A- Biliary duct analysis and observation of distension in common biliary duct and gallbladder, which presented a blood clot in the cranial face. B- Attempted retrograde desobstruction of the common biliary duct with urethral probe number 4. C- Bile collected with swab for culture and antibiogram analysis.

the bile flow deviation, with a small volume of free intraperitoneal fluid, reduced size of the pancreatic parenchyma, but liver still with signs of cholestasis.

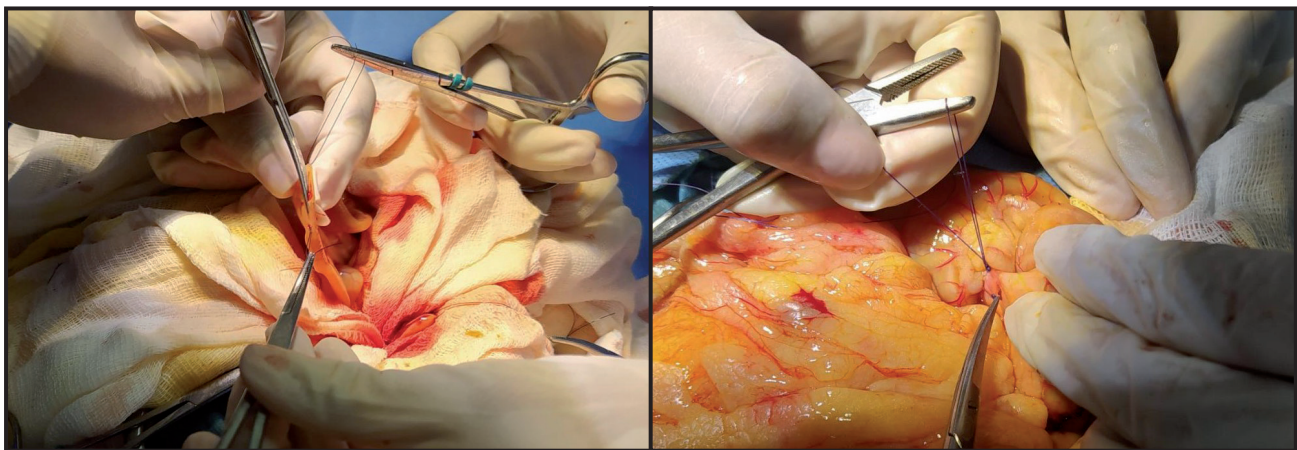
After 10 days of the surgery, prednisolone<sup>4</sup> was administered [Predsim® - 2 mg/kg, v.o., SID, for 15 days]. The bile, pancreas, duodenum and gallbladder culture and antibiogram showed growth of *Enterococcus* sp., while *Proteus mirabilis* was found in the liver. Both were sensitive to marbofloxacin. The his-

topathological analysis identified lymphoplasmacytic enteritis in the duodenum, chronic pericholangitis in the liver, chronic cholecystitis in the gallbladder and no alterations in the pancreas.

Eleven days after surgery, the hemogram revealed leukocytosis without left shift, eosinopenia and relative lymphopenia. In the biochemical profile, the serum levels of ALT and AST increased, while GGT and globulin levels were normalized and TB, DB and



**Figure 2.** A- Approximation of gallbladder to jejunum. B- Suture in simple continuous pattern with poliglecaprone 3-0 thread between the gallbladder seromucosa and the jejunum seromucosa to for flow diversion.



**Figure 3.** A- Material collected from duodenum for biopsy. B- Material collected from pancreas for biopsy

IB decreased, Table 1 (Day +11). Fifteen days after surgery, the suture was removed.

Eighteen days after surgery, new hemogram, biochemical profile and abdominal ultrasonography were performed. The ultrasound revealed enlarged liver, coarse echotexture, no duct dilatation, reduction of peritonitis, cystitis and low volume of free intraperitoneal fluid that was undrainable. The hemogram showed neutrophilia, lymphopenia and hyperproteinemia. In the biochemical profile, ALT and IB decreased while AST, TB and DB increased and GGT continued normal, Table 1 (Day +18). The esophagus tube was removed 20 days after surgery.

