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Gross and histopathological changes in canine chronic kidney disease

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Abstract
Chronic Kidney Disease brings structural and functional changes in one or both the kidneys. The structural changes include changes in glomeruli, renal tubules, interstitial space, renal pelvis and vascular tissue. Total of 9 renal tissue samples were collected from dogs which die due to CKD and submitted to laboratory for histopathological examination. Histopathology examination of collected samples was performed with light microscopy using hematoxylin and eosin stain. On gross examination kidney size was reduced, shape was irregular and capsule was peeled off with difficulty. On histopathological examination changes like lymphoplasmacytic infiltration, tubular atrophy, dystrophic calcification and interstitial fibrosis were indicative of chronic nephritis.

Keywords: CKD, histopathology, gross changes

Introduction
Presence of structural or functional abnormalities in one or both kidneys for more than three months was defined as chronic kidney disease [1]. Structural changes are more remarkable as CKD progresses to more advanced stages. Previous study of Robinson [2] they observed reduction in both kidneys size by two-thirds of normal size, consistency was tough, cortex was pale and capsular surface was nodular. Further on histopathological examination they observed marked loss of functional nephrons, interstitial fibrosis was diffused and focal dense radial fibrosis was more evident in the renal medulla. Tubules were dilated with widespread with focal mineralisation of tubular epithelium and basement membranes. Renal glomerular size was reduced with segmental fibrotic areas, bowman's spaces was dilated. The present work was undertaken to study gross and histopathological changes in kidneys of dogs die due to chronic kidney disease.

Materials and Methods
During post mortem examination of dogs which die due to CKD, kidney specimens were collected. Collected specimens were immersed and fixed in 10% buffered formalin for histopathological analysis. Paraffin-embedded tissues were sectioned at 5 um thickness and stained with hematoxylin and eosin. All microscopic sections were evaluated by a veterinary pathologist.

Result and Discussion
In the present study on gross examination, kidneys size was reduced, shape was altered, color varied from pale to grey (Fig 1), were hard to cut and capsule peeled off with difficulty and hemorrhagic patches were seen in cortical area and at cortico-medullary junction (Fig 2). These gross changes were in accordance with earlier observations of Robinson [2] and Mshelbwala [3].

On histopathological examination of renal tissue common observations were lymphoplasmacytic infiltration in the interstitium (Fig. 3), tubular atrophy, decreased glomerular cellularity (Fig 4), dystrophic calcification (Fig 5) and interstitial fibrosis (Fig 6) indicative of chronic nephritis. These observations were in accordance with observations of Ajay [4] and Lothe [5].

CKD progression is due to the persistence of the original cause and/or to self-perpetuating mechanisms once the functional mass of the kidneys has been reduced to a critical value. There are multiple mechanisms proposed for the progression of CKD, which includes accumulation of immune complexes, increased glomerular filtration, coagulation defects, lipid

abnormalities and calcifications of renal tissue [6]. Irrespective of etiological agent, progression of CKD leads to release of multiple inflammatory mediators and subsequent renal scarring as a result of fibrogenic actions. These changes finally lead to lymphoplasmacytic infiltration in the interstitium, tubular atrophy, decreased glomerular cellularity, dystrophic calcification, interstitial fibrosis and alteration in renal size & shape.

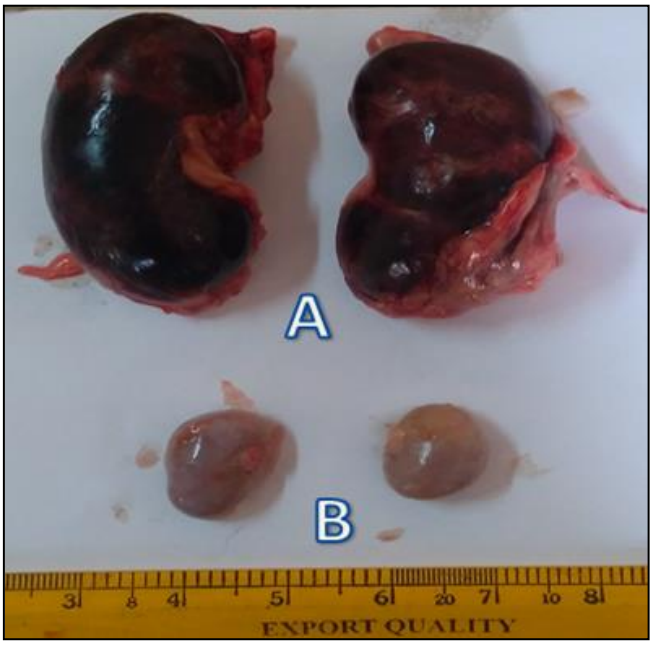

Fig 1: Increased Kidney size in acute renal failure, B. Misshapen, pale to grey color shrunken kidney in CKD.

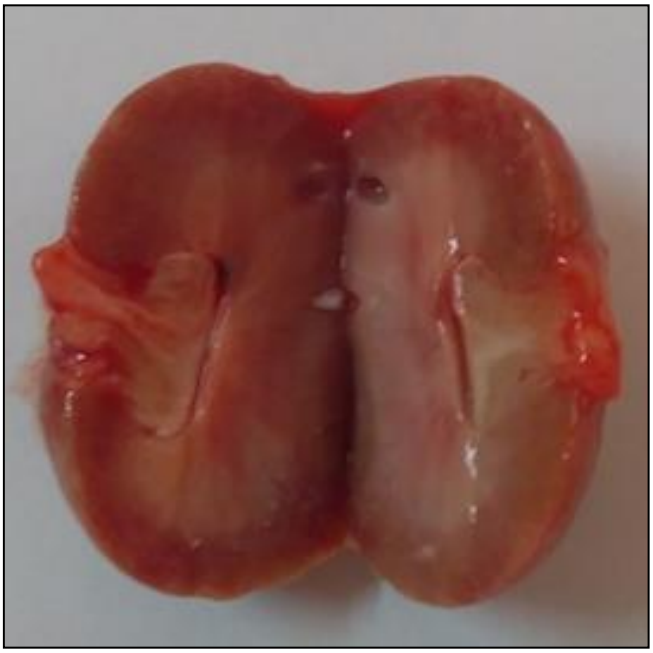

Fig 2: Hemorrhagic patches in cortical area and cortico-medullary junction.

