Short Communication

ULTRASONOGRAPHIC AND HISOPATHOLOGICAL CHANGES IN DOGS WITH CHRONIC RENAL DISEASE

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ABSTRACT

One hundred and two geriatric dogs presented with chronic renal disease were further examined for ultrasonographic and histopathological alterations. Hyperechoic cortex, indistinct corticomedullary junction and altered renal architecture were observed in 80 per cent dogs. The remaining 20 per cent dogs had no significant changes. The histopathological lesions detected in 12 dogs subjected to post mortem examination were interstitial fibrosis, moderate to severe plasma cell infiltration of interstitium, calcification of tubules, loss of tubular epithelial cells, atrophy of glomeruli and thickening of basement membrane.

Key words : Dogs, Chronic ronal disure, histopathological changes

INTRODUCTION

Continuous improvement in veterinary care has resulted in an expanding geriatric pet population which in turn has increased the need to identify and understand conditions of older animals. Chronic kidney disease (CKD) is one of the leading problems in aged dogs (Polzin *et al.,*2000). Nephron damage associated with CKD is usually irreversible and the cause is often difficult to determine. Because of the interdependence of the vascular and tubular components of the nephron, the end point of irreversible glomerular and tubular damage is the same. The main goal of early diagnosis of renal disease and renal failure in dogs is to enable timely application of therapeutic interventions that may slow or halt the disease progression. With this backdrop a study was undertaken to assess the usefulness of ultrasound and histopathology in the detection of chronic renal disease in dogs.

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MATERIALS AND METHODS

One hundred and two dogs presented to the Madras Veterinary College Teaching hospital with clinical signs suggestive of chronic renal disease such as weight loss, anorexia, polyuria and polydipsia, pale mucous membrane, vomiting and melena were taken up for the study. The selected animals were classified into four groups based on International Renal Interest Society (IRIS). Accordingly , dogs having serum creatinine concentration of < 1.4 mg per cent , 1.4-2 mg per cent, 2.1 - 5 mg per cent and >5 mg per cent were categorized as Stage I,(12 cases), Stage II(12 cases), Stage III (24 cases) and Stage IV- (54 cases) respectively. Nephrosonographic examination was performed as per standard procedure (Nyland *et al*., 1995). Postmortem examination was conducted in 12 dogs from stage IV, which died despite supportive medical management. Tissue samples from kidneys were collected in 10 per cent formalin for histopathological studies.

RESULTS AND DISCUSSION

In the present study all the dogs of stage I and stage II showed no changes in nephrosonogram. In stage III and stage IV group put together 80 per cent of dogs (63/78) showed nephrosongraphic changes. The common changes observed were hyperechoic cortex (25 %), indistinct or absence of corticomedullary junction (78%) and altered renal architecture (65%). A normal ultrasonographic picture does not entirely rule out renal disease (Walter *et al*., 1987). End stage kidneys were typically small, irregular and diffusely echogenic with poor visualization of corticomedullary junction and internal renal architecture.

Nyland *et al* (1995) stated that ultrasonographic abnormalities associated with glomerular / interstitial nephritis were mild to moderate cortical hyperechogenicity and decreased corticomedullary demarcation.

In the post mortem examination a moderate to severe adhesion of the capsule to the cortical surface, pitting and granularity of the cortex were noticed in 83.33 per cent (10/12) cases. Irregular cortical surface, with few pale foci on the cortex was also recorded. Confer and Panciera (1997) recorded similar findings in their work. Histopathological examination revealed chronic interstitial nephritis in six dogs, glomerular nephritis in four dogs and focal interstitial nephritis in two dogs. The lesions detected were interstitial fibrosis, moderate to severe plasma cell infiltration of interstitium, calcification of tubules, loss of tubular epithelial cells, atrophy of glomeruli and thickening of basement membrane. Cook and Cowgill (1996) found that 52 per cent glomerular disease and 48 per cent non glomerular disease out of 111 cases of chronic renal disease. Grauer (2005) described histopathologic changes in chronic renal disease as combination of a loss of tubules with replacement fibrosis, mineralization, glomerulosclerosis , glomerular atrophy and foci of mononuclear cells within the interstitium. These histopathologic changes were not process specific, therefore the underlying cause of the renal disease were usually unknown.

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