HISTOPATHOLOGICAL RETROSPECTIVE STUDY OF CANINE RENAL DISEASE IN INDIA DURING 2012-2015

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Histopathological examination of 11 cases of canine renal failure was conducted, collected renal tissues during the post-mortem examinations. The study period is from 2012 to 2015. The histopathological studies revealed cases of Interstitial nephritis-(36.36%), Glomerulonephritis-(36.36%), Tubular nephritis- (18.18%), and Coagulative nephritis- (09.09%). Renal failure was recorded, ranging in the age group from 6 years to 14 years and above.
Keywords: Canine, Renal Diseases, Histopathology, Renal Biopsy.

Kidney disease results from functional or structural abnormalities in one or both kidneys, which are recognized by reduced renal function or the evidence of renal damage. Renal damage can further be defined as microscopic or macroscopic renal pathology.This study evaluated cases of canine renal disease in India within three years and examined the histopathological findings associated with each diagnosis. The distribution of canine renal disease in this study was examined and compared to previous canine studies from other countries**.**

Materials and Methods

The present study was carried out at the Department of Veterinary Clinical Medicine, Ethics and Jurisprudence and Department of Veterinary Pathology; all the cases referred to Teaching Veterinary Clinical Complex (TVCC) during the period from March 2012 to December 2015. Histopathological studies and renal biopsies were carried out in order to assess the structural and functional renal changes in cases wherein the dogs succumb during the course of treatment.

Similarly, the histopathological samples were also collected from 06 dogs that succumbed during the treatment at the time of necropsy examination. The tissues were processed by rapid paraffin embedding technique. The tissue material collected for the histopathological examination was fixed in 10% buffered formalin solution, embedded in paraffin, and cut into slices not exceeding 4 µm in thickness and stained with hematoxylin and eosin(H-E) stain, The stain allowed visualizing disorders in the normal tissue structure under the light microscope showing blue cell nuclei, pink cytoplasm and other structural elements of the renal tissue and helped in the general evaluation of the biopsy collected from the kidney.

Results

In 6 dogs, which died during the course of treatment, the gross appearance was noted. The kidneys of such dogs revealed mild enlargement to the contracted kidney. The capsule of the kidney adhered to the cortex. After the removal of the capsule, the surface of the kidney was observed to be uneven and rough (Fig. 1). Multiple white foci were also observed on some cortices. In a few cases, there was the presence of microcyst on the cortex (Fig. 2), while some revealed haemorrhages (Fig 3a & 3b). These gross appearances were suggestive of chronic interstitial nephritis (Fig. 4). In all cases, the changes observed were bilateral and with more or less of the same intensity. On sections, the kidneys appeared hard, and the medulla of these kidneys was mildly congested, while the cortex was pale.

