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# Canine cholangitis, cholecystitis and gallbladder mucocoeles

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Bile is produced continuously and is composed of 97% water, 0.7% bile salts, 0.2% bilirubin, 0.51% fats (cholesterol, fatty acids, and lecithin) and 200 meq/l inorganic salts. Hepatocytes excrete cholesterol and bilirubin into the canaliculi, then water, bicarbonate and sodium ions are secreted via the lining of the ducts. Bile flows from canaliculi to terminal bile ducts, to hepatic ducts and then the cystic duct (which enters into the gall bladder (GB)). Bile is stored in the gallbladder until stimulation from fatty acids in the duodenum results in release of cholecystokinin and subsequent contraction of the GB. Bile then travels down the common bile duct and enters the duodenum at the major duodenal papilla.

Bile is responsible for the absorption of fats, fat-soluble vitamins (A, D, E & K), and is the route of excretion for bilirubin, cholesterol and a large variety toxins and toxic by-products.

## Cholangitis and cholecystitis

Cholangitis is defined as inflammation of the biliary tract, usually related to bacterial infection, while cholecystitis is defined as inflammation of the gallbladder specifically.

The aetiopathogenesis is not clearly defined, although the two main routes for invasion of the biliary tract involve ascending bacterial infection from the duodenum or haematogenously via the hepatic portal venous blood. The ascending infection is believed to be the most likely and most common source of infection. Inoculation via the portal vein is possible if a large volume of bacteria enter the liver from the portal vein (secondary to breakdown of the intestinal defence mechanisms) and overwhelm the Kupfer cells (hepatic macrophages).

Cholangitis should only occur if the normal biliary defence mechanisms are ineffective or overwhelmed. The biliary defence mechanisms include the sphincter of Oddi (mechanical barrier to ascending infection), flushing action of bile, bacteriostatic action of bile salts and potent local immunological defence mechanisms (Kupfer cells, secretory IgA and mucous). Factors predisposing to cholangitis include biliary stasis, increased biliary pressure, overwhelming hepatobiliary-enteric circulation of bacteria with large portal innocula and cholestasis.

Cholangitis and cholecystitis are uncommon diseases and are relatively poorly described. Recent articles highlight various salient points of the disease.

### What do we know clinically about cholangitis and cholecystitis?

Tamborini *et al*. (2016) described the findings in 27 dogs and Harrison *et al*. (2017) described the findings 54 dogs with histopathologically confirmed cholangitis or cholecystitis and bile culture/cytology results supporting a bacterial aetiology.[1,2]

The most common clinical signs were vomiting (72%), lethargy (70%), inappetence (65%) and diarrhoea (32%). The most common physical findings were pyrexia (>39.5°C, 30%), dull mentation (26%), icterus (26%), abdominal pain (22%) and hepatomegaly (11.1%). The most common clinical pathological findings included increased hepatic enzymes (up to 98%), hyperbilirubinaemia (64%) hypercholesterolemia (56%) and an inflammatory leukogram.

Neither study identified ultrasound (US) findings specific for cholangitis and no single finding could be described as pathognomonic. Hepatobiliary abnormalities were identified in 84% of dogs including increased gallbladder sediment (60%), a distended gallbladder (35-53%) and dilated bile ducts (39-53%), thickened gallbladder wall (>3mm = 35%), gallbladder sludge (35%), free abdominal fluid (31%), heterogeneous hepatic parenchyma (27%), hyperechoic hepatic parenchyma (23%) and a gallbladder mucocoele (23%) (Fig 1). Ninety-eight percent of dogs in the study were diagnosed with neutrophilic cholangitis or cholangiohepatitis on histopathology, of which 78% were chronic.

Concurrent clinical conditions or exposure to immunosuppressant agents were present or administered in 82% of dogs. Concurrent conditions included gallbladder anatomical abnormalities, mucocoeles, cholelithiasis, calcified gallbladder wall, surgery-induced anatomical abnormalities, duodenal stenosis and stricture of the duodenal papilla, pancreatitis and lymphoplasmacytic enteropathy.

