Oral presentations

Comparing the prevalence of pulmonary hypertension in Cavalier King Charles Spaniels and other breeds in dogs with myxomatous mitral valve disease

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OBJECTIVES

To determine if Cavalier King Charles spaniels (CKCS) are more likely to develop pulmonary hypertension (PH) as a result of myxomatous mitral valve disease (MMVD) than dogs of other breeds and also assess whether PH impacts upon survival. A secondary objective was to explore any association between the presence of macroplatelets and PH.

METHODS

Retrospective review of 187 dogs diagnosed with MMVD identified from electronic patient records, 94 CKCS and 93 non-CKCS. Eligible dogs were >5 years of age, weighed <20 kg and had structural cardiac changes due to MMVD on echocardiography (nodular thickening of the mitral valve, dilated left atrium and ventricle) with or without congestive heart failure (CHF). Data retrieved were analysed for the presence of PH (based on tricuspid regurgitation velocity >2.8 m/s), CHF, selected echocardiographic variables and macroplatelets. Survival analysis was also performed, on both cardiac and all-cause mortality.

RESULTS

71/94 CKCS and 52/93 non-CKCS developed PH ($p=0.005$). 57/94 CKCS and 45/93 non-CKCS were in left-sided CHF ($p=0.093$). PH was associated with a greater likelihood of CHF ($p<0.001$) and death (both cardiac ($p<0.001$) and all-cause mortality ($p=0.011$)). CKCS were more likely to experience cardiac death than non-CKCS ($p=0.004$). Macroplatelets were not associated with PH development ($p=0.376$).

STATEMENT (CONCLUSIONS)

CKCS were more likely than non-CKCS to develop PH due to MMVD in this study population. Further investigations are required to determine the reason for this. Macroplatelets were not associated with the development of PH.

Expression of 11-beta hydroxysteroid dehydrogenase 2 is not altered in canine congestive heart failure

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OBJECTIVES

The enzyme 11-beta hydroxysteroid dehydrogenase 2 (11 β HSD2) in the distal nephron protects the mineralocorticoid receptor from activation by cortisol,

allowing it to interact with aldosterone. Mutations of 11 β HSD2 in humans have been shown to cause apparent mineralocorticoid excess, which is characterised by sodium and water retention. Canine congestive heart failure (CHF) is also associated with sodium and water retention. This may, in part, be explained by dysregulation of 11 β HSD2 driving cortisol-stimulated mineralocorticoid receptor activity. Therefore, it was hypothesised that expression of renal 11 β HSD2 is downregulated in canine CHF.

METHODS

Tissue was collected from the renal medulla of dogs that had been euthanased for behavioural reasons, non-cardiac disease or CHF. Dogs euthanased for behavioural reasons were used as a healthy control population. Animals with renal disease or hyperadrenocorticism were excluded. Quantitative polymerase chain reaction (qPCR) was then performed to detect differences in relative 11 β HSD2 expression. One-way ANOVA with Tukey's post-hoc test was used to evaluate differences in δ Ct between groups referenced to GAPDH and mitochondrial ribosomal protein S25.