NIGERIAN VETERINARY JOURNAL
ISSN 0331-3026
Nig. Vet. J., December 2016 Vol 37 (4): 206-216.
ORIGINAL ARTICLE

Retrospective Studies on the Prevalence, Morphological Pathology and Aetiology of Renal Failure of Dog in Lagos and Abeokuta, Nigeria

Mshelbwala, F. M.[1]; Ajayi, O. L.[1]; Adebiyi, A. A.[1]; Olaniyi, M. O.[1]; Omotainse, S. O.[1]; Omotainse, O. S.[1]; Oluwabi, M. T.[1]; Ojebiyi, O. E.[1]; Akpavie, S. O[2] and Kadiri, A. K. F.[1]

[1]College of Veterinary Medicine, Federal University of Agriculture, Abeokuta, [2]Faculty of Veterinary Medicine, University of Ibadan. *Corresponding author: Email: mshelbwalafakilahyel@yahoo.com; Tel No:+2348109064065

SUMMARY

The prevalence and morphological pathology of renal failure in exotic breeds of dog in Lagos and Ogun States, within Southwestern Nigeria were determined from postmortem records of the Department of Veterinary Pathology, College of Veterinary Medicine, Federal University of Agriculture, Abeokuta, between 2012 and 2015. The prevalences of renal failure were determined by calculating the percentage of occurrence among dog carcasses. Formalinized tissue samples of the kidney collected at postmortem during the period were processed and stained with Haematoxylin and eosin for histopathological studies. Leptospira species were demonstrated in the renal tubules using Wartin starry stain. The overall prevalence of glomerulonephritis was 22.81%. Annual prevalence varied from 15% in 2013 to 33.3% in 2014. Age-specific prevalence was highest (10.53%) among older dogs between the ages of 6 years and above. There was no significant difference (P> 0.05) between the prevalence in male and female dogs. Breed-specific prevalence varied from zero prevalence among the local breed to 50% prevalence among Rottweiler cross. Grossly, extra-renal lesions include ulcerative dermatitis and the mucous membrane of the buccal cavity was jaundiced and had ulcers in 57% of the cases. In acute cases, the kidneys were swollen and pale in 60% of the cases. Whereas in chronic cases, the kidneys were pale, shrunkened, and the cortexes were rough and pitted with the capsules adherent to the cortexes in 80% of the cases. Tweenty eight percent showed either left or right smaller kidney with calculi of variable sizes in the pelvis. Microscopically, in the acute cases, the kidneys showed moderate necrosis of tubular epithelial cells and infiltration by lymphocytes and macrophages in the interstitium while in the chronic cases; the tubules are atrophied in 27%. There is protein leak into the Bowman´s space in 60%. The renal interstitium showed severe fibroplasia and the lumen of the collecting ducts contained proteinecious cast. The interstitium is severely infiltrated by lymphocytes and plasma cells in 67% cases. The prevalence of acute and chronic glomerular and interstitial nephritis among exotic breeds of dog in Lagos and Ogun States was significantly high and the gross and histopathological lesions correlated well.

Key words: Postmortem, Renal failure, nephritis, Pathology, Dog.