In another study, no pathognomonic findings for bacterial cholecystitis or bactibilia were identified in 10 dogs with confirmed cholecystitis compared to 30 control dogs.[3] Dogs with cholecystitis were significantly more likely to have immobile biliary sludge (sensitivity of 70% and specificity of 100% for the presence of bactibilia) and had a greater degree of biliary sediment formation than did control dogs. Five of 10 case dogs were Dachshunds (overrepresented, which may suggest a breed predisposition). Of the dogs in which a biopsy was taken, concurrent inflammatory bowel disease was present in 75% of cases. The conclusion of the study was that cytologic evaluation of bile should be considered in the routine assessment of dogs with hepatobiliary disease if immobile biliary sludge is present.

### What can you expect from biliary cultures, and which is better, bile or liver?

Wagner *et al.* (2007) reported on the biliary culture results of 190 dogs and 58 cats.[4] Biliary cultures yielded more positive results (30% [18/60]) than hepatic cultures (7% [15/215]). In patients with histopathologically confirmed cholecystitis, 62% (8/13) had positive biliary cultures. Of the dogs with hepatic inflammation, 23% (7/30) had positive biliary cultures and only 6% (6/103) had positive hepatic cultures. This implies hepatic inflammation may be as a result of

cholecystitis without bacteria cultured from the liver, or that hepatic culture is insensitive. Liver samples obtained by surgery or laparoscopy were more likely to yield positive cultures than those obtained by percutaneous needle biopsy, emphasising the importance of sample size.

*Escherichia coli* (*E. coli), Enterococcus spp., Bacteroides spp., Streptococcus spp.,* and *Clostridium spp*. were the most common bacteria cultured with more than 80% of Enterobacteriaceae susceptible to ciprofloxacin or aminoglycosides.

A concerning trend was that only 30–67% of cultured bacteria were susceptible to first-generation penicillin's and cephalosporin's. Of the 16 cases of *E. coli* identified in the Tamborini *et al*. study, 10 showed resistance to 3 or more classes of antimicrobials, while all of the *Enterococcus spp*. isolates showed resistance to several agents.[1] This highlights the importance of antibiograms for positive case outcomes.

**Which is better, cytology or culture?**

Bactibilia was identified by microscopy in 33% of dogs and cats in one study, while 21% were identified by bacterial culture.[5] The agreement between the two tests was substantial: 85%, and this improved to 94% for animals that received no antimicrobials within 24 hours prior to sample collection.

The conclusion of the study was that concurrent bacterial culture and microscopic examination of bile samples are recommended for all cats and dogs evaluated for hepatobiliary disease.

**Is it safe to perform cholecystocentesis?**

The safety of cholecystocentesis was retrospectively evaluated and found to be relatively safe (with a total of 300 performed in 201 dogs and 51 cats (some animals had more than one cholecystocentesis performed)).[6] The complication rate was low: 8/300 (2.75%) procedures experiencing major complications, with bile peritonitis in 2/300 (0.7%) of cases. Two of the complicated cases had mucocoeles. The most common complications included bile leakage, bile peritonitis, haemorrhage, haemobilia and bacteraemia or sepsis.

In another study, complications after cholecystocentesis were identified 3.4% of dogs.[7] These included presumed peritoneal haemorrhage, gallbladder wall oedema, gallbladder collapse and intraluminal gallbladder haemorrhage.

**Cholecystocentesis – the procedure**

The ventral abdomen is shaved and the skin is prepared with hibitane and alcohol. Percutaneous ultrasound-guided cholecystocentesis is performed under heavy sedation or general anaesthesia with a 22-gauge, 1.5-in needle and 12mL syringe, via a right-sided ventral abdominal, transhepatic approach.[8] There should ideally be at least 2 cm of hepatic parenchyma positioned between the gallbladder wall and the subcutaneous fat. Ideally, the gallbladder should be emptied of its content. Large vascular structures were avoided with the aid of colour flow Doppler.

**Treatment of cholangitis and cholecystitis**

Bile culture and antibiogram are crucial to the success of treatment considering the high rate of resistance to commonly used first line antibiotics (e.g. amoxycillin). It is reasonable to start with enrofloxacin considering the reported resistant bacteria as well as the knowledge that enrofloxacin is appropriate for liver disease. Once culture and antibiogram results are available, the appropriate antibiotic should be used. Supportive treatment entails anti-oxidants support for the liver and helps reduce fibrosis. These include, amongst others, S-adenosylmethionine (SAMe), milk thistle, ursodeoxycholic acid and vitamin E.

