

**Signalment:**

"Maggie"

11 year old FS Toy Poodle

6/9 BCS

History:

Maggie had a two week history of decreased appetite and weight loss. She presented to her primary veterinarian with a one day history of lethargy, refusal of food and water, and vomiting. She was administered Cerenia and Convenia SQ. Bloodwork was submitted, revealing elevated cholestatic enzymes, leukocytosis with a neutrophilia, monocytosis, hyperkalemia, and an increased Na/K ratio (WBC 22.9, NEUT 19694, MONO 916, PLATELET 577, ALT 271, ALP 662, GGT 81, CHOL 432, NA 145, K 5.9, NA/K 25, MG 1.4, T4 0.8). Maggie was brought back to her primary veterinarian the following day and dexamethasone SP was administered IV for presumed hypoadrenocorticism. Elevated pre and post values (Pre 14.8, Post 26.2) were present on ACTH simulation test, ruling out hypoadrenocorticism. Baytril was administered SQ and the patient was referred to VMSG for further evaluation.

Clinical Exam:

Maggie's physical examination was unremarkable except for mild dental calculus and gingivitis, a soft II/VI left systolic cardiac murmur, and soft freely movable subcutaneous abdominal masses previously aspirated as lipomas by her primary veterinarian.

Diagnostic Imaging:

Three-view thoracic radiographs: Unremarkable

Abdominal ultrasound: The bile appeared inspissated, however it did not appear to adhere to the mucosal surface of the gallbladder. The gallbladder wall was severely thickened. There was a mild amount of free fluid in the region of the gallbladder and liver. The duodenum was fluid-filled with poor motility.

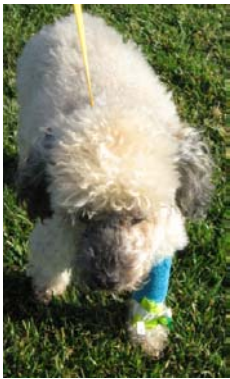


Figure 1



Figure 2



Figure 3

Diagnosis:

Gallbladder mucocele

Treatment/Management:

The patient was hospitalized and placed on IV Norm-R, famotidine, enrofloxacin, and metronidazole as well as SQ Cerenia and hydromorphone as indicated for pain. On in-house bloodwork the following morning there was evidence of progression of cholestatic disease with resolution of the hyperkalemia (ALP 1643 ALT 159 K 3.6). Maggie was premedicated with hydromorphone and atropine. Anesthesia was induced with etomidate and midazolam and maintained with isoflurane and fentanyl. A bupivacaine line block was performed. On abdominal exploration there was a small amount of free abdominal fluid in the cranial abdomen and a distended cystic duct and gallbladder were present. There was no evidence of gallbladder perforation despite adhesions to the lesser omentum and falciform fat. The wall of the gallbladder was thickened. An incision was made in the duodenum and a 5 French red rubber catheter was passed through the major duodenal papilla to confirm patency of the common bile duct. The duodenal incision was closed. An incision was made in the gallbladder, which contained thick, gelatinous bile and was evacuated with suction. The cystic duct was ligated and the gallbladder was removed. Maggie recovered from anesthesia without complications.

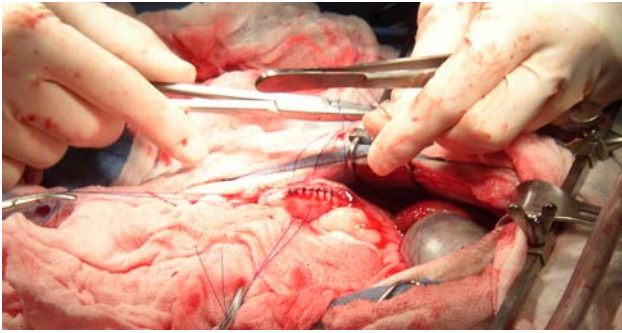


Figure 4



Figure 5

Post-operative care:

Post-operatively, Maggie was maintained on hydromorphone or bupivacaine for pain control, Norm-R qs 20mEq KCl, famotidine, Cerenia, metoclopramide, enrofloxacin, and metronidazole. Maggie was discharged with instructions for enrofloxacin, metronidazole, and Vitamin A administration, suture removal, and activity restriction. A low dose dexamethasone suppression test was recommended two weeks after surgery. On histopathology a gallbladder mucocele with locally extensive chronic active fibrosing and granulating lymphoplasmacytic suppurative fibrinohemorrhagic cholecystitis and hepatitis were diagnosed. Liver and gallbladder cultures were negative. A low dose dexamethasone suppression test performed a month after discharge was consistent with hyperadrenocorticism which was not treated due to lack of clinical signs.