INTRODUCTION

Glomerular disease is a major cause of chronic kidney disease (CKD) in dogs and is occasionally seen in cats (Wilcock and Patterson, 1976; Vanden, 2010; Cynthia, 2005). Acute glomerular and interstitial nephritis can be caused by tubular necrosis from infectious agents such as bacteria, including *Leptospira* species, *Escherichia coli*, *Streptococcus* species, *Staphlococcus* species and *Proteus* species or viruses such as infectious canine hepatitis virus, canine distemper virus and canine herpes virus. The disease can also be caused by obstruction nephropathy from urolithiasis, transitional cell neoplasms of the lower urinary system, trauma, renal ischemia, nephrotoxic drugs such as aminoglycoside and chemicals such ethylene glycol and heavy metals (Cynthia, 2005; Donald, 2010). Whereas chronic glomerular and interstitial nephritis are long standing cases of renal disease that result to nephron loss which are not specific to the original cause of the disease but considered common end-stage responses to many injurious stimuli (Barber, 2000; Vadan and Brown, 2009; Donald, 2010; Shelly and Vaden, 2011)). There are both immunologic and nonimmunologic mechanisms of glomerular injury that can lead to various forms of glomerular disease (Courser and Salant, 1982). Glomerulonephritis is generally considered to be the most commonly occurring glomerular disease in dogs and is the result of immunologic glomerular injury (Courser and Salant, 1982; David *et al*., 1986). Cell mediated immune mechanisms also take part in the pathogenesis of glomerular inflammation. Once the glomerular damage is initiated, the activation of complement, the coagulation cascade and resident cells, influx of neutrophils, monocytes and platelets, release of proteolytic enzymes, synthesis of cytokines, or other growth factors, generation of proinflammatory lipid mediators and alteration of haemodynamic factors contribute to glomerular injury (Courser and Salant, 1982; Cynthia, 2005). Immune-mediated glomerulonephropathy is characterized by deposition or in situ formation of immune complexes in the glomerular capillary wall which then incite inflammatory changes (Courser and Salant, 1982; Walliam, 1985). Animals with primary glomerular disease as a cause of CKD may have somewhat different clinical and laboratory Abnormalities than those with primary tubulointerstitial disease (Horney and Stojanovic, 2013). Glomerulonephritis in dogs results in an excessive loss of protein from the system and can be detected in the urine. Damage to the glomerulotubular basement membrane can result in albuminuria which may lead to hypoalbuminemia, ascites, dyspnea due to pleural effusion or pulmonary oedema and or peripheral oedema which may be reffered to as the nephritic syndrome (Jacob *et al*., 2005; Syme *et al*., 2006). Immune-mediated glomerulonephritis has been associated with chronic diseases such as neoplasia, rickettsial diseases, systemic lupus erythematosus (SLE), heartworm diseases, pyometra, chronic septicaemia and adenovirus infection; in which antigen-antibody complexes persist in circulation, but it is usually idiopathic (Grauer, 2009; Vaden and Brown, 2009; Vaden, 2010). Diagnosis of chronic glomerulonephritis can be done based on clinical signs of uremia and on laboratory evaluation of blood urea nitrogen, creatinine, and phosphorus concentration which are usually increased; urinalysis to determine proteinuria and renal biopsy for histological examination and gross and histopathological findings (Jacob *et al*., 2005). This study investigate the aetiology and prevalence of acute and chronic renal failure in local and exotic breeds of dogs from Lagos and Ogun states, within Southwestern Nigeria, based on gross

and histopathological changes associated with the conditions, between 2012-2015.

MATERIALS AND METHODS

Location and Data Source

This study was carried out on carcasses of dogs from Lagos and Abeokuta the capitals of Lagos and Ogun states, Nigeria, and environs. Abeokuta has the geographical location of 7^{o} 09^{1} N 3^{o} 21^{1}E (Ogunsesan *et al*., 2011).The carcasses were submitted for postmortem examination, to the Department of Veterinary Pathology, College of Veterinary Medicine, Federal University of Agriculture.

Information on the number of postmortem carried out on carcasses of dogs, the ages of dog affected, sexes, breeds and the clinical observation from 2012-2015 were retrieved from the postmortem records. The prevalence of glomerular and interstitial nephritis was determined by calculating the frequency of occurrence of kidney lesions among the carcasses of dogs submitted for postmortem during the study period. Annual variation was determined by calculating annual indices at 95% confidence interval.

Sample Collection and Cultural Isolation

Samples of kidneys presented with pathological lesions were collected at postmortem for microbiology and histopathology, during the study period. Isolation of leptospira species were carried out according to the method described by Faine (1965) using Ellinghausen-McCullough-Johnson-Harris (EMJH) broth medium. Information on other agents associated with the kidney lesions was also obtained from records of Microbiology Laboratory.

