CASE REPORT:

SURGICAL MANAGEMENT OF FELINE SUPPURATIVE CHOLELITHIASIS

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Case Presentation

A 15 year old neutered male domestic shorthair was presented with a one week history of progressive vomiting, decreased appetite, and lethargy. The cat had been taking prednisone (5mg PO q24h) for five years to treat previously diagnosed atopy. Physical exam identified moderate dehydration, palpable thyroid enlargement (Left 3/6; Right 0), dental tartar and gingivitis, slightly underweight body condition (BCS=4/9) with moderate generalized muscle atrophy with prominent spine and hips. The cat also had a persistent heart murmur (grade 2/6) which was first identified four years previously and was not under treatment. Mild abdominal pain was present on abdominal palpation as evidenced by vocalization and splinting of the abdomen.

Diagnostics

Initial chemistry profile, complete blood count, and thyroxin level were performed on our Abaxis in-house lab equipment and pertinent values are listed in **Table 1**. Interestingly, hepatocyte leakage enzyme (alanine aminotransferase) and total bilirubin were normal in this cat. The lack of bilirubin elevation supports the absence of extrahepatic biliary obstruction (EHBO); however, ultrasound is a more sensitive screening tool for EHBO.[1] Serum cholesterol was normal in this patient, which is consistent with previous findings, where 95% of cats with EHBO had normal serum cholesterol assays.[2] This is worth noting since, in dogs, hypercholesterolemia is a common finding with EHBO.

Two-view orthogonal abdominal radiographs revealed a collection of numerous rounded mineral densities in the right cranial abdomen, consistent with the region of the gallbladder. See **Figure 1**.

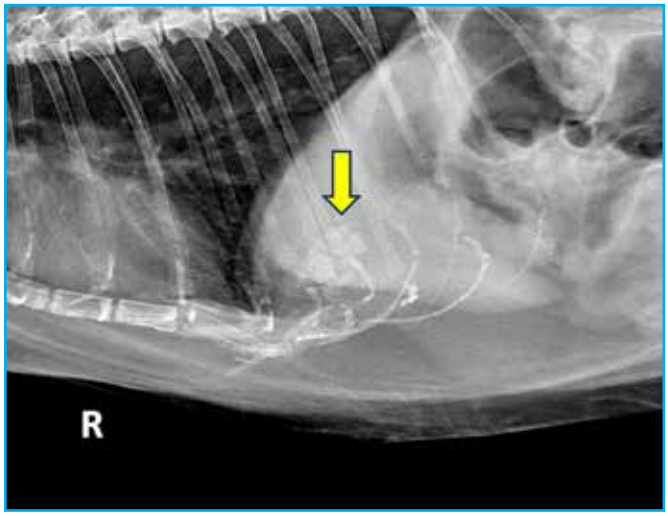

Figure 1. Lateral abdominal radiograph shows the presence of mineral opacity rounded densities (arrow) in the cranial ventral abdomen.

Table 1. Pertinent results of chemistry profile, complete blood count, and thyroxin testing.

Analyte	Patient's value	Reference range	Low, normal, high
Alanine aminotransferase	49 U/L	20-100 U/L	Normal
Alkaline phosphatase	26 U/L	10-90 U/L	Normal
Total bilirubin	0.3 mg/dL	0.1-0.6 mg/dL	Normal
White blood cell count	9.06 10^9/L	5.5-17.0 10^9/L	Normal
Hematocrit	29%	28-44%	Low normal
Thyroxin (T4)	3.2 ug/dL	1.5-4.8 ug/dL	Normal

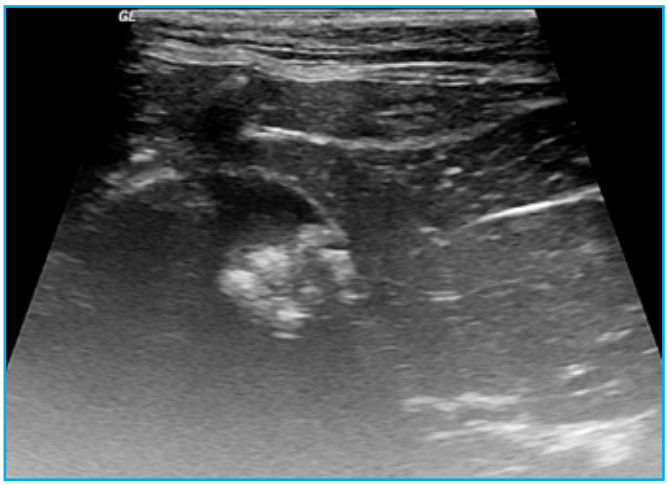

Figure 2. Ultrasound image showing echogenic shadowing choleliths.

