

Cholelithiasis and Choledocholithiasis in the Dog

Gall bladder and bile duct stones in the dog.

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The path of bile in the liver

The components of bile are produced in hepatocytes. Active and passive transporters excrete these components into bile canaliculi. For bile salts and bilirubin these pumps include the canalicular bile salts-export pump and the canalicular conjugate export pump. For cholesterol and phosphatidyl choline the pump is known as the outer leaflet canalicular membrane pump. Combinations of hydrophilic and hydrophobic bile acids surround bilirubin, cholesterol and other components of bile to form soluble micelles for easy transport. The bile canaliculi coalesce into interlobar ducts. Interlobar ducts coalesce into lobar ducts. Lobar ducts join to form the left and right hepatic ducts. The left and right hepatic ducts form the common bile duct that passages to the minor pancreatic duct in the dog and the major pancreatic duct in the cat before reaching the duodenum through a specialised sphincter known as the papilla of Odi. The cystic bile duct joins the gall bladder to the common bile duct.

Gall bladder physiological anatomy

The gall bladder is a tear drop shaped bile storage organ located between the right medial and quadrate liver lobes. The gall bladder wall consists of a mucosal layer, lamina propria, smooth muscle layer and a serosa. It is a complex structure with excretory and absorptive capacity. Bile is modified within the gall bladder. Contraction occurs due to specific vagal activation which can be chemically induced by cholecystokinin, gastrin, and motilin. Relaxation is caused by somatostatin, vasoactive intestinal polypeptide, nitric oxide and pancreatic polypeptide.

Physiology of bile

Bile components include bilirubin, which is mostly conjugated, lecithin, bile salts, other inorganic salts, potassium, sodium, bicarbonate, small amounts of copper and other metals, phospholipids, mainly phosphatidylcholine, deactivated toxins and drugs, some active hormones and cholesterol. The function of bile in the body is complex and involves digestion, hormonal metabolism and drug and toxin management. As far as digestion is concerned bile emulsifies fat and neutralises acid. Bile can be modified within the gall bladder. Supersaturation of cholesterol may be isolated to the gall bladder only and lead to cholesterol stones. Undersaturation of cholesterol and supersaturation of various salts may lead to the formation of other stones. Secondary bacterial infections are common and may exacerbate symptoms of gall bladder disease. This is important because not all gall bladder diseases involve the liver but many do. Differentiating isolated gall bladder disease is important for determining the likely outcome of gall bladder removal. In many cases gall bladder disease can spill over to include peri-cystic hepatic tissue or ascend in the biliary tracts to affect the liver diffusely or even originate in the liver in certain cases and go on to include the gall bladder.

Cholelithiasis

The clinical symptoms of cholelithiasis are similar to other abdominal disorders. Gall stones are most often seen in Miniature Schnauzers, Miniature Poodles, older animals and females. The most common stones are cholesterol stones, bilirubin stones, mixed stones, and calcium salt stones. Calcium stones are seen in cats but rarely in dogs because the canine gall bladder absorbs calcium. The cause of bile stones is not known. However imbalances between bile salts and cholesterol, supersaturation with any bile component and bile stasis may play a role. In human studies cholesterol stones are always associated with cystic bile supersaturation of cholesterol even though choledocal cholesterol levels may be normal. In these specific cases the primary disease is isolated to the gall bladder and correction of any downstream obstruction together with removal of the gall bladder results in clinical improvement. 75% of gall stone cases culture positive for gram negative bacteria.

Predisposition to Supersaturation

Gall bladder dyskinesia, post cystic obstructive disease, hypercholesterolaemia, hypertriglyceridaemia, hyperbilirubinaemia, endocrine disease, decreased gall bladder cholesterol and other bile component absorption, decreased gall bladder cholesterol and other bile component excretion are all predisposing factors to supersaturation of bile.