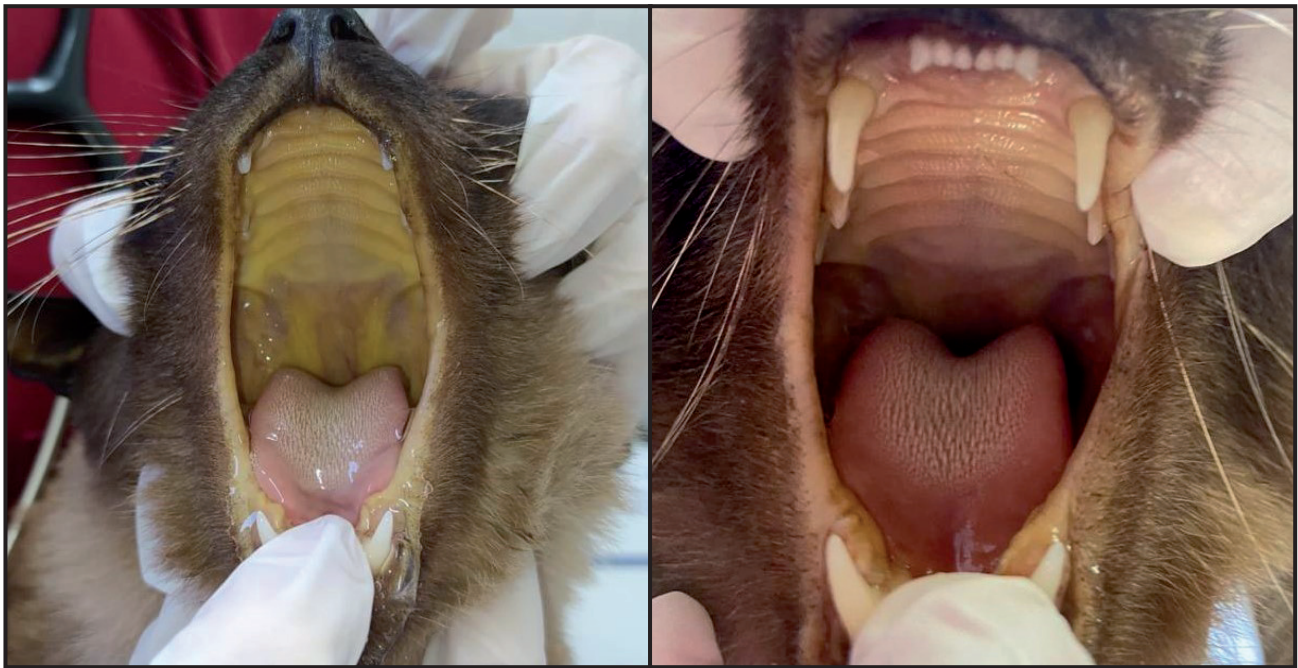
Five months later, new hemogram and biochemical profiling were carried out to monitor the cat. The hemogram did not show relevant alterations. In the biochemical profile, ALT, AST and globulins were still slightly increased while GGT and albumin were slightly decreased and TB, DB and IB were normalized, Table 1 (Day +153).

The patient did not present clinical signs of biliary duct obstruction after the surgery, with only sporadic vomiting (1 to 2 vomiting episodes/month). The cat died 260 days after surgery and according to necropsy, the cause was hyper accurate cardiac failure, unrelated to the cholecystojejunostomy.

#### DISCUSSION

Gallbladder obstruction is a serious disease that requires emergency surgical intervention [14]. The causes are diverse, but it usually affects cats with inflammatory disease [10]. This is supported by the case reported here, where the histopathological diagnosis showed enteritis, pericholangitis and chronic cholecystitis. Although pancreatitis is frequently associated with cholangitis and enteritis [10], the patient in this case did not present histopathological alterations in the pancreas, although this did not exclude the possibility of feline triad disease, since the fragment collected did not present inflammation. Nonetheless, lymphocytic





**Figure 4.** A- A 6-year-old neutered male mixed breed cat received at the clinical surgical service of the veterinary hospital of Rio de Janeiro Federal Rural University, with icterus on the day of surgery (Day 0). B- Cat showing postoperative clinical improvement (Day +3).

inflammation was confirmed in other organs (intestine, bile ducts and gallbladder).

In microbiological tests, bacterial infection was confirmed in all organs. In the liver and bile ducts, bacteria from intestinal microbiota have often been isolated [5,7,8,15]. The culture of the patient's liver indicated *Proteus mirabilis* growth, unlike to the cases reported previously [3,5,7,8,15]. However, this species is common in the intestinal microbiota. *Enterococcus* sp. was found in bile ducts, gallbladder, duodenum and pancreas, corroborating with previous findings [3].

Infections are commonly described in cholangiohepatitis due to the biliary duct anatomy particularity in cats, promoting ascending bacterial migration [1]. The patient had a history of chronic rhinosinusitis, frequently treated with antibiotics, which are mainly hepatically metabolized, in addition to famciclovir (Penvir®). The treatment was suspended because the cat started to vomit and had hyporexia. One of the possibilities is that the drugs used in rhinosinusitis treatment could predispose patients to hepatic and gastrointestinal alterations, causing vomiting, and due to the anatomy of cats common ducts, ascending infection can occur of the bile ducts, pancreas and liver. Those infections can provoke an immune response, evolving to chronic inflammation, bile duct obstruction and the clinical signs observed in our patient. The cat presented icterus, vomiting and inappetence when

the obstruction was diagnosed. But many animals are not icteric in the begging of bile duct obstruction [12], suggesting that our patient's biliary flow obstruction was at an advanced stage.