Thoracic radiographs were normal. Abdominal ultrasound identified a cluster of convex echogenic bodies within the gallbladder. See **Figure 2**. The gallbladder itself appeared otherwise normal, and the bile duct was unremarkable (i.e. there was an absence of distention and tortuosity). The remainder of the abdominal ultrasound did not uncover other abnormalities. Therefore, there was no supportive evidence for EHBO on imaging.

Surgical Treatment

The decision to take this cat to surgery was based primarily on the following: the acute development of clinical signs (vomiting, lethargy, poor appetite), possible risk to develop EHBO if left untreated, potential for better outcome with earlier intervention,[3] and the opportunity to biopsy and culture intra-abdominal tissues.

Prior to surgery, the cat was hospitalized and received an analgesic (buprenorphine sustained release 0.20 ml given SC) and intravenous fluids overnight. Fluids were given judiciously due to the presence of a heart murmur, but they were considered beneficial pre-operatively given the clinically evident dehydration.

The following day, the cat went to surgery for abdominal laparotomy. The surgery for this particular patient focused on the gallbladder and liver to minimize anesthetic time and associated morbidity. If it were not for anesthetic considerations, it would have been reasonable to also biopsy the pancreas and small bowel in this patient. During surgery, an enlarged but intact gallbladder was identified. Choleliths were palpable within the gallbladder; however, choledocholiths (within the bile duct) were not identified and the bile duct appeared grossly normal – all consistent with the ultrasound findings.

A cholecystectomy was performed. The gallbladder was gently and bluntly dissected from its hepatic fossa and hepatic attachments. There was mild hemorrhage from the hepatic tissue which was successfully managed with application of a hemostatic powder. See **Figure 3**.

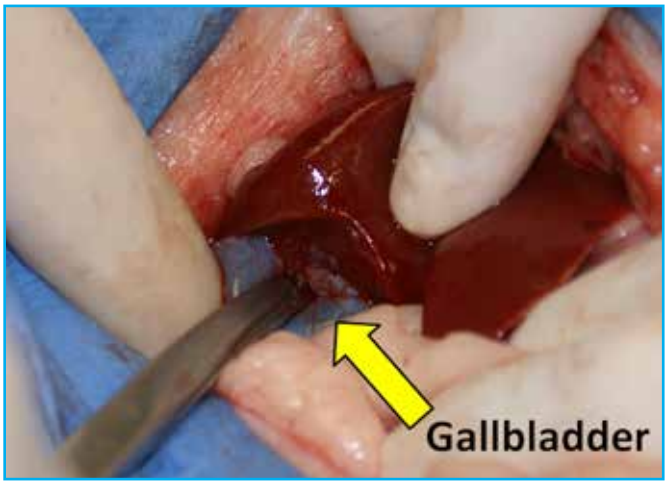

Figure 3. Intra-operative photo of gall bladder dissection to free from the liver.

This blunt dissection with Metzenbaum scissors was continued until the gall bladder was completely free from hepatic parenchyma. At that point, mobilization of the gallbladder allowed good visualization of the bile duct. Hemoclips were applied to the bile duct and cystic artery to achieve complete occlusion of both. See **Figure 4**.

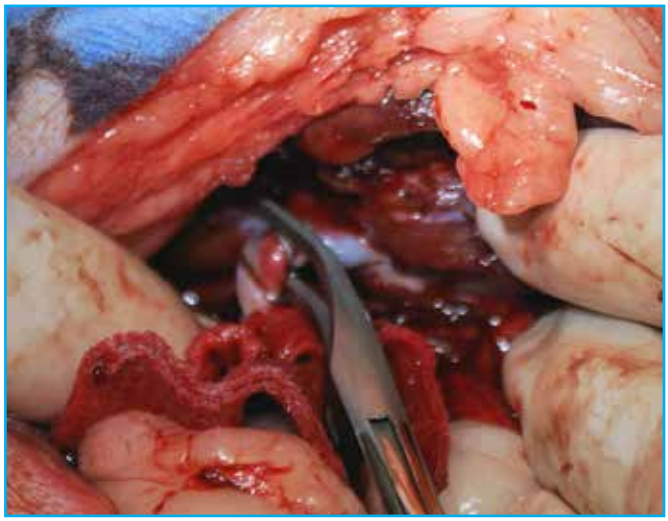

Figure 4. Intra-operative photo of Hemoclips on the bile duct prior to transection.