Hepatic enzymes should be monitored every 3-4 weeks and it is advised to re-culture ideally when the patient has been off antibiotics for 3 days. In chronic cases, antibiotics only give temporary resolution and cholecystectomy is often required for complete resolution.

**Prognosis is dogs with cholangitis and cholecystitis**

Tamborini *et al*. found that gallbladder rupture was a poor prognostic indicator with 50% of dogs with rupture not surviving to discharge. Discharged dogs had a guarded to fair prognosis with 17/18 alive at 2 months, although 5/10 re-evaluated had persistent liver enzyme elevation 2–12 months later.

Harrison *et al.* found the median patient survival was 671 days and if no cholecystectomy was performed there was a 2.1 greater hazard for death ($CI_{95\%}$: 1.0–4.3) compared to cholecystectomised dogs.

**Gallbladder Mucocoeles**

A gallbladder mucocoele (MC) is defined as an abnormal distention of the gallbladder by an inappropriate accumulation of mucous and inspissated bile salts. Histopathologically, mucocoeles are characterised by cystic mucinous epithelial hyperplasia and inflammatory infiltrates. Ultrasonographically, mucocoeles appear as non-dependant sludge or more organised striations (kiwi/stellate/starfish).

**Pathophysiology**

Grossly a MC is an accumulation of green mucinous material with lamellar striations. This can lead to a functional obstruction of the common bile duct (CBD), resulting in further resorption of the liquid component of the bile. Histopathology is classified as a cystic mucinous hyperplasia. Many diseases have been associated with this condition including hepatic disease, pancreatic disease, diabetes mellitus, hyperadrenocorticism and hypothyroidism.

Concurrent cholecystitis rates vary from 9 to 43%. *Enterococcus, Escherischia coli, Enterobacter* and *Clostridium spp* are the most common bacteria identified and are most likely are secondary to intestinal ascending infection. It is hypothesised that this may be due to reduced immune competence in dogs with endocrinopathies.

Nutritional and environmental factors are also thought to play a role including dyslipidaemias, as are genetics as in the case of the over-

representation of Shetland sheepdogs, cocker spaniels, dachshunds and miniature schnauzers.

Hyperadrenocorticism is commonly associated with MC development. In an experimental design, beagles were exposed to exogenous corticosteroids (equivalent of 2 mg/kg prednisolone PO sid) for a sustained period of time (12 weeks) and compared to controls.

A reversible change in the bile acid composition with decreased conjugated bile acids was identified. The shift to less water soluble, more hydrophobic caustic bile acids may be responsible for mucosal irritation and hence trigger mucinous hyperplasia.

In humans, diabetes mellitus is strongly associated with gallbladder disease. This could be attributed to hypercholesterolaemia and hypertriglyceridaemia as well as hypo-motility due to DM mediated neuropathy. The relevance in dogs is pure extrapolation and of a low level of evidence, however a fairly strong association exists. People with hypothyroidism have a predisposition to changes in bile salts and reduced GB emptying. Thyroxin will relax the sphincter of Oddi. Evidence in dogs lies in the increased odds ratio of GB MC in dogs with spontaneous hypothyroidism.

Is biliary sludge a precursor of mucoceles?

DeManaco *et al*. (JVIM 2016) followed 45 dogs with biliary sludge for 12 months to determine if sludge predicted MC formation.[9] No significant worsening (increase volume or greater organisation) was identified over the 12 months in 88% of all the dogs. The biliary sludge was regarded as an incidental finding. No risk factors were identified.

**Signalment**

Middle aged to old dogs, with no definitive breed predisposition. Overrepresented breed include Shetland sheepdogs, cocker spaniels, miniature schnauzers and dachshunds. There is no apparent sex predilection.

**History**

Subacute to chronic non-specific clinical signs including vomiting, anorexia, lethargy, diarrhoea, polyuria-polydipsia and abdominal discomfort.

**Physical examination**

Icterus, non-specific signs of peritonitis, or no clinical signs.