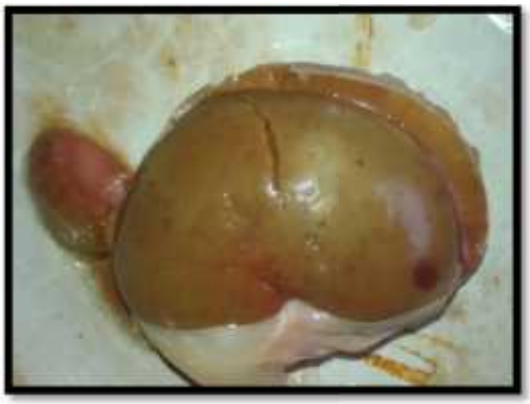

Fig. 1. Mild Enlarged kidney with rough uneven corticular surface

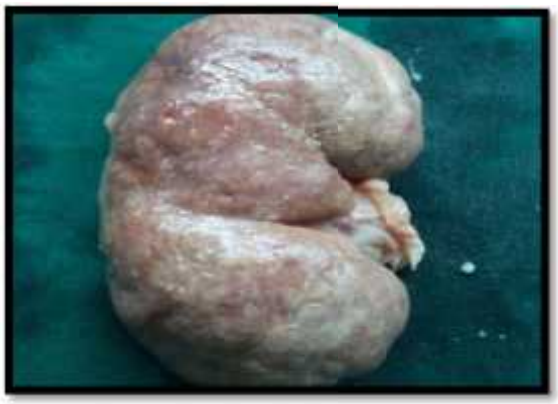

Fig. 2. Contracted kidney with microcyst on the corticular surface

Fig. 3: Diffuse contracted pale kidney with multifocal haemorrhages

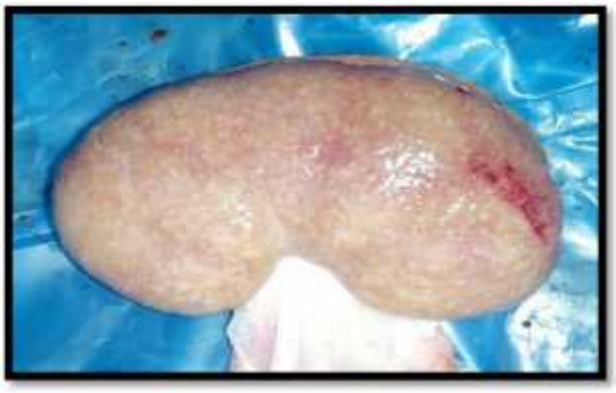

Fig. 4: Diffuse chronic interstitial nephritis. Diffuse interstitial fibrosis is responsible for the fine pitting of the capsular cortical surface, which is stippled red, the result of bands of fibrous tissue surrounding islands of the renal cortex.

Histological sections of kidney tissues revealed a varying degree of tissue alterations (Fig 5 to 16). The cortex of the kidney revealed primarily and extensive necrosis and sloughing of tubular epithelial cells from proximal convoluted tubules. In some cases, the presence of hyaline casts was also evident. The tubule space was reduced. Surrounding such tubules, there was evidence of the proliferation of fibrous connective tissue. In a few cases, a large number of inflammatory cells and the majority of lymphocytes were seen to be accumulated in

between tubules. Only in one case, there was evidence of haemorrhage in the

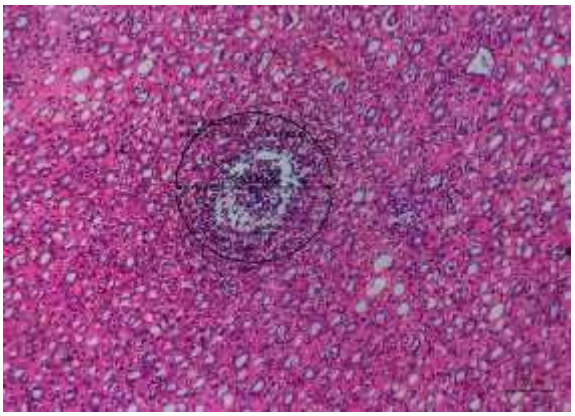

Fig. 5: Section of a kidney showing medullary abscess (H&E)

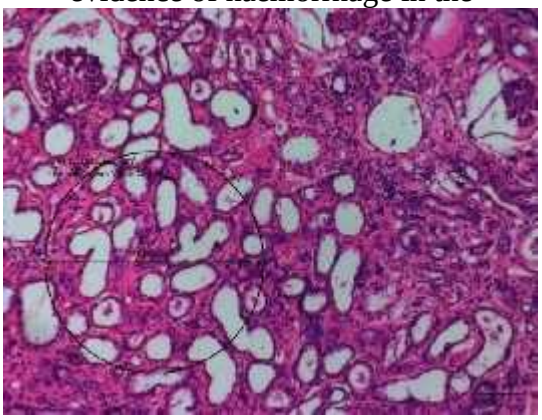

Fig. 6: Section of a kidney showing cyst in the cortex area (H&E)

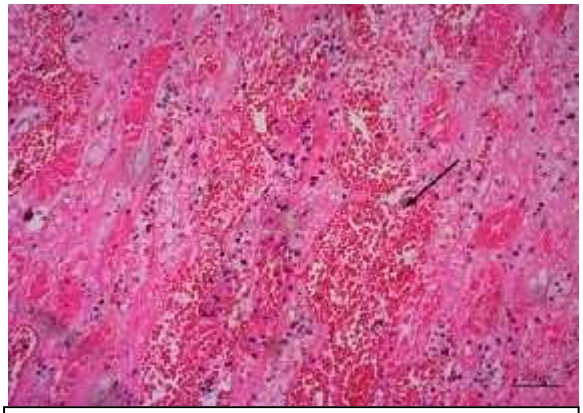

Fig. 7: Section of a kidney showing severe haemorrhages in tubules (arrow) (H&E)

