Clinic 17 - Just Dogs

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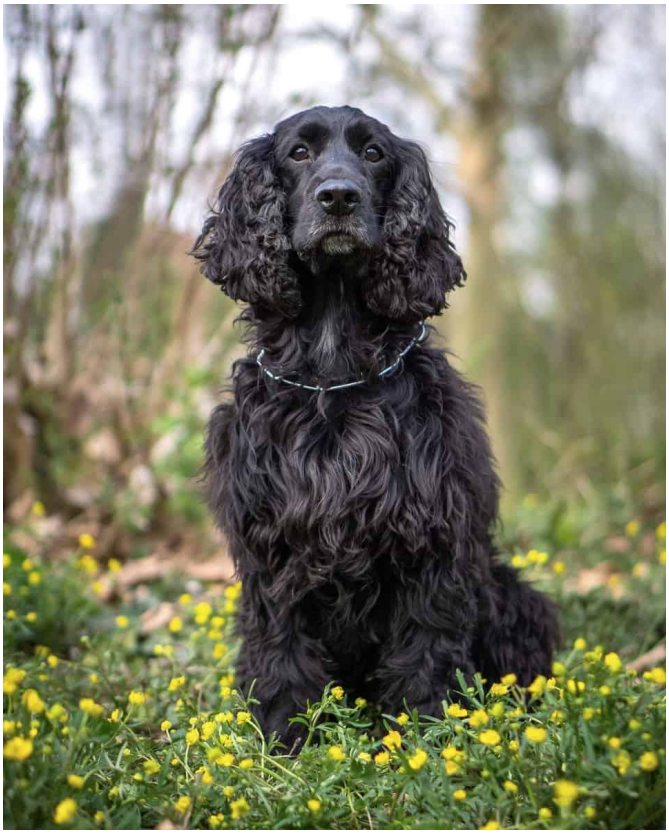
Gallbladder Mucocele



Name: Milly

Age: 9 years 11 months

Species: Canine, Cocker Spaniel

Weight: 20 kgs

Sex: Female, Spayed

Differentials

- Pancreatitis
- Dietary indiscretion
- Parasitic infection
- Leptospirosis
- Gallstones
- Primary liver disease
- Neoplasia
- Gastric ulceration
- Acute kidney injury
- Hyperadrenocorticism
- Hypothyroidism

Case Summary

A 9 year 11 month, female, spayed Cocker Spaniel diagnosed with a gallbladder mucocele (GBM) exacerbated by *E. coli* colonization and mucosal hyperplasia with secondary hepatitis, cholangitis, and cholestasis. She originally presented with acute vomiting, anorexia, abdominal pain, and mild diarrhea. Hypercholesterolemia, hyperbilirubinemia, elevated ALP/ALT/bile acids were also present. Definitive diagnosis was made using ultrasonography, and treatment was cholecystectomy.

Gallbladder Mucoceles

Pathophysiology

A gallbladder mucocele (GBM) is a buildup of mucus and bile acids in the gallbladder. The etiology of GBM is not entirely understood. However, there are some predisposing factors that have been seen to lead to GBM, including decreased gallbladder motility and/or concurrent gallbladder obstruction or partial obstruction. The decreased flow rate of bile and mucus through the gallbladder can result in an accumulation of those biliary components to form a mucocele. There are several possible disease processes that are associated with the formation of GBM, including pancreatitis, choleliths, hyperadrenocorticism, hypothyroidism, and genetics. Choleliths can cause intrabiliary obstruction or partial obstruction, as well as inflammation, all of which would cause reduced bile flow through the gallbladder. Similarly, pancreatitis can cause local inflammation which could cause extra biliary obstruction, also reducing flow. Hyperadrenocorticism causes an increase in cortisol in the blood, which reduces gastrointestinal motility, including gallbladder motility, resulting in decreased bile flow. Cocker Spaniels, Shetland Sheepdogs, and Shih Tzu breeds have a predisposition for developing GBM. This is possibly due to inborn errors of lipid metabolism, which results in hypertriglyceridemia, which could prevent the proper digestion of fats and is correlated with GBM.