**Laboratory abnormalities**

Severely elevated liver enzymes (ALT/ALP/GGT), increased total bilirubin and leucocytosis. Cholesterol may be elevated in the case of extra-hepatic bile duct obstruction. Other clinical pathological abnormalities attributable to an underlying endocrinopathy or changes may also be associated with co-morbid disease including diabetes mellitus, hyperadrenocorticism, hypothyroidism and pancreatitis. It should however be cautioned, any acute disease could impact on TT4 and thyroid stimulating hormone.

**Diagnostic imaging**

Ultrasonography helps exclude other causes of vomiting including gastro-intestinal obstruction or neoplasia and other hepatobiliary diseases including acute liver injury, neoplasia, choleliths and pancreatitis.

Ultrasonographic findings with a mature or clinical MC include an enlarged gallbladder, thickening of the gallbladder wall (>3mm), double rimmed gallbladder wall, with non-motile sludge or most typically a kiwi fruit appearance (Fig. 2 & 3).

Differentials for a double rimmed gallbladder wall include hepatitis, cholangitis, right heart failure, renal failure, pyelonephritis and portal hypertension. Gallbladder rupture is a common finding with mature MC's (approx. 50%) and is commonly identified as discontinuity of the wall of the fundus, while pericholecystic hyperechoic fat is suggestive but not diagnostic. Unfortunately, abdominal ultrasonography is a low sensitivity test for the identification of gallbladder rupture.

**Management**

Surgical intervention is the standard of care which requires cholecystectomy and retrograde lavage of the CBD. It is crucially important that the need for surgery is correlated to physical examination findings as well as laboratory and ultrasound findings, as moderate immobile biliary sludge accumulation in itself, is not an indication for surgery and may be incidental. It becomes difficult to motivate surgery in a completely asymptomatic patient. When surgery is undertaken, biopsies of the gallbladder, bile and liver should be collected for histopathology and culture.

Medical management as the sole therapy is largely unsuccessful. Where hypothyroidism has been diagnosed, medical management may be curative. Medical therapy in the majority of cases is unable to transform a semisolid MC into liquid bile. Supportive care for symptomatic patients includes analgesia, antibiotics, choleretics, hepato-supportive supplements including anti-oxidants. Diagnosis and management of the underlying endocrinopathy should be initiated.

**Prognosis**

Widely varying mortalities have been reported (14-68%). Older studies did not relate survival to pre-surgical bile leakage and rupture, but dogs in a recent study with GM and gallbladder rupture with bile peritonitis at the time of surgery were 2.7 times more likely to die than dogs without gallbladder rupture and bile peritonitis.[10] Success of surgery is also surgeon-experience-dependant. Postoperatively, hypotension was very highly associated with mortality.

Several prognostic factors/variables were identified in dogs with gallbladder MC's that underwent cholecystectomy. Increased odds of death were associated with the Pomeranian breed, age, recognition of jaundice by the owner, total bilirubin concentration and concurrent hyperadrenocorticism.[11] Vomiting at presentation was associated with decreased odds of death. The presence of hypothyroidism or diabetes mellitus did not impact outcome.

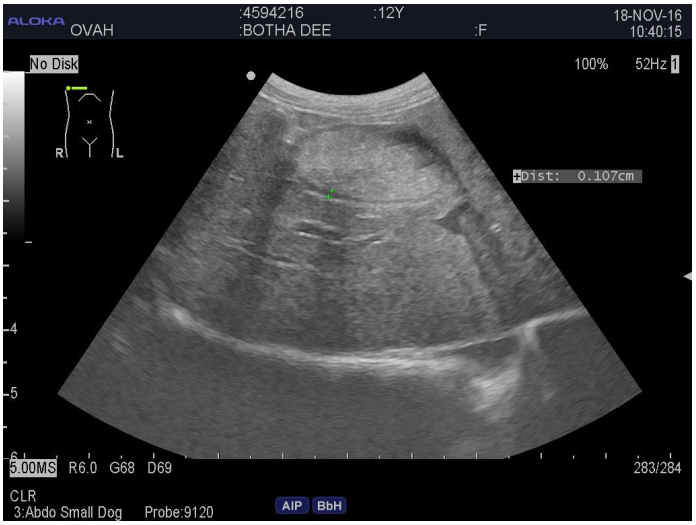



*Fig 1. A twelve-year old female Miniature Schnauzer with confirmed bacterial cholecystitis. The gallbladder is moderately distended with homogenously hyperechoic material that occupies most of the gallbladder lumen. Image courtesy of Dr. S Dancer.*