Discussion:

Gallbladder mucoceles are conglomerations of inspissated bile which form a stellate, or “kiwi” pattern on ultrasound and can result in an extrahepatic bile duct obstruction. They are thought to be secondary to mucosal hyperplasia and biliary stasis, and can be associated with cholecystitis with or without bacterial infection. Cocker spaniels are overrepresented, [1, 5] as are Shetland Sheepdogs [7]. Hypothyroidism has also been associated with mucocele formation, potentially causing a delay in gallbladder emptying. [2] An association of gallbladder mucoceles with liver disease, pancreatitis, or hyperadrenocorticism have not been proven [6]. Once thought to be rare, they have become a common cause of extrahepatic biliary disease [8].

In patients where a biliary mucocele is found incidentally, medical management with antibiotics, cholergectics, and hepatoprotectants with recheck ultrasonic evaluation is appropriate as long as the client understands the potential risks [8]. In patients with clinical signs (vomiting, anorexia, lethargy, pyrexia) or biochemical abnormalities (elevated bilirubin, ALP, GGT, AST), surgery is indicated [1], although there have been reports of patients with clinical signs which were successfully managed medically [3].

About 50% of dogs will have a loss of gallbladder wall integrity or rupture. Ultrasound has a 85.7% sensitivity for diagnosis of rupture [1], most commonly identified as echogenic pericyclic or diffuse echogenic peritoneal fluid. There is a greater likelihood of gallbladder wall necrosis and rupture if bacterial cultures are positive, however, infection is rarely associated with gallbladder mucoceles [5]. The most common type of bacteria isolated from gallbladder mucoceles are gram negative anaerobes, likely originating from the gastrointestinal tract [8].

Although biliary surgery has a high rate of mortality in dogs, the majority of losses are in the immediate post-operative period; studies have found that mortality rate within the first 14 days of a cholecystectomy is 22-40% [8]. Dogs that are discharged from the hospital following surgery for a gallbladder mucocele have an excellent prognosis; one and two year survival rates have been reported to be 66% each. An increased risk of death has been associated with any one of the following: a higher GGT, BUN, bilirubin, or phosphorus, increasing age, biliary diversion procedures, elevated pre-anesthetic heart rate, or pancreatitis [4].

References:

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- 2) Pike FS, Berg J, King NW, Penninck DG, Webster RL. Gallbladder mucocele in dogs: 30 cases (2000-2002). *J Am Vet Med Assoc.* May 2004;224(10):1615-22.
- 3) Walter R, Dunn ME, d’Anjou MA, Lecuyer M. Nonsurgical resolution of gallbladder mucocele in two dogs. *J Am Vet Med Assoc.* 2008 Jun 1;232 (11): 1688-93.
- 4) Amsellem PM, Seim HB, Seim HB 3rd, MacPhail CM, Bright RM, Twedt DC, Wrigley RH, Monnet E. Long term survival and risk factors associated with biliary surgery in dogs: 34 cases (1994-2004). *J Am Vet Med Assoc.* November 2006;229(9):1451-7.
- 5) Crews JC, Feeney DA, Jessen CR, Rose ND, Matisse I. Clinical, ultrasonographic, and laboratory findings associated with gallbladder disease and rupture in dogs: 45 cases (1997-2007). *J Am Vet Med Assoc.* February 2009; 234 (3): 359-366.
- 6) Wells, Andrea. Gallbladder Disease. Santa Barbara and Ventura Veterinary Medical Association lecture, 2/12/2009
- 7) Aguirre, et al. Gallbladder disease in Shetland Sheepdogs: 38 cases (1995-2005). *J Am Vet Med Assoc.* July 2007; 231 (1): 79-88.
- 8) Quinn R, Cook AK. An update on gallbladder mucoceles in dogs. *Veterinary Medicine.* April 2009. 169-175

Figures:

- 1) Maggie, post op
- 2) Abdominal ultrasound: gallbladder mucocele
- 3) Abdominal ultrasound: mild free peritoneal fluid around gallbladder and liver
- 4) Cholecystectomy surgery: closure of incision into common bile duct
- 5) Maggie, post op