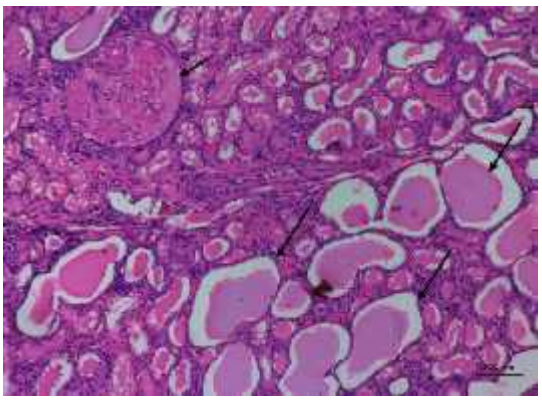

Fig. 8: Section of the kidney revealed retention cysts filled with protein-rich fluid (Long arrows) accumulation of fluid in glomeruli (Small arrow) (H&E)

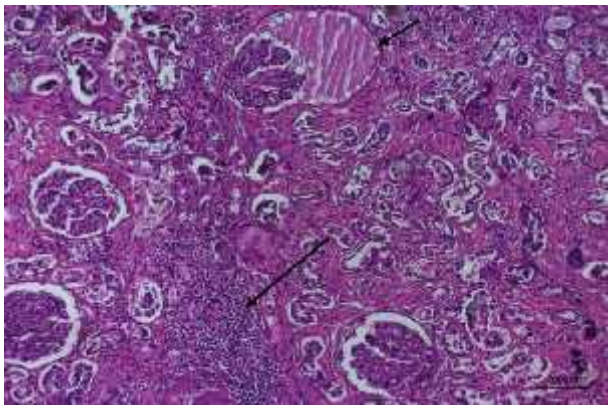

Fig. 9: Section of a kidney showing accumulation of fluid in Bowman's capsule (small arrow) and inflammatory cells in the cortex (big arrow) (H&E)

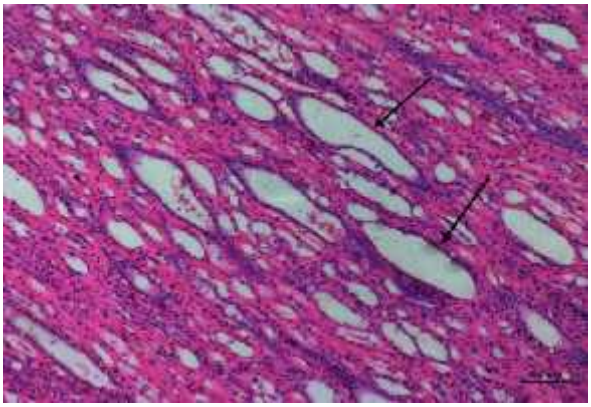

Fig. 10: Section of a kidney showing medullary cysts (H&E)

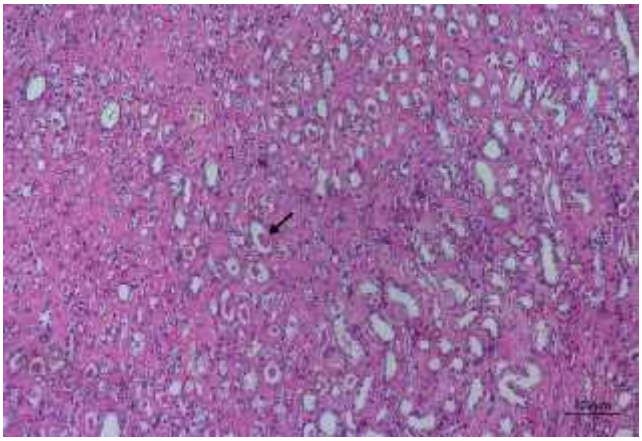

Fig. 11: Section of a kidney showing the hyaline cast in medullary tubules (H&E)

