CHOLECYSTOLITHIASIS IN A LABRADOR BITCH: 
A CLINICAL REPORT ON A RARE CASE

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ABSTRACT

Canine Liver Disorders are one of the major challenges in small animal practice. While hepatic parenchymal diseases are common, disorders of gall bladder and biliary tree are uncommon in dogs. A rare case of cholecystolithiasis in a Labrador dog and its clinical presentation and outcome was recorded. Abdominal ultrasonographic examination of the dog documented a normal liver and kidney architecture; however in the gall bladder, there was a hyper echoic mass with sludge measuring around two centimeter in diameter, confirmed the presence of Cholecystolith.

Key words: Canine Liver, Ultrasonography, Cholecystolith

The commonest affections of gall bladder and biliary tree included cholangitis, cholecystitis, cholecystolithiasis and choledocholithiasis. Among these the incidence of cholecystolithiasis is very less as evidenced from the published veterinary literatures. The incidence of disorders restricted to the gallbladder and the biliary tree is low, when compared with the parenchymal hepatic affections that occurs in dogs (Veronica et al. 2006). The extrahepatic biliary tract obstruction (EHBO) in dogs is caused most frequently by pancreatic disease, biliary carcinoma, pancreatic carcinoma and intestinal neoplasia. The biliary or intestinal inflammations are less commonly recognized in clinical practice. Cholelithiasis is considered an uncommon cause of EHBO, as canine bile has a very low lithogenic index, because of the low concentration of cholesterol and free Ca+ present in it. (Willard & Fossum, 2005; Veronica et al.2006). Choleliths are often fortuitous findings at necropsy or during imaging. They may often cause no problems but may be associated with cholecystitis or rupture (Willard & Fossum, 2005). This report describes about a clinical case of a cholecystolithiasis in a Labrador dog and its outcome.

Case History and Clinical Presentation

A six year old intact female Labrador retriever dog was presented to the Small Animal Medical Outpatient Clinic of the Madras Veterinary College Teaching Hospital, with the history of anorexia, vomiting, difficulty in walking, clay coloured faeces, dark brownish urine and panting.
Physical examination revealed a dull and depressed animal with weakness and an obese body condition (45 kg). The conjunctival mucous membrane was congested and popliteal lymphnodes were enlarged. Vital signs of the animal included a normal temperature (103.4°F), normal pulse rate (140/min) and panting type of respiration. Abdominal palpation revealed moderate pain on right cranial ventral abdomen. The fecal examination revealed clay coloured feces with no parasitic ova. A complete blood count and serum biochemistry revealed near normal haematological values and an elevations in the liver enzymes such as Serum glutamic pyruvic transaminase (92 IU/L), Serum alkaline phosphatase (1648 IU/L) and Gamma glutamyl transferase (35U/L) levels. Abdominal radiographic assessment was unremarkable. Lateral radiography of the thorax revealed mild bronchial infiltration. The dog was subjected to Electrocardiography and it showed low voltage QRS complexes with normal sinus rhythm. Based on above findings, tentatively the case was diagnosed as hepatitis.

Abdominal ultrasonographic examination of the dog documented a normal liver and kidney architecture; however in the gall bladder, there was a hyperechoic mass with sledge measuring around two centimeter in diameter suggestive of Cholecystolith (Fig.1). This confirmed the diagnosis as cholecystolithiasis. As the owner preferred for a nonsurgical management option, the case was conservatively managed with Amoxicillin and Cloxacillin (10 mg/kg i/v two times daily), Metronidazole (10mg/kg i/v), Dextrose Normal Saline (10ml/kg i/v), Ondansetron (0.1 mg/kg i/v) for five days along with Ursodeoxycholic acid (15 mg/kg/ day divided over two doses), vitamin E (10 IU/kg/ day) and Silymarin (20 mg/kg/day) orally for two weeks. Review after two weeks of therapy, revealed that the dog has improved and was doing well.

The occurrence of cholecystoliths are rarely recognized in small animal practice and published in the Veterinary literature. On exploration of the previous reports on cholecystoliths in canines, there were hardly four or five, which are found valuable. The present case is the only one recorded at this hospital in the recent times. The pathogenesis of cholelithiasis in dogs is unknown. Proposed causes for formation of choleliths include trauma, biliary stasis, diet alterations, cholecystitis and parasitic or bacterial biliary infection (Veronica et al. 2006). Choleliths in dogs, as in human beings, have been classified as cholesterol, pigment or mixed. The stones were reported to appear dark brown or black in colour, and were soft. (Rothuizen, 2005). Mixed or cholesterol choleliths were reported to contain more than 70% cholesterol monohydrate plus an admixture of calcium salts, bile acids and pigments, proteins, fatty acids and phospholipids (Willard & Fossum, 2005). Although people commonly developed dietary induced cholesterol gallstones , canine gall stones were reported to usually contain bilirubin, calcium and mucin (Prowse, 1984). Parasites of gall bladder, bile ducts or both are seldom diagnosed in small animal practice. (Willard & Fossum, 2005).

In dogs with cholecystoliths, if associated cholecystitis is present, they may exhibit vomiting; icterus and anorexia, are most common complaints. Inconsistent fever, abdominal discomfort and ascites may also present. ( Willard & Fossum, 2005).All of such findings were observed in the present case. Increased levels of Alanine aminotransferase are commonly observed when inflammation ascends to hepatic parenchyma. Increased serum alkaline phosphatase with or without hyperbilirubinemia was typically noticed in extrahepatic biliary tract obstruction. Hypercholesterolemia was observed secondary to obstructions. (Willard & Fossum, 2005).

Gall stones are typically identified by ultrasonographically as hyperechoic foci or by the observation of acoustic shadowing originating from
the gall bladder. (Willard & Fossum, 2005) as documented in this case. Gallbladder sludge is considered a precursor of cholelithiasis in human. However, the higher prevalence of gallbladder sludge, and the lower prevalence of cholelithiasis in dogs, as compared to humans, suggest that biliary sludge in dogs rarely results in cholelith formation (Catherina et al. 1998). Rothuizen (2005) reported that the non-calcified stones may resolve in response to oral medication with Ursodeoxycholic acid along with the Silymarin and Vitamin E. Surgical options for the cholelithiasis in dogs are reported to be cholecystotomy, choledochotomy and, finally, by cholecystectomy and biliary diversion (Fossum, 2002). The improvement in the dog’s health status observed after two weeks of therapy in this case indicated the beneficial effects of the medical therapy in the clinical management of cholecystolithiasis in dogs.

REFERENCES


Figure 1:
Abdominal Ultrasound Image of the Cholecystolith