Histopathology

Formalinized tissue samples of the visceral organs obtained at postmortem during the period under study were retrieved from an archive, and were processed and cut at 5µm thick and stained with haematoxylin and eosin stains for histomorphological study. The stained tissue slides were examined with the light microscope and the lesions were described.

Warthin and Starry Silver Staining of the Kidney Tissues.

Samples positive for leptospira species were processed and cut 5µm thick and impregnated with Silver nitrate according to Warthin and Starry method for demonstration of leptospira organism in the kidney tissues.

RESULTS

The dogs had clinical signs of pale mucous membranes and jaundice in 62% of the cases; there were red based ulcerations in the fore and hind limbs and the buccal cavity in 57.1% of the cases. The dogs were weak (38.5%) and anorexic in 57.7% of the cases; emaciation in 46.2% of the cases and there was fever in 50.0% of the cases (Table I). The overall prevalence of glomerular and interstitial nephritis in exotic breeds of dog in Lagos and Ogun States was 22.81%, whereas the annual incidence ranged from 15.4% in 2013 to 33.3% in 2014 (Table II). Age-specific prevalence was highest (10.53%) among older dogs between the ages of 6 years and above and was zero among younger dogs between the ages of less than 1 to 2 years (Table III). Sex-specific prevalence was similar between

TABLE I: Clinical observations associated with renal failure among exotic breeds of dog

S/N	Clinical Signs	Number of renal failure	Number of clinical signs	(%)
1.	Ulcerations	26	15	57.7
2.	Emaciations	26	12	46.2
3.	Weakness	26	10	38.5
4.	Fever	26	13	50.0
5.	Anorexia	26	15	57.7

TABLE II: Annual prevalence of renal failure among exotic breeds of dog

Year	Number Postmortem	number of renal failure	(%)
2012	32	6	18.75[b]
2013	26	4	15.40[b]
2014	36	12	33.30[a]
2015	20	4	20.00[b]
Overall	114	26	22.81[b]

Values with the same superscript are not significantly different P > 0.05

TABLE III: Age-specific annual prevalence of renal failure among exotic breeds of dog

Year	NO. of Postmortem	Age bracket in years and number (%) of renal failure				
		< 1-2	2-3	4-5	6 and above	Overall
2012	32	0 (0.00)	1 (3.13)	2 (6.25)	3 (9.38)	6 (18.75)
2013	26	0 (0.00)	1 (3.85)	1 (3.85)	2 (7.69)	4 (15.38)
2014	36	0 (0.00)	3 (8.33)	4 (11.11)	5 (13.89)	12 (33.33)
2015	20	0 (0.00)	1 (5.00)	1 (5.00)	2 (10.00)	4 (20.00)
Total	114	0 (0.00)	6 (5.26)	8 (7.02)	12 (10.53)	26 (22.81)

TABLE IV: Sex-specific annual prevalence of kidney failure among exotic breeds of dog

Sex	Number examined	Year and number (%) of kidney failure				
		2012	2013	2014	2015	Overall
Male	65	3 (4.62)	4 (6.15)	6 (9.23)	2 (3.08)	15 (23.08)
Female	49	2 (4.08)	3 (6.12)	4 (8.16)	2 (4.08)	11 (22.45)
Overall	114	5 (4.39)	7 (6.14)	10 (8.77)	4 (3.51)	26 (22.81)

TABLE V: Breed-specific prevalence of renal failure among exotic breeds of dog

Breed	NO. of PM	NO. of Cases with Renal Lesion	Percentage (%)
Alsatian	30	8	26.66
Rottweiler	16	6	37.50
Bull Mastiff	10	2	20.00
German Shepard	6	0	0.00
Culcasian	6	2	33.33
Rott Cross	4	2	50.00
Boerboel	12	4	33.33
Ridge Back	4	0	0.00
French Mastiff	8	2	25.00
Local	10	0	0.00
Total	114	26	22.81

male (23.08%) and female (22.45%) dogs (Table IV). The breed-specific prevalence ranged from 0-50% with the local Mongrel having the zero prevalence while the Crossed breed had the highest prevalence (37.50%) (Table V). Disease-specific prevalence showed that renal failure was highest in Leptospirosis (61.53%) and lowest in Babesiosis (3.84%) (Table VI).