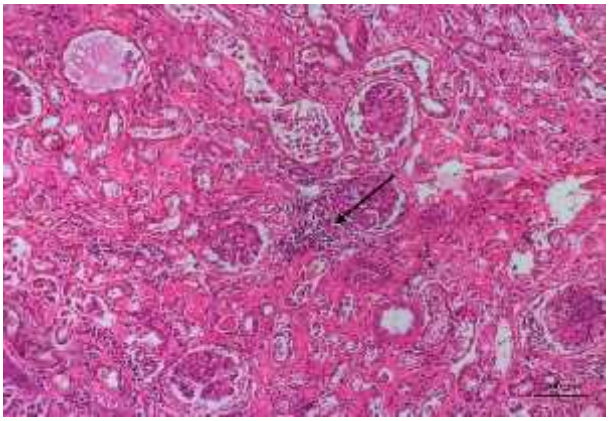

Fig. 12: Section of a kidney showing huge infiltration of inflammatory cells (arrow) in the interstitial space, fibrous connective tissue proliferation, and infiltration in the

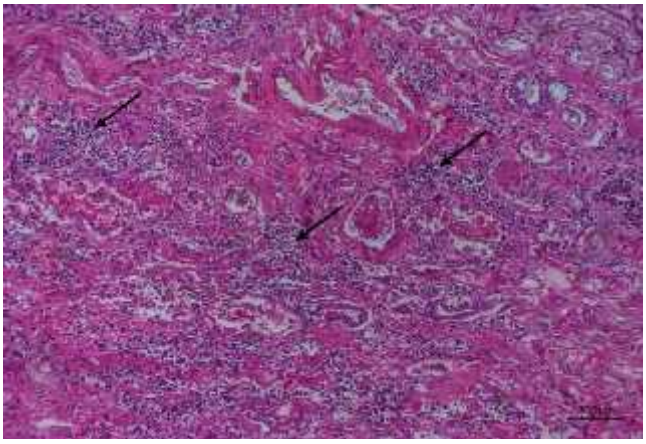

Fig. 13: Section of a kidney showing huge infiltration of inflammatory cells (arrow) in the interstitial space, (H&E)

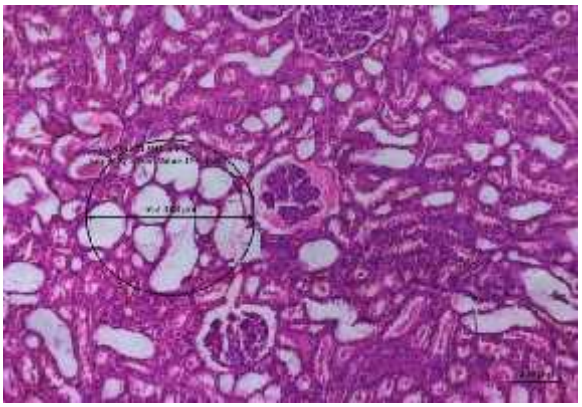

Fig. 14: Section of a kidney showing sloughing of tubular epithelial cells. Formation of multiple microcyst in the renal parenchyma and congested glomeruli

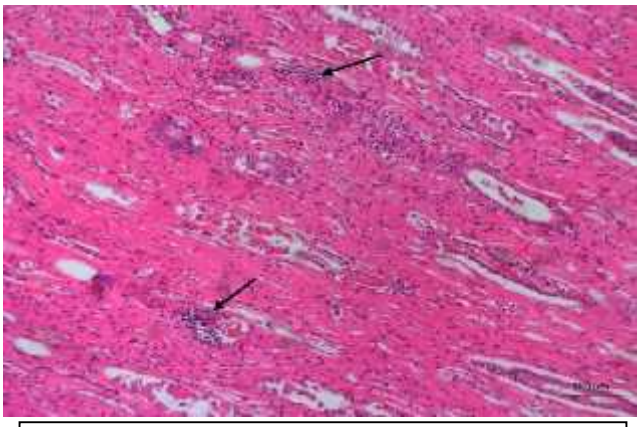

Fig. 15: Section of a kidney showing huge infiltration of inflammatory cells (arrow) in the interstitial space of medulla (H&E)