The increased hepatic enzymes in this case, associated with ultrasonography, which indicated a mass obstructing the bile duct, were sufficient to diagnose the total mechanical obstruction of the extrahepatic biliary duct, requiring surgery.

When possible, duct desobstruction should be prioritized, followed by bile flow diversion if unsuccessful, using cholecystoduodenostomy to reduce the risk of ulceration in the duodenum [12]. In spite of that, our surgical team chose to perform cholecystojejunostomy because the advantages when compared to cholecystoduodenostomy, such as easier manipulation and the jejunum migration to the gallbladder, reducing surgery time and the risk of hypotension caused by manipulation and tension of the gallbladder, as well as reducing the chances of bile reflux [9,12]. The suture between the gallbladder and jejunum in this case was not performed in two layers as recommended by Radlinsky [14], since the gallbladder had a blood clot that made it impossible to separate the serosa and mucosa to adjust the stoma size (to approximately 3 to 4 cm). The blood clot in gallbladder was caused by the puncture previously made to collect bile for culture and antibiogram analysis.

The surgical team decided to suture the seromucosa with the stoma, respecting the dimensions recommended by Radlinsky [14], to avoid stenosis. The technique has shown efficiency in biliary flow diversion, promoting well-being and life quality to patients. In our case, no serious consequences were observed. The hepatic enzyme levels decreased significantly 158 days after the surgery, and even though ALT and AST were not normalized, the specific cholestasis enzymes, such as bilirubin, AF and GGT, normalized, indicating efficient resolution of the extrahepatic biliary duct obstruction. The hepatic lesions before the surgery along with the drugs used during and after surgery can cause alterations to hepatocytes, explaining the high levels of nonspecific enzymes after treatment. However, the TB concentration improved, so it is possible to assume that the hepatic function improved.

The post-operative abdominal ultrasonography indicated focal peritonitis in biliary flow diversion and free intraperitoneal fluid. The focal peritonitis was caused by the inflammatory reaction to suture and the free fluid was associated with abdominal flush during surgery. Both alterations are common and regressed over time.

Extended antimicrobial therapy after surgery is very important, since the majority of cases have positive bacterial culture [12]. The chosen antibiotic must have a broad spectrum based on the bacteria that colonize the gastrointestinal tract identified in the culture and antibiogram results [4]. Marbofloxacin was prescribed for 30 days and all the isolated microorganisms were sensitive to it, increasing the surgical treatment's success. The use of anti-inflammatory doses of corticoids is recommended [4]. Prednisolone at 0.5 mg/kg

dose was prescribed to reduce the inflammation in the biliary tract and intestine, and proved to be efficient according to the ultrasound results.

The bile flow diversion prognosis is always reserved [4,13]. The cat in this case was relatively healthy, and when showing clinical and ultrasonographic signs of cholangitis, normal treatment was administered. The cat lived for 260 days after surgery, dying of hyper accurate cardiac failure according to the necropsy report.

#### MANUFACTURERS

<sup>1</sup>Novamed Fabricação de Produtos Farmacêuticos Ltda. Manaus, AM, Brazil.

<sup>2</sup>Siemens Medical Solutions USA Inc. Mountain View, CA, USA.

<sup>3</sup>Multilab Indústria e Comércio de Produtos Farmacêuticos Ltda. Jaguariúna, SP, Brazil.

<sup>4</sup>Brainfarma Indústria Química e Farmacêutica S.A. Anápolis, GO, Brazil.

<sup>5</sup>DrogaVET. Rio de Janeiro, RJ, Brazil.

<sup>6</sup>Inovat Indústria Farmacêutica Ltda. Guarulhos, SP, Brazil.

<sup>7</sup>Momenta Farmacêutica Ltda. São Paulo, SP, Brazil.

<sup>8</sup>Farmace Indústria Químico-Farmacêutica Cearense Ltda. Barbalha, CE, Brazil.

<sup>9</sup>Cristália Produtos Químicos Farmacêuticos Ltda. São Paulo, SP, Brazil.

<sup>10</sup>Medsonda Indústria e Comércio de Produtos Hospitalares Descartáveis Ltda. Arapoti, PR, Brazil.

<sup>11</sup>Bioline Comercial Ltda. São Paulo, SP, Brazil.