Pathology

Gross Lesions

In the acute cases of glomerular and interstitial nephritis, which were mostly associated with leptospirosis (70%),

colibacillosis (10%), other bacteria (15%), and unidentified cause (5%), the kidneys were inflammed and pale; and there were multiple whitish foci on the renal cortexes in 50% of the cases with haemorrhages in 30%. Whereas in the chronic cases, which were mostly associated with long standing cases of leptospirosis (70%), and in some cases trypanosomosis (10%), babesiosis (10%), and unidentified cause (10%). There was ulcerative dermatitis covering the carpal and metacarpal, tibia and fibula areas and thigh muscles of both hind limbs in 37% of the cases and there were also ulcers (2-3 cm) on the gums of the upper jaws and the mucous membranes of the eye. The mucous membrane of the oral cavity was moderately icteric in 57% (figure 1). The lungs were moderately congested and oedematous in 90% of the cases, with focal ecchymotic haemorrhages. The heart was globosed in 52% of the case. The liver was markedly enlarged and had yellowish discolouration (64%). The spleen was also markedly enlarged (60%), the left or right kidneys were smaller than normal in 28% of the cases, renal capsules were adhered to the cortex which was rough, pitted (figure 2) and the ratio of the cortex to medulla of the affected kidney were smaller than normal in 90% of the cases. There were calculi in the pelvis in 23% of the cases (figure 3). About 10ml of dark viscous fluid was observed in the uterus in 5% of the cases. There were

Table VI: Disease-specific prevalence of renal failure among exotic breeds of dog

Organism	Number of Kidney Lesion	Percentage of Disease	Type of lesion
E. coli infection	26	5 (19.23)	Acute
Leptospirosis	26	6 (61.53)	Acute/chronic
Trypanosomosis	26	2 (7.70)	Chronic
Babesiosis	26	1 (3.84)	Chronic
Other infection	26	2 (7.70)	Chronic

Figure 1: Photograph of ulcerative gingivitis measuring approximately 2 cm in diameters on the gum of the upper jaws (arrow) and icteric mucous membrane of the buccal cavity of an Alsatian dog that died of Leptospirosis

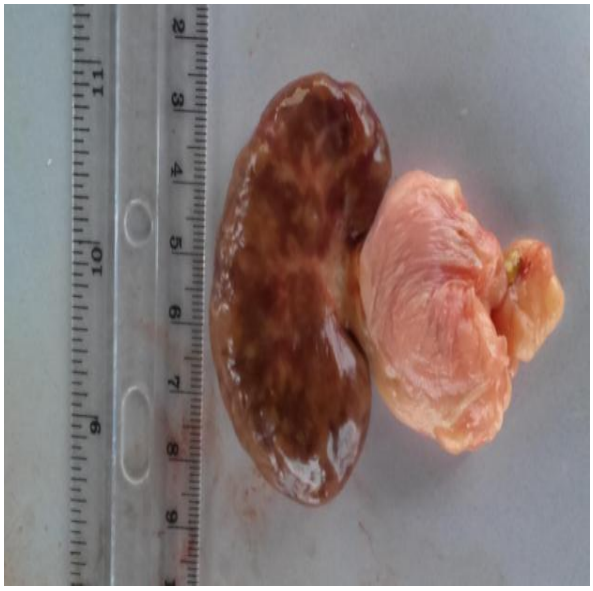

Figure 2: Photograph of the kidney of an adult Alsatian dog that died from chronic leptospirosis showing shrunkened, rough, pale and pitted cortex

