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Case Report

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Gallbladder mucocele in a dog: an ultrasonography and pathology report

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Abstract: A 9-year old male, German shepherd dog was presented to the Madras Veterinary College Teaching Hospital with a history of a chronic ailment that had increased over the previous few months. A diagnosis of chronic renal failure and hepatitis was made and abdominal ultrasonography revealed the presence of gallbladder mucocele (GBM). The etiology, diagnosis, necropsy, and histopathological lesions were discussed.

Key words: Gallbladder mucocele, ultrasonography, renal failure, hepatitis

1. Introduction

A gallbladder mucocele (GBM) is an inappropriate accumulation of mucus or inspissated bile in the gallbladder lumen. Once considered a rare condition, prior to the 1990s, GBMs are becoming an increasingly common diagnosis especially in older, small to medium-sized dogs (1). The extension of bile-laden mucus into the cystic, hepatic, and common bile ducts may result in various degrees of extrahepatic biliary obstruction and is one of the most common causes of extrahepatic biliary disease. GBM may result from chronic injury to the epithelial lining of the biliary system since hypersecretion of mucin is the typical physiologic response of any epithelial lining to injury (2). Ultrasonographic evidence of an enlarged gallbladder with an immobile stellate or finely striated bile pattern is diagnostic for a GBM (3). Recently, Shetland sheepdogs were identified as a breed that is predisposed to GBM formation, suggesting a genetic predisposition (4).

2. Case history

A 9-year old male German shepherd dog was admitted to the Madras Veterinary College Teaching Hospital with a history of chronic ailments, anorexia, vomiting, dullness, depression, and alopecia. The physical examination revealed icteric visible mucus membrane and evinced pain on abdominal palpation. Blood, serum, and urine samples were collected for routine hematology, serum biochemical analysis, and urinalysis. Abdominal ultrasonography was performed. Due to the progressive development of renal failure signs and a steady increase in SGPT, BUN, and creatinine, the animal was euthanized on humane grounds with the consent of the owner. Appropriate tissue samples were collected in 10% formalin for histopathological studies. A sterile swab was taken from the contents of the gallbladder.

3. Results and discussion

The ultrasonography of the abdomen revealed a mixture of stellate and kiwi fruit-like patterns on the gallbladder (Figure 1). The serum biochemical analysis revealed elevated levels of SGPT (246 U/L), BUN (130 mg/dL), and creatinine (4.74 mg/dL). A complete hematology revealed normocytic normochromic anemia and neutrophilia. The urine sample was positive for protein. Culture examination revealed no bacterial growth.

On postmortem examination, grossly, the gallbladder was thickened, gray-white, greatly distended (11 × 6 cm), and tensed (Figure 2). On incision, at the opening of the biliary passage, there was thickened semisolid greenish black bile. The rest of the gallbladder was severely thickened and contained a thick white gelatinous mucus material mixed with scanty inspissated greenish bile occupying the lumen (Figure 3).

The liver was enlarged; the borders were rounded, mottled, gray-brown with a yellow tinge, and hard to incise. Hepatic lymph nodes were enlarged and edematous. The kidney revealed multiple cysts of varying sizes ranging from 0.2 to 1 cm in diameter on the cortex of both kidneys. The stomach was completely empty and coated with mucus. The intestinal serosal surface had focal

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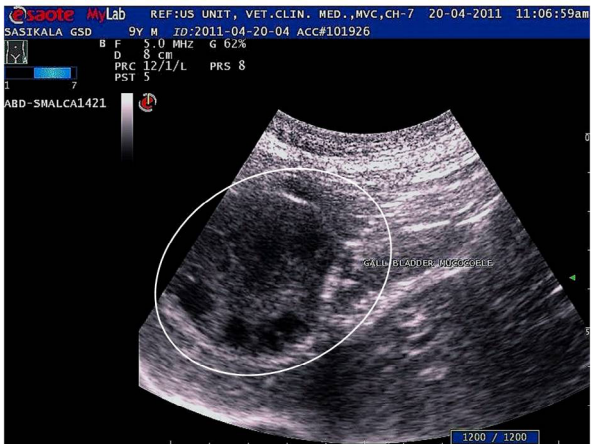

Figure 1. Ultrasonography of liver showing mixture of stellate and kiwi fruit-like pattern of gallbladder.

