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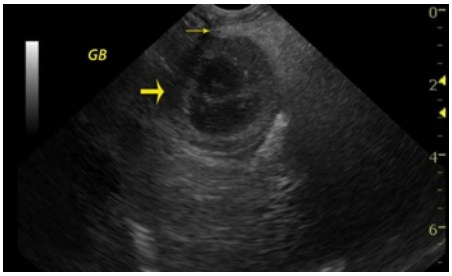
GALLBLADDER MUCOCOELES – 3 PAGES



For some great tips on defining a Gall Bladder Mucocoeles, please visit http://www.sonopath.com/downloads/Interview_GBM_100223.pdf at SonoPath.com.

A gallbladder mucocoele exists when the gallbladder becomes overly distended with an excessive accumulation of mucus due to cystic mucosal hyperplasia. Previously, pathologists noted this finding at necropsy and considered it an age-related incidental lesion. More recently it has become evident that not all gallbladder mucoceles remain silent clinically, and that they can be associated with extrahepatic bile duct obstruction (EHBDO), cholecystitis, and gall bladder wall rupture. Approximately 50% of mucoceles can result in necrosis and rupture of the gallbladder, typically at the neck or fundic region[1, 2]. Etiology remains unknown, but it is suspected to be related to diseases of cholestasis and lipid metabolism. Suggested causes include primary or secondary gall bladder motility disorder, dyslipidemia, extra-hepatic bile duct obstruction, and primary or secondary disorder of the mucus producing cells. Clinical correlation is seen with cholangitis, cholecystitis, choleliths, biliary obstruction from cholelithiasis or neoplasia, pancreatitis, and abnormal gall bladder motility[3]. Bacterial infection of the gallbladder has been associated with increased mucin production in the dog, and may be pathologic in creating the excessive mucus accumulation seen in this condition. In humans, hypercortisolism is related to chronic cholecystitis and changes to the bile biochemical composition[4]. In dogs, hyperadrenocorticism is frequently found to be coexistent, but a co-relational mechanism has not been ascertained. A current study is underway by our team assessing endogenous and exogenous excessive cortisol and the clinical emergence of mucoceles in dogs.

Clinical signs and Diagnostics:

Several retrospective studies have characterized this disorder. Most common signs include vomiting (87%), abdominal pain (87%), anorexia (78%), lethargy, icterus (57%) and fever (26%). The average duration of illness is 5 days. Leukocytosis with or without a left shift and elevated hepatic enzymes (SAP 100%, GGT 86%, ALT 77%, AST 60%) and bilirubin (63%) are most often seen. Ultrasonographic findings may include a distended gallbladder with centrally suspended luminal content and a hypoechoic intralumenal rim, thickening of the gallbladder wall, intralumenal stellate echogenic striations (the kiwi fruit sign), or the presence of non-dependent intralumenal contents or sludge. A hypoechoic ring seen around the gallbladder may indicate wall edema or early rupture. Presence of free fluid and localized echogenic hepatic parenchyma and intraabdominal fat may are also consistent with bile leakage and peritonitis. Pain is often noted on interrogation of the area. Dilation of the common bile duct may also be present [1,3].

Mucoceles are most commonly seen in middle aged to older dogs, with a median age of 10 years. Dogs as young as 3 years old have been reported, however. Miniature Schnauzers, Shetland Sheepdogs, Cocker spaniels, Shihtzus, Pugs, Bichons, Schipperkes, West Highland White Terriers, and Scottish Terriers appearing to be overrepresented breeds. Feline patients are less commonly also affected. A predisposition to mucocele formation in Shelties prompted an investigation and discovery of a specific genetic mutation that results in a disorder of phospholipid translocation in the liver. In the future, we may be able to screen young Shelties for this mutation which would allow us to monitor gall bladder mucocele development with ultrasound over time and thus offer early dietary and medical management or even surgical intervention as needed.

Common predisposing factors include middle to geriatric age, hyperlipidemia/hypercholesterolemia, gallbladder dysmotility, and cystic hyperplasia of the gallbladder wall. Abnormal adrenal hormones may play a causal role in altering GB mucus. Vacuolar hepatopathy is a common coexistent problem, and so investigation of underlying disorders associated with VH is advised. Since there exists an association between hypercholesterolemia/hyperlipidemia and GB mucoceles, these patients should be screened for hypothyroidism, adrenal hyperplasia syndromes (Cushing's disease and sex hormone problems), diabetes mellitus, pancreatitis, exogenous exposure to glucocorticoids, and necroinflammatory liver disease. (Hypothyroid dogs are 3x more likely to develop a mucocele and Cushinoid dogs are 29 x more likely to develop a mucocele). The NYSCVM at Cornell has recently verified that dogs with pre-existing risk factors may rapidly develop a mucocele after the initiation of glucocorticoid therapy.

Patients may be asymptomatic despite labwork abnormalities, even in the face of ultrasonographic evidence of a mucocoele. These patients are at risk of further complications such as gallbladder rupture, peritonitis, sepsis and related coagulopathies, and should be considered for surgical intervention [3].

Treatment:

Surgical intervention is recommended due to the significant risk associated with rupture, peritonitis and sepsis, and as medical management may not be effective [2,5]. These patients must be adequately stabilized prior to surgery, and a coagulation panel prior to surgery is recommended. Use of ursodeoxycholic acid (Actigal) is NOT recommended if any sonographic aspects of wall inflammation or emergent perforation are present in this condition due to risk of rupture[3]. Percutaneous centesis of the GB is not recommended in the presence of a mucocele. A few reports with low numbers of dogs have found medical therapy to be somewhat effective in very early subclinical stage. Therapy with Actigal at 20-25 mg/kg divided BID as well as SAMe at 20-40 mg/kg per day was utilized along with sequential sonographic and laboratory evaluations to assess for progression or resolution. ***However, there are no defined rules as to sonographic and enzymatic aspects with regards to differentiating a clinically significant mucocele versus a non-clinical stable mucocele that may become clinical in the future. Each patient must be assessed individually for this reason. Our team has seen very dramatic mucoceles not causing any overt clinical signs while other patients may have minor mucoceles causing significant clinical signs and necessitate urgent cholecystectomy to avoid the onset of bile peritonitis due to rupture.***

Antibiotic therapy directed towards gram-negative, enteric bacteria (E. coli, Klebsiella, Enterobacter, etc) and anaerobic bacteria is indicated, and will likely be necessary for several weeks following surgery. Culture and sensitivity results should guide long-term treatment. A cholecystectomy with possible bile duct transposition is the procedure of choice. The bile duct is often flushed to remove sludge and debris to help avoid post-surgical obstruction. Cholecystotomy for removal of GB contents is not advised because re-formation of mucocele concretions has occurred in several dogs treated in this manner. At the time of surgery, liver biopsies and culture and sensitivity of gallbladder contents should be obtained. Liver biopsies may show normal tissue, vacuolar hepatopathy, or mild to moderate portal hepatitis/fibrosis with bile duct proliferation. Ultrasound guided cholecystocentesis is not recommended in these cases due to the risk of gallbladder rupture and seeding the abdomen with bacteria. Rupture of the gallbladder constitutes a surgical emergency, and carries a worse prognosis for survival. Clinical impressions from several investigators note that upon surgical explore of asymptomatic

mucocoele patients, there is evidence of prior localized peritonitis and fibrosis, likely from tears in the neck of the gallbladder resulting in a minute amount of bile leakage.

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