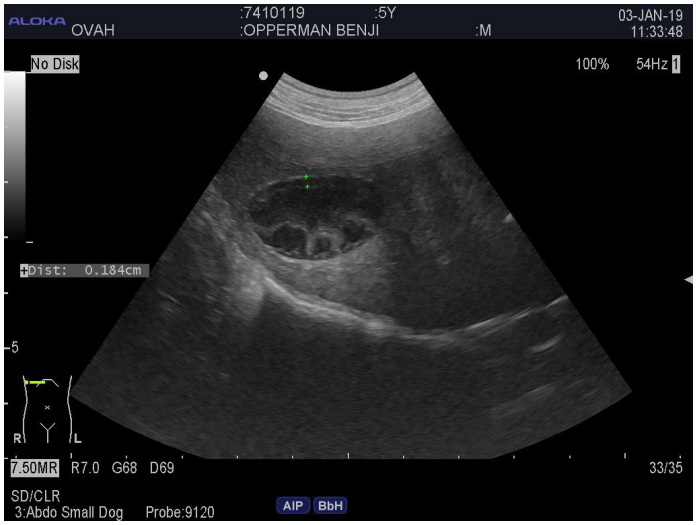



*Fig 2. A seven-year old male Pomeranian with a mature mucocoele. The gallbladder contains a well-defined hyperechoic stellate structure suspended in its lumen. The gallbladder wall is subjectively thickened with a hypoechoic outer rim and hyperechoic inner rim visible. Image courtesy of Dr. S Dancer.*