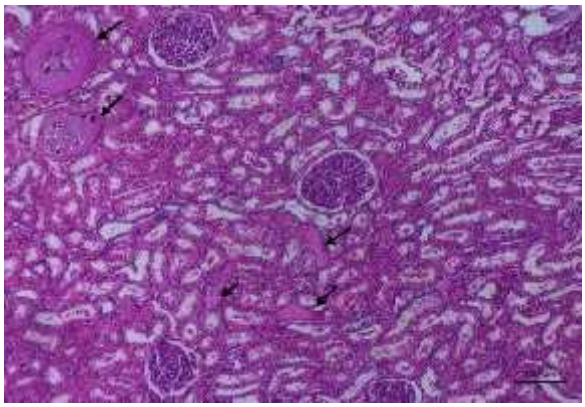

Fig. 16: Section of a kidney showing blocking of tubules with an accumulation of protein and minerals (arrows) (H&E)

cortex. In most cases, there was evidence of cystic conversion of tubules. The cysts formed were of varied sizes. In one case, there was an accumulation of fluid in cysts indicative of retention/polycystic kidney. The glomeruli of these cases appeared to be smaller in size. In the cases in which retention cysts were observed, fluid was seen accumulated in Bowman's capsule of the glomeruli. Bowman's capsule was without glomeruli tuft, forming a larger cyst in the cortex. The medullary areas of the kidney were not affected much compared to the cortex. In the medulla, major alterations were dilated tubules with the formation of a micro cyst. There was an increase in fibrous connective tissue and inflammatory cells around the tubules in the medulla. In one case, microabscesses were found in the medulla.

The specific types of nephritis noted in the present investigation are presented hereunder-Interstitial nephritis- Most of the glomeruli had lost architecture, and condensation of glomeruli was seen. There was increased space between the glomeruli and the capsule. Moreover, 70 % of the glomeruli in the cortex showed degenerative changes. Infiltration of mononuclear cells and proliferation of the connective tissue was observed in the interstitial spaces. The periarterial area showed degenerative changes and proliferative smooth muscle fibers. The changes were suggestive of Interstitial nephritis (Fig. 17).

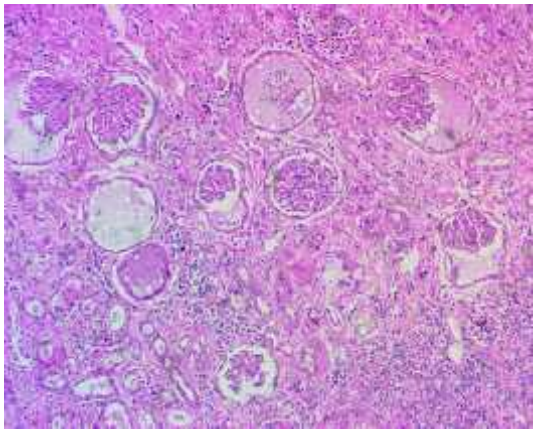

Fig. 17: Section of a kidney showing interstitial nephritis (H&E, 10X)

Glomerulonephritis- The glomeruli tuft was loose and evacuated. The connective tissue around the glomeruli showed cloudy swelling and degeneration. Nuclei of connective tissue were lost. In many tubules, the lumen was occupied by albuminoid material. Degenerative changes in the tubule were more prominent at the corticomedullary junction. The changes were suggestive of glomerular nephritis(Fig. 18).

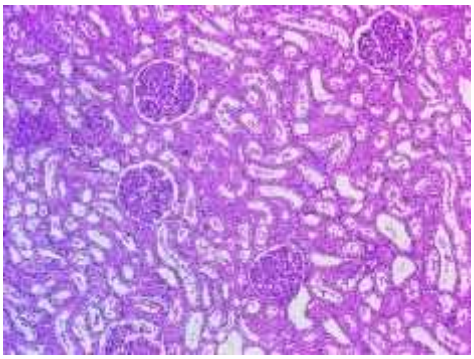

Fig. 18: Section of a kidney showing Glomerulonephritis (H&E, 10X)

Tubular nephritis - Thickening of the glomerular capsule was evident with some of the capsules showing proliferation of connective tissue around. Infiltration of mononuclear cells was noted in between the tubules. There was a loss of tubular epithelium, and desquamated cells were seen in the lumen of the tubules. Glomeruli showed degenerative changes. Similarly, the tubules were dilated. Few tubules were seen with albuminoid material, and tubular degeneration was evident. The tubular epithelium also showed degenerative changes, and the tubule became low cuboidal to flat, giving the appearance of a dilated lumen. The cell boundaries were lost in some tubules. The histopathological changes suggested Tubular nephritis (Fig. 19 & 20).