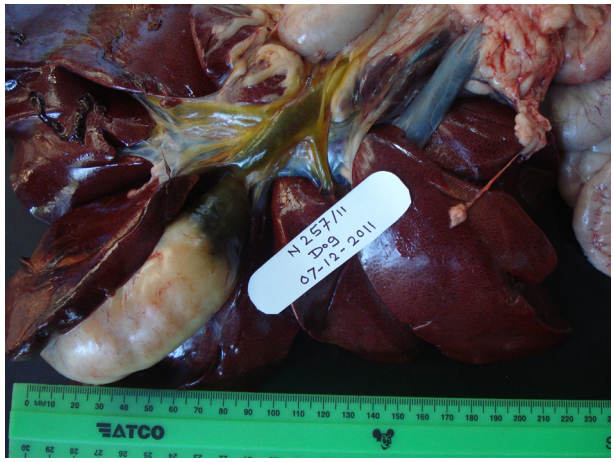

Figure 2. Gallbladder: white, thick, and distended.

congestion and the mucosal surface had multifocal ulcers in the duodenal part.

Microscopic examination of the gallbladder revealed mucosal cystic hyperplasia characterized by the presence of large cysts filled with mucinous material. The cysts were lined with low cuboidal epithelium (Figure 4).

Liver histopathology revealed a periportal fibrosis (Figure 5) with moderate bile duct hyperplasia (Figure 6), hepatic vacuolation, and cholestasis. The kidney revealed a chronic nephritis, moderate tubular degeneration, and multiple cysts.

The principal gross abnormality associated with GBM was gallbladder enlargement secondary to the build up of an excessive amount of mucus within the lumen (5). Ultrasonographically, the finely striated and stellate bile pattern was consistently associated with macroscopic evidence of a GBM. The characteristic appearance of this bile pattern was important to distinguish between GBM and biliary sludge as reported by Besso et al. (3). This was highly appreciated in this case both ultrasonographically and macroscopically. Studies reported that the odds of mucocele formation in dogs with hyperadrenocorticism were 29 times those of dogs without the condition (6). Surgical correction via cholecystectomy is recommended for dogs diagnosed with a GBM.

The characteristic histological finding in dogs with GBM was mucosal epithelial hyperplasia (5) as observed in this case. Our findings on hepatic histopathology were in accordance with those of Newell et al. (7). They observed inflammatory portal infiltrate, bile duct hyperplasia, vacuolation, and varying degrees of fibrosis in 76% of the cases with GBM. While the exact etiology and pathogenesis of GBM are unknown, histologic findings have consistently demonstrated a dysfunction

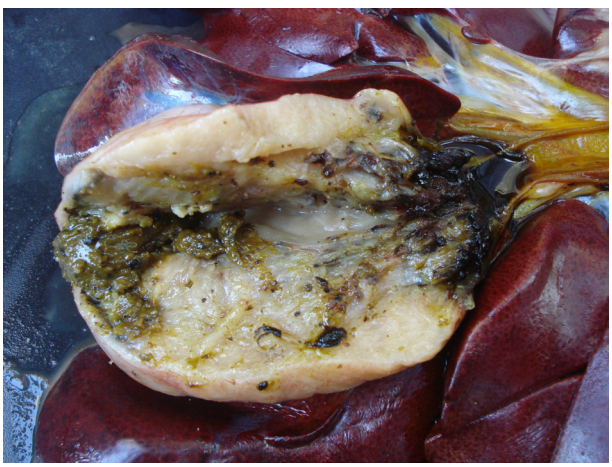

Figure 3. Gallbladder, opened, showed thick white gelatinous mucus material mixed with scanty inspissated greenish bile.

