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Serum biochemical and urinary parameters of renal impairment in dogs with primary chronic enteropathy

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- 1 Evaluation of serum biochemical and urinary parameters suggesting renal involvement in a
- 2 population of dogs with primary chronic enteropathy
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8 Approximately a half of IBD human patients show extra-intestinal manifestations, in which 4-23%

may develop renal and urinary involvement. These findings may be linked to several conditions,

such as the immune-system response of the primary chronic enteropathy (CE), reduction in short-

chain fatty acids, or endotoxemia. No specific studies have been conducted in dogs, except for those

describing familiar protein-losing nephropathy and enteropathy in soft-coated wheaten terriers.

13 The aim of this study was to describe alterations of selected serum biochemical and urinary

parameters suggesting renal injury in dogs with CE.

15 Retrospective multicentric (University of Pisa and Turin) study including dogs with CE. CE

diagnosis was made after the exclusion of intestinal diseases of other etiologies and extra-intestinal

diseases. Dogs with history of previous kidney or low urinary tract diseases (previous

clinicopathological finding and/or imaging alterations) and with severe proteinuria (urine protein-

to-creatinine ratio (UPC)>2) were excluded. Canine Chronic Enteropathy Activity Index Score

(CCECAI), muscular condition score (MCS; 3-point scale), serum albumin, urea, creatinine,

presence of glycosuria, proteinuria (UPC>0.5) and urinary casts were recorded for each dog. Dogs

with albumin <2.7 mg/dL were classified as protein-losing enteropathy (PLE). Dogs with showed

glycosuria, proteinuria and/or urinary casts were classified as having "kidney injury". Mann-

Whitney u-test was used to compare CCECAI of dogs with and without kidney injury. Chi-square

test was used to evaluate the association of PLE and presence of kidney injury, and proteinuria

26 (UPC>0.5).

One-hundred-six dogs with CE were included. Fifty-two dogs (49%) had mild-to-severe reduction in MCS. Only 6/106 dogs (6%) had azotemia (median creatinine 1.6 mg/dL; range 1.5-2.4 mg/dL), whereas 40/106 dogs (38%) showed kidney injury. In particular, 2 dogs had glycosuria, 23 dogs had proteinuria, and 23 dogs had urinary casts. CCECAI was not different between dogs with, and without kidney injury (both median=4; p=0.9). Forty-four dogs were classified as PLE. The prevalence of kidney injury was not different between PLE, and not-PLE (p=0.3) dogs, whereas PLE dogs showed a higher prevalence (61%) of proteinuria, than non-PLE dogs (p=0.03 OR 2.8 95%CI 1-6.8). Serum markers of kidney injury should be interpreted with caution in CE dogs, since approximately half of our dogs showed a reduction in muscular mass. On the other hand, assessment of urinary markers of "kidney injury" may be useful and advisable, especially due to the high risk of proteinuria in PLE dogs.