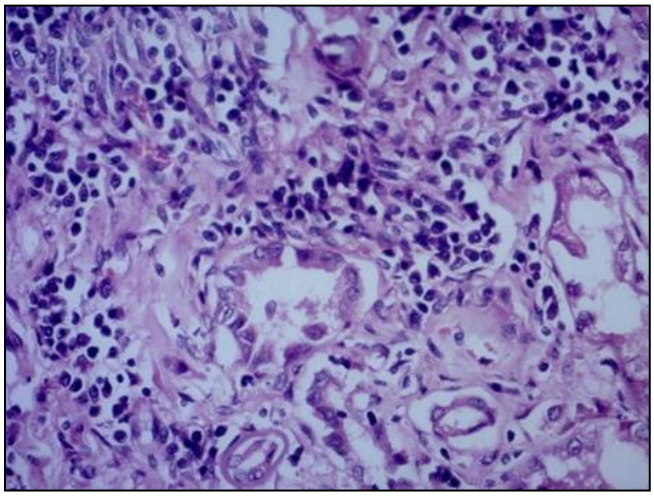

Fig 3: Multifocal lymphoplasmacytic interstitial nephritis in CKD (H&E, 40X)

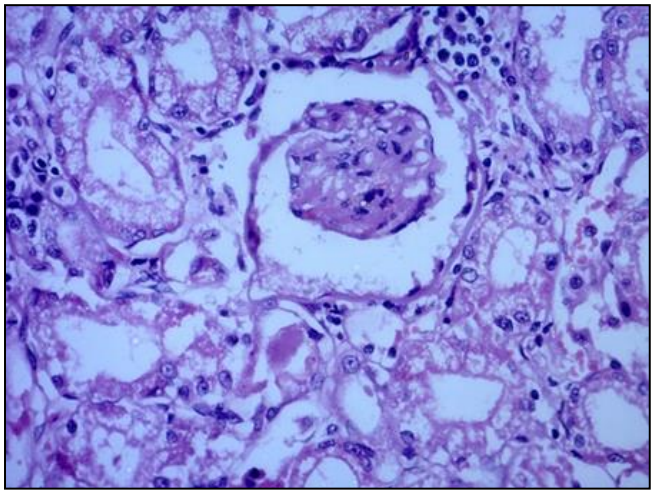

Fig 4: Decreased glomerular cellularity in CKD (H&E, 40X)

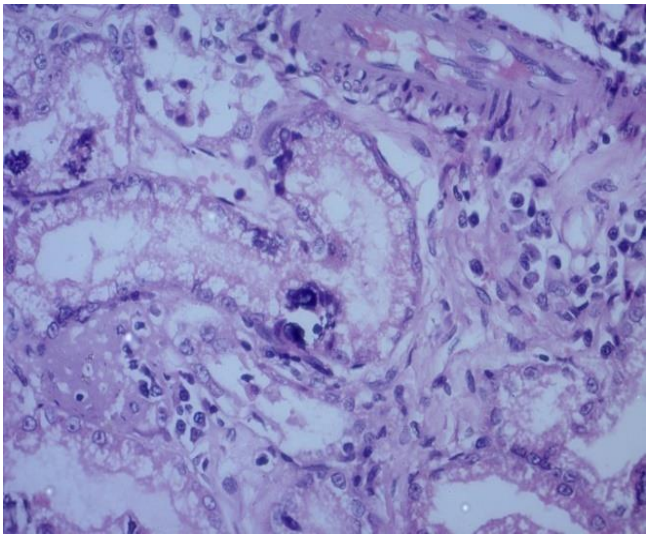

Fig 5: Dystrophic calcification in CKD (H&E, 40X)

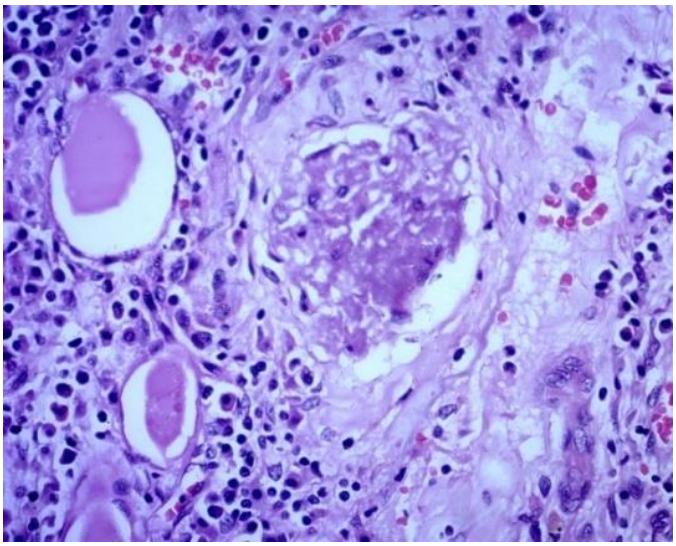

Fig 6: Tubular proteinosis in CKD (H&E, 40X)

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