Common Problems

The major risk of GBM is a gallbladder rupture. This can lead to rapid mortality without emergency intervention. Mortality is associated with release of bile acids into the peritoneum, causing widespread peritonitis and eventual sepsis. Lesser effects of GBM are suppurative cholangitis and/or cholangiohepatitis, both of which can cause extrahepatic bile duct obstruction, accelerating the pathogenesis of the GBM.

Diagnosis

GBM is commonly diagnosed via ultrasonography, with visualization of a "kiwi" gallbladder. The appearance on ultrasound is related to the hyperplasia of the gallbladder and the mucus attached to it appearing as projections into the lumen. Simple mucus in the gallbladder would appear as gravity-affected hyperechoic areas. In other words, the mucus should fall to one side or the other as the animal is turned if the mucus in the gallbladder is not forming a mucocele. A mucocele will not be impacted by gravity, so it will remain constant with turning the animal. Initial lesion localization is achieved by blood chemistry analysis with marked increase in ALP (induced with bile duct obstruction/impairment). More definitive diagnostic procedures besides ultrasound include gallbladder cytology to understand the types of inflammatory cells involved, and liver biopsy to determine liver involvement

Treatment Plan

Gallbladder mucoceles are emergent findings that are usually treated with surgical intervention. Due to the high risk of gallbladder rupture, surgery to remove the gallbladder (cholecystectomy) is the recommended treatment. Alternatives to this treatment are gallbladder lavage to flush out the mucus or medical management with ursodeoxycholic acid (Ursodiol). Gallbladder lavage leaves the gallbladder intact, which can allow for future GBM precipitation. Gallbladder lavage is less advantageous than surgery and therefore not recommended. Medical management with Ursodiol is also an option in an attempt to dissolve the mucus over time. This takes a significant amount of time to produce results, however, so a patient with a more imminent GBM (showing painful clinical signs or at risk of rupture), would not be a candidate for this treatment. Both the lavage and Ursodiol are recommended treatments for patients with an early GBM (not yet adhered to the mucosal layer), since there is a higher chance of rapid success and less of a chance of the GBM returning.

Cholecystectomy

The abdomen should be opened and purse string suture is placed in the gallbladder. At this point a small incision is made into the gallbladder through the purse string suture, and a red rubber catheter is placed to ensure patency and location of the cystic and common bile ducts. The duct is then ligated to completely close off the cystic bile duct from the common bile duct. The cystic duct should also be ligated to prevent leakage of bile acids into the abdomen. The gallbladder and the cystic bile duct can then be surgically removed. In cases of GBM, the gallbladder is filled with thick black to green viscous fluid, representing the bile acids and mucus buildup.

ACTH Producing Neoplasm

Malignant endocrine tumors of the gallbladder are uncommon, but there have been human cases of neuroendocrine tumors of the gallbladder producing ACTH via paraneoplastic syndrome, leading to Cushing's-like symptoms and presentations. This tumor would obstruct the gallbladder and would also decrease motility with excess cortisol, predisposing to GBM. In canines, neuroendocrine tumors of the pancreas have also been shown to have ACTH secreting paraneoplastic syndrome, promoting the same effect.

Pancreatitis and GBM

Pancreatitis can inhibit lipoprotein lipase which is an enzyme that breaks down circulating triglycerides which can lead to an increase in cholesterol which is a possible likely mechanism in this case.

Hyperadrenocorticism

The most common causes of hyperadrenocorticism (Cushing's disease) are pituitary dependent (secondary) and adrenal gland dependent (primary). Secondary processes can involve a functional tumor of the pituitary, producing ACTH, and leading to increased stimulation of adrenal glands leading to secretion of large amounts of cortisol. Primary indicates a disease process of the adrenal glands (such as an adrenal adenoma) leading to increased production of cortisol without increased stimulation due to ACTH. Less common causes include prolonged use of a corticosteroid medication or ACTH producing hormone elsewhere in the body such as a ACTH producing neuroendocrine carcinoma of the gallbladder. Symptoms include polyuria/polydipsia, fat deposits around the body, pot belly, polyphagia, alopecia, and immunosuppression (meaning Cushings predisposes patients to development of many diseases). There are two methods of testing for Cushing's: ACTH stimulation testing and low dexamethasone suppression testing. The former has a high sensitivity, low specificity, and is affected by disease states. The latter has high specificity, low sensitivity, and is not affected by disease states.

Citations

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