A hemostat was applied more closely to the fundus of the gall bladder to allow an approximately 3mm section in which to sharply transect the bile duct and cystic artery and excise the gallbladder en block. The area around the site of transection was gently packed with a gauze square prior to

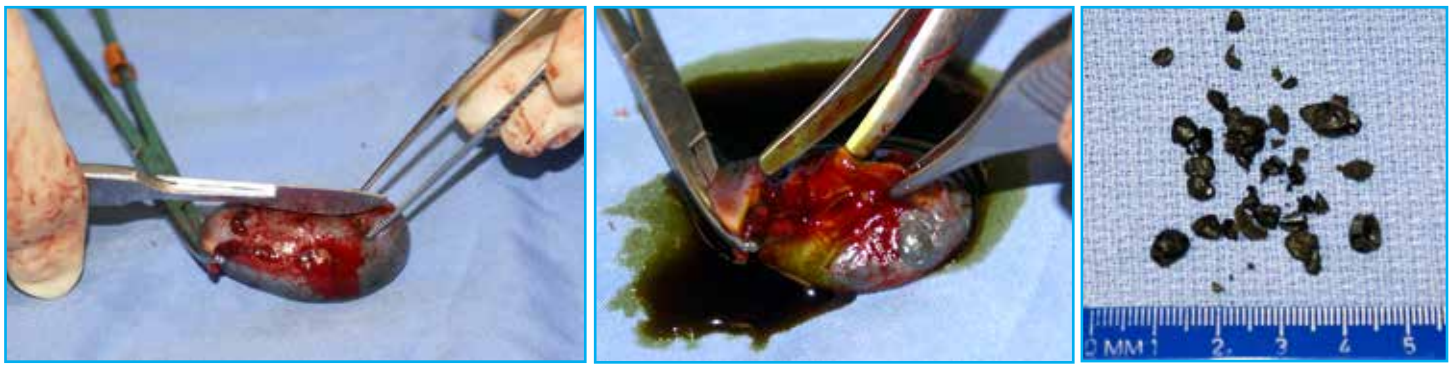

Figure 5 a, b, c. After gallbladder excision. The gallbladder is opened to view the numerous dark choleliths inside.

transection to minimize contamination of the abdomen with bile and its associated components and, thus, prevent bile peritonitis. Fortunately, seepage of bile was not an issue in this patient at the time of transection, but gauze placement was considered a safe-guard.

The liver had rounded margins, but, otherwise, there was no gross areas of abnormality; therefore, a single wedge biopsy of was taken from an easily accessible liver margin. This biopsy site was closed with 4-0 polydioxanone in a single horizontal mattress suture.

After excision cholecentesis was performed to obtain a sample of biliary contents for aerobic and anaerobic culture and sensitivity. The gallbladder was then sharply incised and the contents were examined. See **Figure 5**. The liver and gall bladder were submitted for histopathology. Due to financial constraints, the choleliths were not submitted for analysis. We speculate that a calcium component is likely given the radio-opacity of these stones.

Histopathologic Tissue Analysis

- Gallbladder: Multiple gall stones. Epithelial hyperplasia and minimal lymphoplasmacytic inflammation.
- Liver: Diffuse, moderate, periportal, lymphoplasmacytic hepatitis, and multifocal neutrophils.

The biggest challenge in this case, and others, may be determining causality. As Dr. Center put it, "It is clear that disease or stasis of bile flow involving the gallbladder and major bile ducts can lead to cholelithiasis as surely as cholelithiasis can injure these structures leading to bile stasis."[1] Therefore, the hepatitis identified in this case may be secondary to choleliths or part of their pathogenesis. Also, remember in this particular patient that inflammatory changes may be dampened by the administration of corticosteroids.

