LIITE 4: Artikkeli – Chronic Hepatitis in the English springer spaniel

CHRONIC HEPATITIS IN THE ENGLISH SPRINGER SPANIEL

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Canine chronic hepatitis (CH) is seen with an increased incidence in certain breeds, including the Bedlington, West Highland White and Skye Terrier, Cocker Spaniel, Doberman, Dalmatian and Labrador Retriever. In some breeds CH is thought to be associated with abnormalities in copper metabolism. In the majority of cases of CH however, the aetiology is poorly understood. The authors have recently noted a previously undocumented increased incidence of CH in the English Springer Spaniel (ESS). The purpose of this study was to describe the history, clinical signs, clinicopathologic abnormalities and outcome in such cases.

ESS presented to referral and general practices in the UK and Norway for investigation of elevated liver enzymes, clinical signs of liver disease and histopathological confirmation of CH were identified. Clinical, laboratory and pathological data were obtained from case records or by contact with the referring veterinarian. Formalin fixed or paraffin embedded liver tissue was obtained for review by one of the authors using a panel of seven liver-specific stains including rubeanic acid.

Thirty four cases were identified. Mean age was 3 years 4 months (range 7 months - 9 years) and 24 female and 10 male dogs were represented. 28/34 dogs had been vaccinated within the proceeding 12 months. Clinical signs at presentation included vomiting, depression, lethargy and anorexia. Pyrexia was documented in 12 dogs and 26 dogs were jaundiced. Biochemical changes in all cases included moderate to marked elevations in ALP and ALT and mild to moderate elevations in total bilirubin. Other variable findings included, hypocholesterolaemia, hypercholesterolaemia, hypoalbuminaemia, decreased urea, elevated bile acids and prolonged coagulation times. Neutrophilia was documented in 12 of 21 dogs. Results of abdominal imaging were non-specific. Histological examination of liver tissue demonstrated changes consistent with CH including hepatocellular apoptosis or necrosis, a variable mononuclear or mixed inflammatory infiltrate, regeneration and fibrosis. There was no evidence of increased copper accumulation in any dog. Treatment included one or more of dietary modification, antioxidants, antibiotics, ursodeoxycholic acid and corticosteroids. Follow up data was available in 26 dogs and 16 of these died or were euthanased within a mean of 4 months following initial diagnosis. The median survival time of the remaining dogs was 7 months.

This study suggests that the ESS might be predisposed to a type of CH without copper accumulation which carries a poor prognosis. Further investigations in to the aetiology and possible genetic basis of this condition are indicated.