Gall Bladder Mucoceles in the Dog

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Definition
A mucus filled gall bladder distention causing dysfunction.

Cause
Gall bladder mucoceles are thought to be more common than is currently diagnosed. Histopathology of the gall bladder wall reveals cystic mucinous hyperplasia. Progressive distention results in ischaemic necrosis and eventually gall bladder rupture. Inflammation is only associated with disease after ischaemic necrosis is evident. Obstruction of the extracystic biliary tract is usually secondary and not primary. They are more common in dogs with hypertriglyceridaemias and hypercholesterolaemias. Breeds such as Miniature Schnauzers, Cocker Spaniels and Shetland sheep dogs which are predisposed to hyperlipidaemias are also prone to developing gall bladder mucoceles. Many dogs with endocrinopathies are predisposed to hyperlipidaemias and GMC. The most common of these endocrinopathies are hyper-adreno-corticism, 29 times more likely to develop a GBM, and hyper-thyroidism, 3 times more likely to develop GBM. The administration of progestagens to dogs has also been associated with the development of GBM. In a study of dogs with GBM, 50% of cases had a genetic insertion mutation in the gene ABCB4 which encodes for a phosphatidyl choline transporter. Phosphatidyl choline protects the biliary epithelium from the cytotoxic detergent effects of bile.

Clinical signs
Patients are usually middle aged to older and of either sex. Common clinical signs of gall bladder mucoceles include vomiting, right cranial abdominal pain, anorexia, hyporexia, lethargy, polydipsia, polyuria and diarrhoea and are therefore not specific enough to make a diagnosis.

Biochemistry
Normal to neutrophilic leucocytosis, mild non regenerative anaemia, increased alkaline phosphatase, increased alkaline transferase, increased gamma glutamyl tranpeptidase increased total bilirubin, hypertriglyceridaemia hypercholesterolaemia and bilirubinuria. This biochemistry profile closely resembles hyperadreno corticism. There appears to be a close relationship between hyperadrenocorticism and gall bladder mucoceles. Dogs with HAC are 29 X more likely to develop GMC than dogs with normal functioning adrenal glands. This relationship has not been completely clarified. No single biochemical abnormality is seen with every case of GMC.

Imaging Gall Bladder Mucoceles

Radiographs may be normal or demonstrate a cranial mass effect or gas radio-opacities within the gall bladder if an emphysematous cholecystitis is present.

Abdominal ultrasonography may show an enlarged gall bladder, a distended common bile duct, immobile or echogenic organised bile, a stellate or striated biliary pattern and either a normal, thickened or ruptured gall bladder wall. Signs of gall bladder rupture include a discontinuous gall bladder wall, hyper-echoic peri-cholecystic fat, pericholecystic fluid or peritonitis, striated or stellate mucocele material may be seen within the abdomen and the gall bladder may be difficult to visualise. Ultrasonographic signs of hyperadrenocorticism or pancreatitis, if concurrently present, may also be seen. In one study approximately 1/3 of gall bladder mucoceles documented at surgery did not have strong presurgical ultrasonographic evidence of a GBM. Similarly ultrasonographic evidence of peritonitis with previous or current GBM rupture was inconsistent.

Bile aspiration technique
Bile aspiration is done trans cutaneous, trans hepatic and ultrasound guided. The gall bladder must be emptied. A post procedure scan to check for leakage must be performed. Cytology and cultures are done on the aspirates even though 72% cultures are negative.

Treatment GBM
Medical treatment involves the use of appropriate antibiotics. The most common isolates are E.coli, a gram negative facultative anaerobe, gram positives and other anaerobes. Remember only 28% of cultures are ever positive. A choloretic may be used provided the bile tract is patent. Pain control and anti-inflammatories may also be useful. The unfortunate fact is that medical treatment is unproven benefit except in a few case based studies.

Nutrition
If the animal is not eating, just like any other disease appropriate and adequate nutrition is instituted. No proven recommendations on diet can be made because the studies are not done. The basic principles used for the treatment of liver diseases apply. Namely avoid diets exacerbating any underlying causes, such as high fat diets with pancreatitis. Use the most amount of high biological value proteins or amino acids that the patient can tolerate and include all the essential amino acids in the diet. The diet should probably be cholesterol and cholesterol precursor deficient. Copper deficient but potassium, zinc and vitamin c enriched. Carbohydrates together with the protein and amino acids should provide adequate energy for rehabilitation which is equal to that of an active patient.

Surgery
Consider primary and secondary liver disease when deciding to perform surgery. Most patients have concurrent liver disease. A liver biopsy is always taken at the time of surgery. Common concurrent liver diseases include portal fibrosis with bile ductile proliferation, neutrophilic cholangitis, lymphoplasmacytic cholangitis. There is no doubt that a well-established gall bladder mucocele will not respond completely to medical treatment alone. If the disease isolated to the gall bladder a complete response to a

cholecystectomy, with retrograde or antegrade bile duct flushing, is expected. Surgical mortality is 21-40%. Interestingly there is no difference in survival between patients with gall bladder rupture and those with intact gall bladders at the time of surgery but those with a septic bile peritonitis had a 55-73% mortality versus a 0-13% mortality for aseptic bile peritonitis. Infection rates vary from 9-44% in studies.
Post-surgical complications have included pulmonary thrombo-embolisms, aspiration pneumonia, severe pancreatitis, bile peritonitis, sepsis and systemic inflammatory response. Hypokalaemia, severe neutrophilia, post-operative elevations in lactate levels, and post-operative hypotension were associated with a poor outcome. Of those that survive the post-operative period, 66% are alive at 2 years. Laparoscopic surgery is complicated and it is difficult to flush the bile duct using this modality. It is described but best reserved for uncomplicated cases.

The decision to do surgery
Consider offering surgery to all patients on detection due to poor potential surgical outcome once symptomatic. Reserve medical management to early cases or cases that are in any case a poor surgical candidate, such as those with severe concurrent liver disease. Intensive monitoring protocols are necessary. Consider laparotomy immediately if a patient is symptomatic.

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