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

**Objectives**

The enzyme 11-beta hydroxysteroid dehydrogenase 2 (11βHSD2) in the distal nephron protects the mineralocorticoid receptor from activation by cortisol, allowing it to interact with aldosterone. Mutations of 11βHSD2 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 11βHSD2 driving cortisol-stimulated mineralocorticoid receptor activity. Therefore, it was hypothesised that expression of renal 11βHSD2 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 11βHSD2 expression. One-way ANOVA with Tukey’s post-hoc test was used to evaluate differences in δCt between groups referenced to GAPDH and mitochondrial ribosomal protein S25.

**Results**

There was no difference (P=0.15) in the relative renal medullary expression of 11βHSD2 between dogs euthanased for behavioural reasons (n = 10), non-cardiac disease (n = 6) and CHF (n = 9). Dogs euthanased for CHF had underlying dilated cardiomyopathy or atrioventricular valve dysplasia.

**Impact**

There is no evidence that modification of expression of 11βHSD2 contributes to the development of canine CHF. However, translational and post-translational modifications of 11βHSD2 and also breakdown of cortisol may be altered, and this warrants further investigation.

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