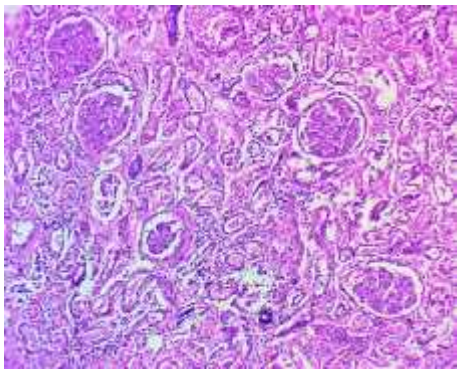

Fig. 19: Section of a kidney showing Tubular nephritis (H&E,

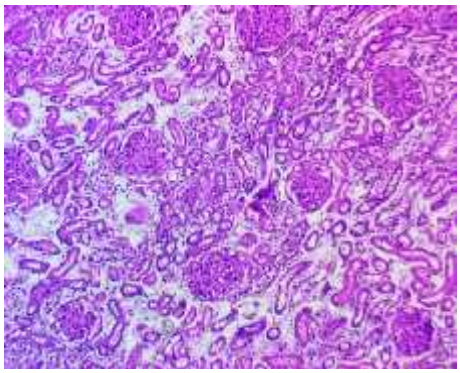

Fig. 20: Section of a kidney showing Tubular nephritis (H&E,

Coagulative nephritis- All tubules showed the presence of coagulated pink mass and haemorrhages in interstitial space. There were severe infiltrations of mononuclear cells. The glomeruli showed degenerative changes. Connective tissue proliferation was observed in the kidney parenchyma. The changes suggested Coagulative nephritis (Fig. 21).

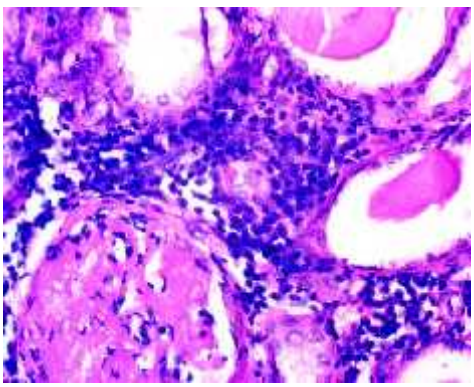

Fig. 21: Section of a kidney showing Coagulative nephritis (H&E,

Discussion

The histopathological alterations in acute and chronic renal failure have been extensively studied and reported. Kavitha *et al.* (2013b) also reported similar gross observations. In their study, moderate to severe adhesion of the capsule to the cortical surface, pitting, and granularity of the cortex were noticed in 83.33 percent of cases. Irregular cortical surface, with few pale foci on the cortex, was also recorded in dogs suffering from chronic renal failure.

The necropsy findings and histopathological studies revealed the presence of glomerular cysts, atrophy of

glomerular tufts, and sclerosis of the interstitial tissue. It isin agreement to the study of Macdougall (1986), who during a retrospective analysis reported a study of kidney tissue for light and electron microscopy and immunohistochemistry in one hundred and eleven dogs with canine chronic renal disease. Fifty-two percent had glomerular nephritis (GN), and forty-eight percent were non-glomerular (NGN) disease, which included glomerular type as focal, diffuse mesangial proliferative, diffuse endocapillary proliferative, mesangiocapillary, diffuse crescentic, diffuse sclerosing, amyloid, unclassifiable.

The 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 the interstitium, calcification of tubules, loss of tubular epithelial cells, atrophy of glomeruli, and thickening of the basement membrane. Grauer (2005) described histopathologic changes in chronic renal disease as a combination of a loss of tubules with replacement fibrosis, mineralization, glomerulosclerosis, glomerular atrophy, and foci of mononuclear cells within the interstitium. Sawale *et al.* (2012) also reported interstitial nephritis and cystitis in a dog. Kavitha *et al.* (2013a) also recorded 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 the interstitium, calcification of tubules, loss oftubular epithelial cells, atrophy of glomeruli, and thickening of the basement membrane.

Conclusions

Histopathological examination in dogs that died due to renal affections is equally informative since it gives an idea of etiopathology to be understood in a better way which in turn helps in research leading to a better understanding of the diseased condition. The gross and histopathological findings in this study correlated well and present classical cases of chronic and acute glomerulonephritis in dogs.

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