<sup>12</sup>Ace Indústria e Comércio Ltda. Goiânia, GO, Brazil.

<sup>13</sup>Ourofino Saúde Animal Ltda. São Paulo, SP, Brazil.

<sup>14</sup>Laboratório Bravet Ltda. Rio de Janeiro, RJ, Brazil.

<sup>15</sup>HalexIstar Indústria Farmacêutica S.A. Goiânia, GO, Brazil.

<sup>16</sup>Medquímica Indústria Farmacêutica Ltda. Juiz de Fora, MG, Brazil.

<sup>17</sup>Biolab Sanus Farmacêutica Ltda. Bragança Paulista, SP, Brazil.

<sup>18</sup>Sespo Indústria e Comércio Ltda. Paulínia, SP, Brazil.

**Declaration of interest.** The authors alone are responsible for the content and writing of the paper. The authors report no conflicts of interest.

#### REFERENCES

- Buote N.J., Mitchell S.L., Penninck D., Freeman L.M. & Webster C.R.L. 2006.** Cholecystoenterostomy for treatment of extrahepatic biliary tract obstruction in cats: 22 cases (1994–2003). *Journal of the American Veterinary Medical Association*. 228(9): 1376-1382. DOI: 10.2460/javma.228.9.1376
- Center S.A. 2009.** Disease of the gallbladder and biliary tree. *Veterinary Clinics of North American: Small Animal Practice*. 39(3): 543-598. DOI: 10.1016/j.cvsm.2009.01.004
- Edwards M. 2004.** Feline cholangiohepatitis. *The Compendium of Continuing Education for the Practicing Veterinarian*. 26(11): 855-861.
- German A. 2009.** Colangite felina. *Veterinary Focus*. 19(2): 41-46.
- Harvey A.M. & Greeffydd-Jones T.J. 2010.** Feline Inflammatory Liver Disease. In: Ettinger S.J. & Feldman E.C. (Eds). *Textbook of Veterinary Internal Medicine – Diseases of the Dog and the Cat*. 7th edn. St. Louis: Elsevier Saunders, pp.1643-1648.
- Hespanha A.C.V., Silvestre A.C.S., Tosato G.S. & Garcia J.N.N. 2018.** Colecistoduodenostomia devido a obstrução total de ducto biliar comum em felino: relato de caso. *Veterinária em Foco*. 15(2): 38-46.

- 7 **Johson S.E. 2004.** Hepatopatias crônicas. In: Ettinger S.J. & Feldman E.C. (Eds). *Tratado de Medicina Interna Veterinária*. 5.ed. São Paulo: Manole, pp.1369-1398.
- 8 **Johnson S.E. & Shering R.G. 2008.** Doenças do Fígado e Trato Biliar. In: Birchard S.J. & Sherding R.G. (Eds). *Manual Saunders de Clínica de Pequenos Animais*. 3.ed. São Paulo: Roca, pp.765-829.
- 9 **Lehner C & McAnulty J. 2010.** Management of extrahepatic biliary obstruction: a role for temporary percutaneous biliary drainage. *Compendium Continuing Education for Veterinarians*. 32(9): E1-E10.
- 10 **Mayhew P.D., Holt D.E., McLearn R.C. & Washabau R.J. 2002.** Pathogenesis and outcome of extrahepatic biliary obstruction in cats. *Journal of Small Animal Practice* 43(6): 247-253. DOI: 10.1111/j.1748-5827.2002.tb00067.x
- 11 **Mayhew P.D. & Weisse C.W. 2008.** Treatment of pancreatitis-associated extrahepatic biliary tract obstruction by choledochal stenting in seven cats. *Journal of Small Animal Practice* 49(3): 133-138. DOI: 10.1111/j.1748-5827.2007.00450.x
- 12 **Mehler S.J. & Bennett R.A. 2006.** Canine extrahepatic biliary tract disease and surgery. *Compendium Continuing Education for Veterinarians* 20(4): 302-314.
- 13 **Nelson R.W. & Couto C.G. 2006.** *Medicina Interna de Pequenos Animais*. 2.ed. Rio de Janeiro: Elsevier, pp.531-533.
- 14 **Radlinsky M.G. 2004.** Cirurgia do sistema biliar extra-hepático. In: Fossum T.W. (Ed). *Cirurgia de Pequenos animais*. 4.ed. Rio de Janeiro: Elsevier, pp.618-632.
- 15 **Richter K.P. 2005.** Doenças do Fígado e do Sistema Hepatobiliar. In: Tams T.R. (Ed). *Gastroenterologia de Pequenos Animais*. 2.ed. São Paulo: Roca, pp.283-348.