Microbiology: Culture and Sensitivity Results

From the submitted cholecentesis sample, two organisms were cultured: (1) non-hemolytic Streptococcus and (2) Enterococcus sp. Both isolates were sensitive to amoxicillin and marbofloxacin, which the cat had already been prescribed post-operatively. Both of these organisms have been previously reported in bile samples from cats with cholelithiasis[4] and EHBO.[3] The role of bacteria in the pathogenesis of choleliths is not clear at this time.

Outcome

Post-operatively, the cat remained in the hospital for three days. Treatment consisted of fluids, an anti-nausea medication (maropitant), antibiotics (ampicillin and enrofloxacin), and nutritional supportive via syringe feeding. He was discharged on oral marbofloxacin, amoxicillin, (three week course each of antibiotics) and Denamarin. The owner declined placement of an esophagostomy tube at the time of surgery, but one week post-operatively due to poor appetite at home, one was placed. Note that a previous study found poor outcomes in two cats with concurrent hepatic lipidosis and cholelithiasis.[4] Therefore, if hepatic lipidosis is not present initially, aggressive nutritional support is indicated in these patients to prevent its development.

Sixteen days post-operatively, the owner reported several episodes of vomiting over the preceding two days. The cat was re-evaluated and radiographs confirmed proper position of the esophagostomy tube. A chemistry panel at that time was also normal, showing no evidence of azotemia, normal hepatic enzymes, and normal total bilirubin. A feline pancreatic lipase immunoreactivity level (fPLI) was performed, and results were equivocal for pancreatitis with a value of 4.0 ug/L (normal <3.5ug/L, abnormal >5.4ug/L). The antiemetic maropitant was added to the treatment regimen. Esophagostomy tube feedings were given more frequently in smaller amounts, and medications were given

via the esophagostomy tube. The owner reported modest improvement in energy level and decrease, but not resolution, of vomiting with these alterations.

At twenty nine days post-operatively, the cat relapsed into frequent vomiting and became lethargic again. Antibiotic course had already been completed at this point. Denamarin was discontinued, on the chance it was the cause of emesis, the maropitant dose frequency was increased to twice daily. At thirty-two days post-operatively, the owners elected euthanasia due to intractable vomiting and profound lethargy.

Discussion

Presentation of cholelithiasis as biliary colic in cats is uncommon, as most cases are asymptomatic. In fact, cholelithiasis is often an incidental finding on ultrasound and may not warrant aggressive intervention in outwardly healthy cats.[1,3] However, when present, the most common symptoms for cats with choleliths are progressive vomiting, dehydration, anorexia, icterus, lethargy, and possibly abdominal pain.[1,3] Icterus in association with cholelithiasis could be due to extra-hepatic biliary obstruction, and this finding should be an impetus for ultrasound and possible surgical intervention.

This patient had numerous choleliths present. This is consistent with previous feline studies[4] and canine studies.[5] Depending on composition, choleliths can be either radiodense or radiopaque, but all are hyperechoic on ultrasound. The most common cholelith location is the gallbladder, and these are termed cholecystoliths. Also recognized, are choledocholiths, which can be present in the common bile duct, hepatic, and interlobular bile ducts.[1]

If cholelith analysis is performed, stones can be classified based on their components. Most small animal choleliths contain either calcium carbonate (aka "brown-pigment" stones) or bilirubin polymers (aka "black-pigment" stones), and less commonly cholesterol crystallization.[1] Mixed stones are possible as well.[4] This is opposed to humans, where cholesterol containing stones are most prevalent. Calcium containing brown-pigment stones are frequently associated with bacterial infections and biliary stasis.[1]

Surgical complications to be cognizant of when considering hepatobiliary surgery include (1) induction of bile peritonitis and (2) inadvertent ligation of pancreatic duct during cholecystoduodenostomy or cholecystojejunostomy, which results in exocrine pancreatic insufficiency.[4] Recall that the feline common bile duct fuses with the pancreatic duct before opening into the duodenum, and this is speculated to account for the concurrence of pancreatic and biliary disease.[3] Beyond clinical signs, this is another reason why an fPLI was reasonable to perform in this patient.

Finally, for any cat with cholelithiasis, the clinician should screen for hemolytic anemia as a possible underlying etiology. Chronic hemolytic anemia has been linked to bilirubin cholelithiasis in humans[6,7] and a cat. The case report in a cat[8] documents genetic pyruvate kinase (PK) deficiency as the underlying cause of chronic hemolysis; Abyssinian and Somali breeds are predisposed to this disease.

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