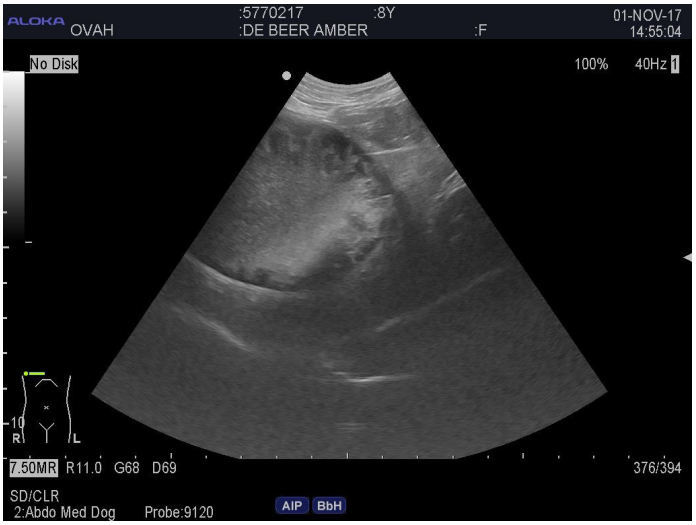
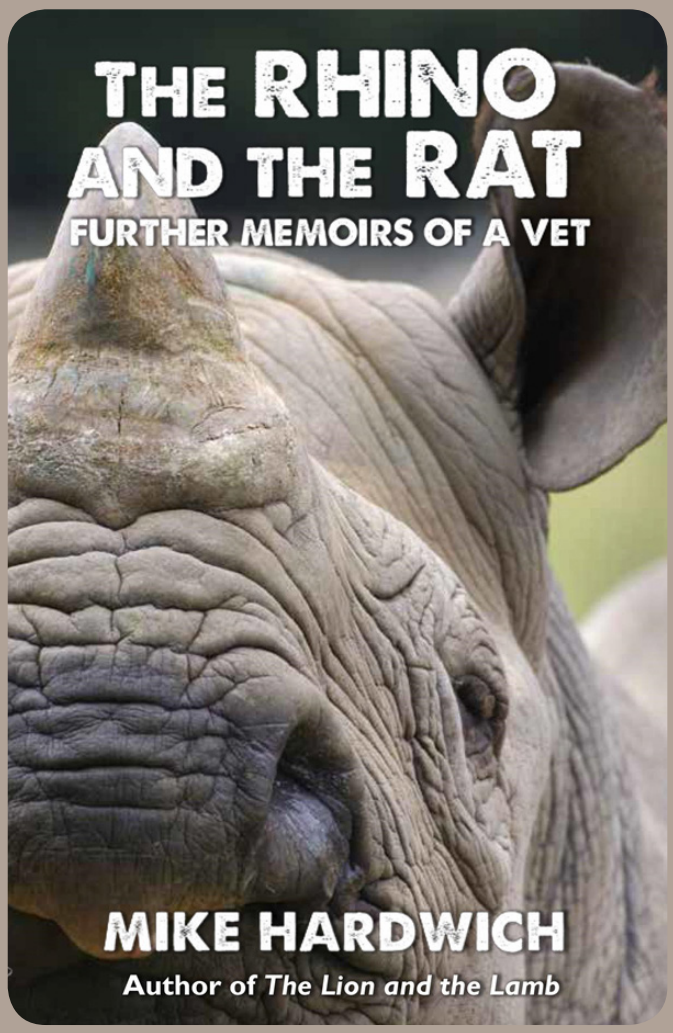
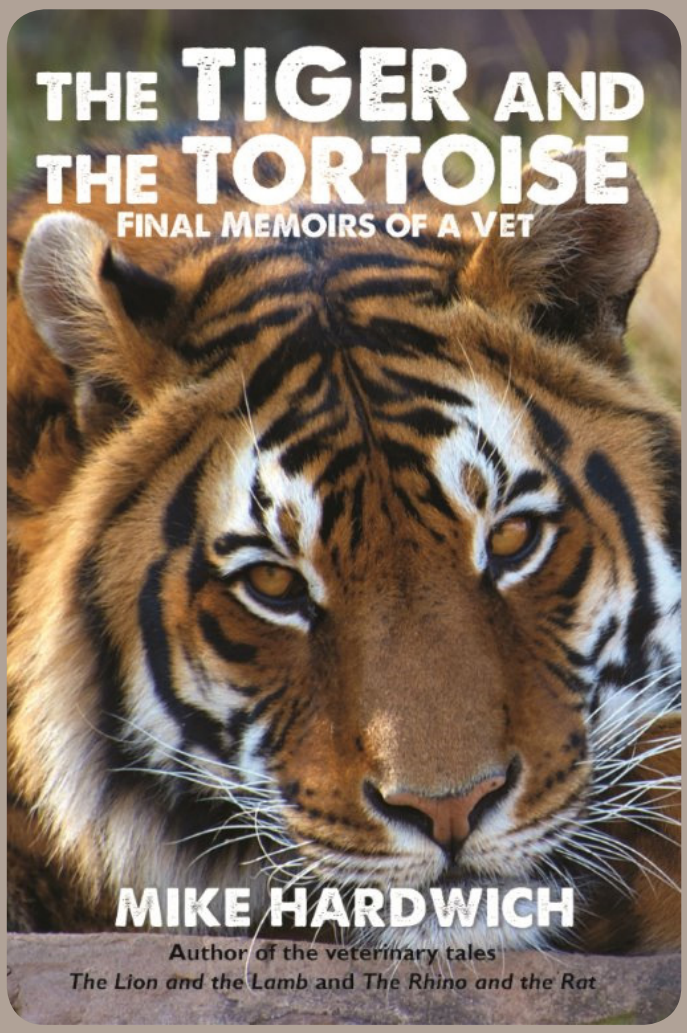



*Fig 3. An eight-year old female mixed breed with a mucocoele. A well-defined, stellate hyperechoic structure suspended is present within the gallbladder. Finger-like projections extend from the central structure peripherally to the gall bladder wall. Image courtesy of Dr. S Dancer.*

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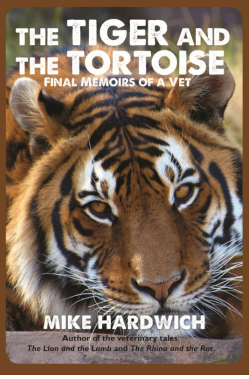



**As a country vet whose territory covers the rich valleys and farmland of KwaZulu-Natal, his clients range from cattle farmers to owners of domestic pets, from game ranchers to circuses. The demands on a vet are constant and often arrive at very inconvenient times. Called upon day and night, Mike brings to each case his skills, ingenuity and years of experience, and although he never loses sight of his aim of preserving and improving the lives of the animals he is called upon to treat, sometimes he is sorely challenged by their owners.**