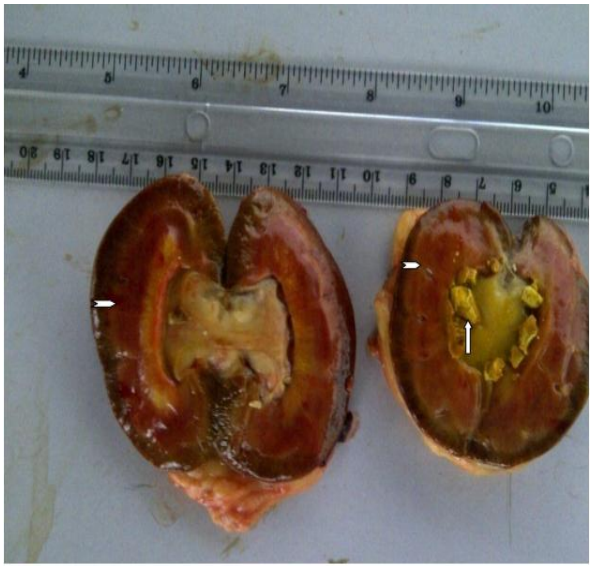

Figure 3: Photograph of an apparently normal sized kidney (right) and smaller left kidney with calculi in the pelvis (arrow), the ratio of the cortex to the medulla was small and the medulla of both kidneys were congested (arrow heads), from an adult Alsatian dog that died from chronic Leptospirosis

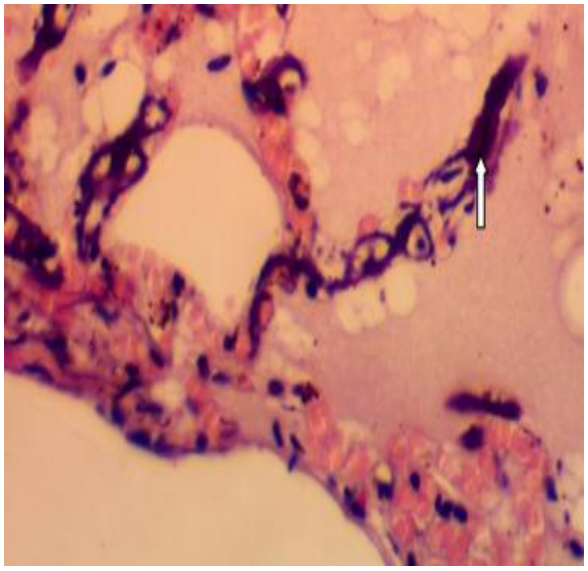

Figure 4: Section of the lung of a dog that died from chronic renal failure showing severe calcification of the alveolar wall (arrow) (x400; H & E)

ulcerative and haemorrhagic gastritis and enteritis in 46% of the cases.

Histopahology

Section of the lung shows congestion and oedema, inflitration by neutrophils, lymphocytes and macrophages in the alveolar wall in 70% of the cases and also showed Perivascular infiltration. There are severe calcifications of the alveolar walls in 50% of the chronic cases (figure 4). Multiple foci of calcification were also observed in different tissues and organs in many of the cases of renal failure examined in this study. Section of the liver showed moderate multifocal necrosis of hepatocytes and infiltration by lymphocytes and plasma cells. There is severe hyperplasia of macrophages and plasma cells in the spleen. Section of the kidney showed severe necrosis of tubular epithelial cells in 95% cases, which were desquamated. There were moderate lympho-plasmacytic infiltrates in the interstitium in acute cases. Whereas in chronic cases, there is necrosis and desquamation of tubular epithelial cells, atrophy of renal tubules, marked lympho-plasmacytic infiltrates, thickened glomerular basement membrane and protein cast in the Bowman´s space in 60% of the cases (figure 5). There is marked interstitial fibrosis and Protein casts in collecting ducts. Section of the intestine shows marked oedema, haemorrhages, necrosis of mucosal epithelial cells and infiltration by lymphocytes, neutrophils and macrophages.