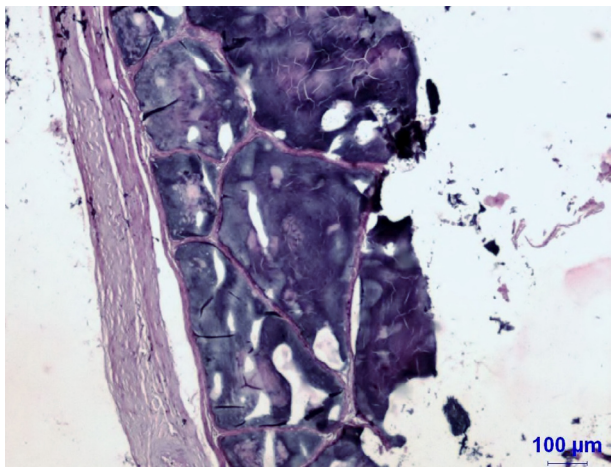

Figure 4. Liver: gallbladder mucosal cystic hyperplasia with multiple cysts. H&E stain.

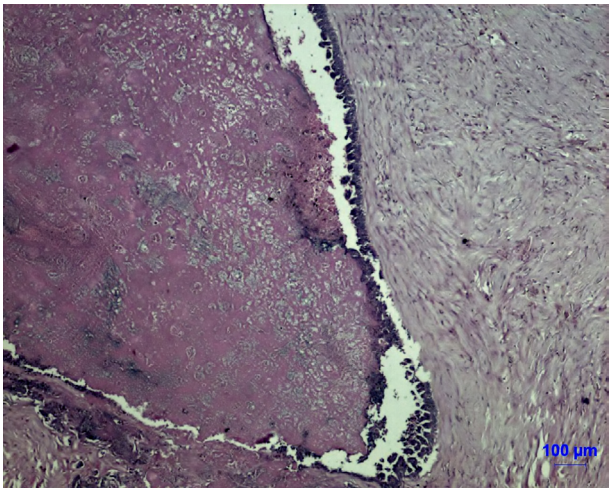

Figure 5. Liver: periportal fibrosis and bile duct epithelial hyperplasia. H&E stain.

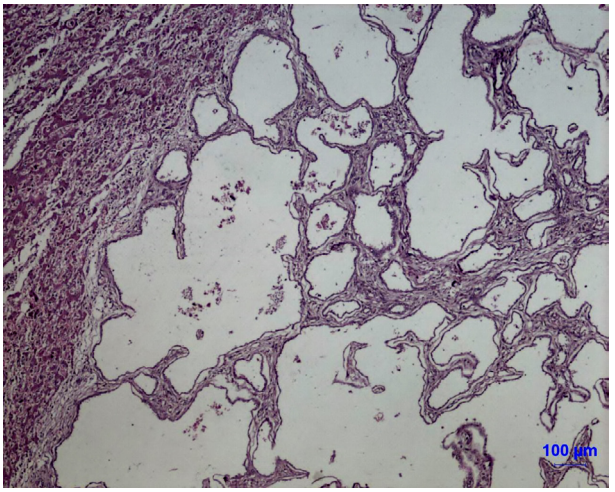

Figure 6. Liver: bile duct hyperplasia. H&E stain.

and proliferation of the mucus secreting glands in the gallbladder wall. The secreted mucus caused distension, obstruction, and in some cases rupture of the gallbladder (4). The elevated serum enzymes clearly indicated the involvement of the kidney and liver.

As reported previously, GBM may result from cholecyctitis (8) or necrotizing cholecyctitis (9). However, our results did not support an inflammatory or bacterial etiology.

Cholecystoduodenostomy and cholecystectomy appeared to be the acceptable treatments for GBM (10). We could not attempt treatment because of the chronic renal failure and the poor body condition of the animal. Recent studies suggested that an insertion mutation in the ABCB4 gene was associated with gallbladder mucocele formation in dogs and might be useful for studying potential medical and/or dietary treatments for ABCB4 associated hepatobiliary diseases in people (2).

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