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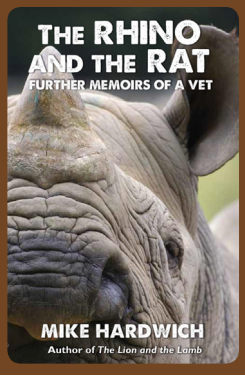



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# MULTIPLE-CHOICE QUESTIONS

**QUESTION 1**
**Select the FACTOR THAT PREDISPOSES to cholangitis.**
a. Small intestinal bacterial overgrowth
b. Hypercholesterolaemia
c. Biliary stasis
d. Deficiency of Vitamins A, D E & K
e. Previous antibiotic therapy

**QUESTION 2**
**Select the CORRECT statement regarding cholangitis.**
a. The pathognomonic finding for cholangitis is gallbladder sediment on ultrasound
b. Dilated bile ducts are only indicative of cholangitis
c. Only biliary cytology should be performed to confirm bacterial cholangitis
d. *Staphylococcus* spp. is one of the most commonly isolated bacteria
e. The most common clinicopathological finding was increased hepatic enzymes

**QUESTION 3**
**Select the CORRECT treatment option for cholangitis at the point of initial diagnosis.**
a. Cholecystectomy
b. Ultrasound guided complete cholecystocentesis
c. Corticosteroids
d. Culture-based antibiotic therapy
e. Liver supportive treatment

**QUESTION 4**
**It is important to consider concurrent diseases at the point of diagnosis that may have contributed to the development of cholangitis. Select the one disease NOT associated with the development of bacterial cholangitis**
a. Inflammatory bowel disease
b. Gastric ulceration
c. Cholelithiasis
d. Pancreatitis
e. Corticosteroid therapy

**QUESTION 5**
**Select the disease NOT associated with the development of gallbladder mucocoeles.**
a. Inflammatory bowel disease
b. Hyperadrenocorticism
c. Diabetes mellitus
d. Hypothyroidism
e. Pancreatitis

**QUESTION 6**
**Select the CORRECT answer regarding biliary sludge.**
a. Biliary sludge is always indicative of an endocrinopathy
b. Biliary sludge is always indicative of bacterial cholecystitis
c. In the majority of cases biliary sludge will develop into a mucocoele
d. If biliary sludge is identified during abdominal ultrasound, bile should be aspirated
e. Biliary sludge is often an incidental finding

**QUESTION 7**
**Select the CORRECT option to complete the sentence: The diagnosis of a clinically significant mucocoele is based on clinical signs, ...**
a. abdominal ultrasound and a positive bacterial culture
b. elevated liver enzymes and a history of hyperadrenocorticism
c. a positive bile culture and a history of diabetes mellitus
d. abdominal ultrasound and elevated liver enzymes
e. elevated liver enzymes and a history of diabetes mellitus

**QUESTION 8**
**Select the CORRECT option for the management of a dog that is clinically ill due to a gallbladder mucocoele.**
a. Conservative management
b. Surgical management
c. The patient should be stabilised and surgery performed only if gallbladder rupture is suspected
d. If the patient is hypothyroid, conservative management should be implemented
e. Immobile biliary sludge accumulation in itself is an indication for surgery

**QUESTION 9**
**Select the factor ASSOCIATED WITH an increased odds of survival in dogs that undergo cholycystectomy for gallbladder mucocoeles.**
a. Age
b. Recognition of jaundice by the owner
c. Total bilirubin concentration
d. Concurrent hyperadrenocorticism
e. Vomiting

**QUESTION 10**
**Select the CORRECT statement regarding biliary disease.**
a. Cholangitis is a common disease and should be considered in any dog with raised hepatic enzymes
b. Cholecystectomy is the initial treatment of choice in dogs with bacterial cholecystitis
c. Hyperlipidaemia of Miniature Schnauzer is associated with the development of cholecystitis
d. Gallbladder mucocoeles are commonly associated with endocrinopathies
e. Gallbladder mucocoeles are usually secondary to bacterial cholecystitis

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