Warthi and Starry Staining

Warthin and Starry silver staining demonstrated Leptospira organisms in many of the renal tubules of the dogs. They appeared either as a whole spiral organism or granules within the tubular lumina (figure 6).

Microbiology results

Bacterial isolates associated with the kidney lesions were *Leptospira species* (61.53%)

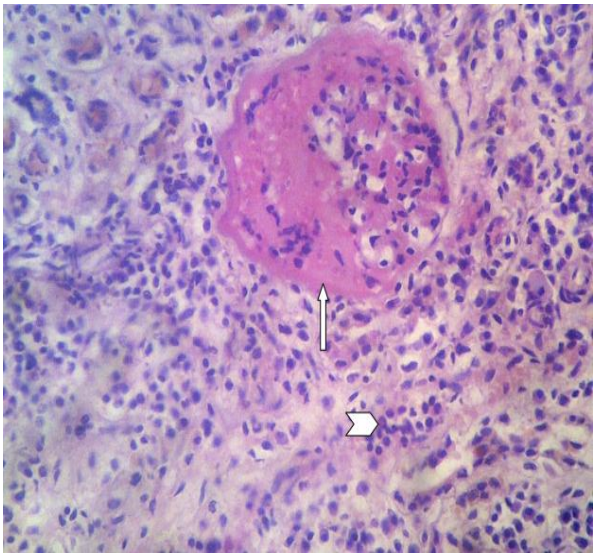

Figure 5: Section of the kidney showing profuse protein leak in bowman´s space (arrow), loss of nephrons, fibrosis and infiltration by lymphocytes, macrophages and plasma cells (arrow head) associated with chronic glomerulonephritis in an adult Alsatian dogs that died from chronic leptospirosis (x400; H&E)

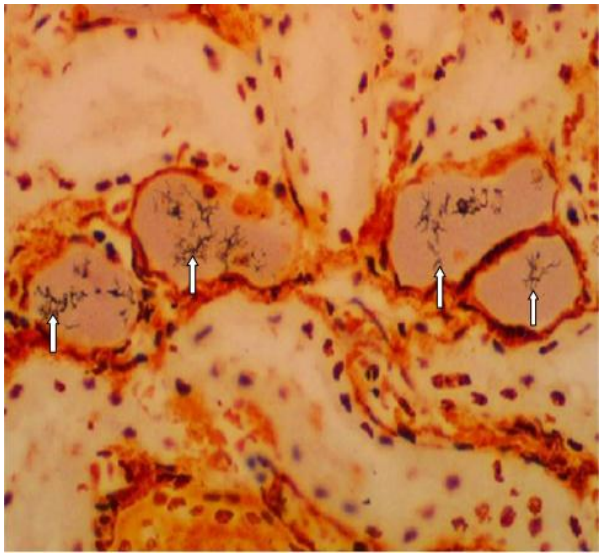

Figure 6: Section of the kidney of a dog that died of leptospirosis showing numerous leptospira organisms in the renal tubules (arrows) (x400; Warthin and Starry stains)

and *Escherichia coli* (19.23%). Others were not identified (7.7%). Examination of thin blood smear revealed Trypanosoma species in 7.70% whereas Babesia species occurred in 3.84% cases.

DISCUSSION

The clinical signs of weakness, anorexia and emaciations observed in the dogs prior to death might suggest the terminal nature of the disease at the time they were presented at the Veterinary Teaching Hospital. These clinical signs are known to be associated with terminal cases of kidney failure (Jacobs, 2005; Cynthia, 2005). The clinical signs of renal failure observed in this study agreed with the report of O´Neill *et al*. (2013) who observed halitosis, weight loss, polyuria, polydipsia, urinary incontinence, vomiting, decreased appetite, lethargy and diarrhea in dogs with chronic kidney disease in the United Kingdom. The cases of glomerular and interstitial nephritis in this study involved both kidneys with uremic lesions. The extra-renal lesions of skin ulcers and the ulceration in the mucosal membranes of the buccal cavity are consistent lesions in renal failure. The ulcers resulted from uremia arising from the inability of the failing kidney to excrete urea and other waste products of metabolism (Jacobs, 2000; Cynthia, 2005)

