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SUBCLINICAL EHRLICHIOSIS: MAY CAUSE KIDNEY ALTERATIONS?

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We hypothesized that renal glomerulopathy is a common finding in dogs with subclinical ehrlichiosis and pro-inflammatory cytokines may be associated to renal injury. The aim of this study was to evaluate renal cortex biopsies in 15 dogs with subclinical ehrlichiosis diagnosed by PCR and enzyme-linked immunosorbent assay (ELISA) and in 17 healthy dogs as a control group. Dogs with presence of clinical signs, comorbidities and/or azotemia were excluded. Biopsy material was examined by light microscopy (LM). Sections were stained with hematoxylin and eosin, periodic acid Schiff, Jones methenamine silver, Masson´s trichrome, and Congo Red. Cytokine quantification of tumor necrosis factor alpha (TNF-α), interferon gamma (INF-γ) and interleukin 6 were assessed through ELISA using commercial kits specific to dogs (Milliplex), according to the manufacturer's instructions. LM abnormalities were identified in 14 dogs (93.3%) from the ehrlichiosis group, but most findings were subtle. Mesangial cell proliferation (40.0%), synechiae (40.0%), globally sclerotic glomeruli (33.3%), ischemic glomeruli (33.3%), focal segmental glomerulosclerosis (FSGS) (33.3%), focal thickening of the glomerular basement membranes (26.7%), hydropic degeneration (26.7%) and interstitial fibrosis and tubular atrophy (IFTA) (20.0%) were frequent. Cytokines was increased in subclinical phase on levels of TNF-α, INF- γ, and IL-6 in comparison to control. These results suggest that TNF-α, INF- γ and -IL-6 may be involved in the pathogenesis of tubule-glomeruli injury and subclinical ehrlichiosis may lead in chronic kidney disease (acknowledgement to FAPESP for financial support - #2014/21506-2).