Clinical signs cholelithiasis

Some gall bladder choleliths are asymptomatic or at least under our radar for symptoms. If clinical signs are present as already mentioned they are often similar to other abdominal diseases. Symptoms may arise from gall bladder inflammation and infection. These symptoms would include fever, abdominal pain, anorexia or hyporexia, nausea and/ or vomiting, jaundice and secondary hepatic disease (ascites, bleeding tendency, hypoalbuminaemia, raised liver enzymes, systemic inflammation, shock). Signs related to biliary tract obstruction includes acholic stools, gall bladder distention (pain), jaundice, signs of peritonitis with gall bladder or bile duct rupture (diffuse abdominal pain, inflammatory exudate with bile) and sign of secondary hepatic disease.

Differential diagnosis cholelithiasis

As mentioned earlier the clinical symptoms of cholelithiasis are similar to other abdominal disorders. The more common of these are pancreatitis, gastroenteritis, gastrointestinal foreign bodies, abdominal neoplasia and most other hepatic diseases.

The Diagnosis of Cholelithiasis

Nonspecific findings

The eventual diagnosis of cholelithiasis would entail imaging gall stones, either radiographically or more commonly using ultrasonography. Nonspecific biochemical findings include a stress leukogram or a left shift neutrophilia, anaemia of chronic disease, bilirubinuria, elevated total bilirubin, elevated ALP, elevated GGT, elevated ALT, elevated AST, hypercholesterolaemia, prerenal azotaemia, hypoalbuminaemia, hypoglycaemia. A left shift neutrophilia is commonly associated with a bile duct rupture.

Bilirubin

Hyperbilirubinaemia is inconsistent but common in CL and CDL. Bilirubinuria is a potential normal finding in the dog but always an abnormal finding in the cat. Bilirubin is derived from the breakdown of haemoglobin and is conjugated with glucuronide in the liver. The preferred method of excretion is in the bile. Causes of significant bilirubinuria is, haemolysis as a result of pre-hepatic overload, liver disease, extra-hepatic biliary obstruction, fever and starvation. An example of a bilirubin cast is seen on the lower left corner of the slide.

Diagnostic imaging Radiographs

Radiographs are indicated but choleliths are often not radio-opaque due to lack of calcium salts. Gas radiolucencies with secondary anaerobic or facultative anaerobic infections causing an emphysematous cholecystitis may be seen.

Ultrasonography

On ultrasound the bladder wall is thickened with an irregular mucosal lining. Biliary tree dilation takes 5-7 days to occur. It is seen as multiple round to elongated anechoic bile vessels depending on the angle of section. Choleliths may be visible within the bile ducts. Pericholecystic fluid may be noted in some severe cases and may represent an inflammatory exudate or bile leakage. Choleliths and choleliths have a hyperechoic surface with a complete distal shadow depending on their density. Ultrasound guided percutaneous transhepatic bile aspiration and choliopancreatography techniques are possible. Bile aspiration can be used for cytology and culture.

Endoscopy

Endoscopic retrograde choliopancreatography can be non-invasively performed to detect bile duct obstruction.

Scintigraphy

^{99m}Tc-diisopropyl iminodiacetic acid is excreted in the bile and can be used to assess bile movement in the liver.

MRI / CT

These are useful imaging techniques especially for defining the extent of multiple obstructive, inflammatory and neoplastic lesions.

Medical treatment cholelithiasis

In one study in dogs with cholelithiasis dogs that underwent medical treatment only, unsuccessful abdominal exploration to locate the problem, cholecystojejunostomy, choledochotomy or sphincter of odditomy all died. Full resolution by medical treatment alone is rarely successful. Initial stabilisation and pre-surgical diagnostics and preparation is the physician's role in this disease. Medical treatment includes supportive care with IV fluids, electrolytes, antibiotics (culture and sensitivity based), analgesia and

anti-nausea medication. If completely obstructed call the surgeon to re-establish patency

Surgical Treatment

In one study cholecystectomy was associated with 86% survival, cholecystotomy with 50% survival and cholecystectomy in combination with choledochotomy with only 33% survival. The numbers were very small. The type of procedure performed depends on the location of the obstruction and integrity of the surrounding structures. Bile duct choleliths must be removed and may require choledochotomy. If the duct is stenotic or at high risk of developing stenosis a surgically placed choledochal stent is indicated. If the lesion is “unstentable” a cholecystoduodenostomy or a cholecystojejunostomy is indicated.

References

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