Oral presentations

Gallbladder mucocoele in the Border terrier: another manifestation of gluten intolerance in the breed?

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OBJECTIVES

To determine if there is evidence of gluten intolerance as the cause of gallbladder mucocoele (GBM) formation in Border terriers (BTs), a breed previously known to suffer from paroxysmal-dyskinesia a systemic manifestation of gluten-intolerance. Testing of serum anti-canine gliadin IgG (AGA IgG), anticanine transglutaminase-2-IgA autoantibodies (TG2 IgA) and cholecystokinin levels

(CCK) were performed. We hypothesised that there would be serological evidence of gluten intolerance compared to controls with an increase in AGA IgG and TG2 IgA and a decrease in CCK.

METHODS

Medical records from two referral hospitals were obtained from 2011 and 2019 to identify BTs with GBM, non-BTs with GBM and control BTs with non-biliary disease. Enzyme-linked immunosorbent-assays were performed on stored serum samples for AGA IgG, TG2 IgA and CCK. Statistical analysis was performed using Kruskal-Wallis to identify differences between the groups.

RESULTS

Fifteen BTs with GBM, 17 non-BT with GBM and 14 control BT with non-biliary disease were identified. Median TG2 IgA in BTs with GBM was 0.73 which was significantly greater than in control BTs at 0.41 ($p < 0.045$). Median CCK in BTs with GBM was 13pg/ml, which was significantly lower than in control BTs at 102.6pg/ml ($p < 0.016$).

STATEMENT (CONCLUSIONS)

This is the first study to suggest a mechanism for GBM formation in the BT. The finding of impaired CCK secretion and increased TG2 IgA is homologous to gallbladder disease identified in human coeliac disease. Therefore the results suggest gluten intolerance as a possible cause of GBM formation in BTs.

A retrospective study assessing ultrasonographic progression of canine biliary sludge (BS) over time

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OBJECTIVES

To establish a relationship between the presence of BS and development of gall bladder mucocoele (GBM) in dogs.

METHODS

Cases were identified by searching the diagnostic imaging database. Inclusion criteria included presence of BS, with at least one follow up ultrasonographic examination. Signalment, length of follow up, number of ultrasonographic examinations, concurrent disease, serum biochemistry, medications and changes to sludge character were recorded for analysis.

RESULTS

23 dogs met inclusion criteria. 11/23 (47%) had increased alkaline phosphatase and 12/23 (52%) had hypercholesterolaemia at presentation. 20/23 had concurrent disease. 3/23 had elevated liver enzymes only. Mean number of repeat examinations was 3.5 (range 2-9) and 10/23 cases had follow-up longer than 12 months (range 0.75-92 months). 14/23 (60%) cases were prescribed ursodeoxycholic acid (UDCA). 6/23 cases (26%) [4 from the UDCA treatment group] saw decreases in volume of BS. 2/23 cases had complete resolution. 6/23 saw increases in amount or organisation of BS [5/6 despite UDCA treatment]. 9/23 cases had static levels of BS. 1 case progressed to GBM 2 years after BS diagnosis, and this dog

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