The overall prevalence (22.81%) of renal failure in dogs in this study was high compared to the work of Cynthia (2005) who reported a prevalence of 10% among dogs. The prevalence of chronic kidney disease in dogs varied from 0.05% to 3.74% in the United Kingdom (O´Neill *et al*., 2013). The prevalence of glomerular disease was reported to be 52% while that of non glomerular kidney disease was 48% in dogs associated with kidney diseases in East Anglia and West Midlands in the United States (MacDougall *et a*l., 1986). There is paucity of reports on the prevalence of kidney diseases in dogs in Nigeria.

The high prevalence of kidney failure in older dogs between the ages of 6 years and above suggest that old age can be a predisposing factor to renal failure. Kidney diseases are known to be associated with old age between the ages of 5-6 years (Dugold *et al*., 1986; Finco *et al*., 1994; Cynthia, 2005). O´Neill *et al*. (2013) reported that advancing age, small body size, and periodontal disease are risk factors that predispose to kidney diseases. However, chronic renal failure has also been reported in both young and old dogs (Smarts *et al*., 2016).

The prevalence of renal failure in this study was similar between male and female sexes. This might suggest the involment of unknown environmental factors which predisposed the dogs to renal diseases rather than hormonal factors. This agreed with the reports of previous workers who found no significant difference in the prevalence of renal failure between male and female dogs (Cynthia, 2005; Beidun *et al*., 2007).

The high prevalence of the condition in Crossed breed and Rottweiler in this study might suggest a genetic predisposition of the breeds to chronic renal failure. This is in agreement with the study of Cook (1993) who observed atrophic glomerulopathy in chronic renal failure in four Rottweilers, each less than 1 year, in which all the 4 dogs had severe azotemia and massive protein losing nephropathy. Breeds of dogs such as Cocker spaniel, Cavalier king and Charles spaniel have been reported to be prone to kidney diseases (O´Neill *et al*., 2013).

Causes of chronic glomerular and interstitial nephritis include congenital malformation of the kidneys (birth defect), chronic bacterial infection of the kidney (Pyelonephritis) with or without kidney stone and diseases associated with the immune system in which antigen-antibody complexes persist in circulation (Cynthia, 2005; Grauer, 2009; Vaden and Brown, 2009). These are in agreement with the causes reported in this study. It is possible that cases of acute renal diseases in this study progressed to chronic glomerulonephritis as reported by previous workers (Grauer, 2009; Vaden and Brown, 2009; Vaden, 2010).

The causes of the kidney disease that lead to acute and chronic renal failure in these cases were mostly of bacterial origin. *Leptospira species* and *E. coli* were isolated from the conditions in this study. The gross and histopathological lesions, especially the lympho-plasmacytic infiltration in the interstitium are consistent with renal leptospirosis (Baber, 2000). *Leptospira species* and *E. coli* are known to cause acute and chronic nephritis with associated haemolysis of red blood cells and subsequent jaundice (Baber, 2000; Donald, 2010). However, parasitic infections have been associated with some of the chronic form of the glomerular and interstitial nephritis in this study. Trypanosome species are known to cause anaemia which can result to hypoxia in different organs and subsequent renal damage (Radostits *et al*., 1991; Urquhart *et al*., 2002; Thirnavukkarasu *et al*., 2004; Mshelbwala *et al*., 2015). Kidney diseases associated with trypanosomosis in which there was severe glomerulonephritis have been reported in dogs (Van Velthuysen and Florquin, 2000). Parasitic diseases are known to cause chronic infections leading to antigen-antibody complexes formation, which are deposited in the glomerular basement membrane and the basement membrane of the capillary tuft thereby inciting severe inflammatory response and subsequent fibroplasia (van Velthuysen and Florquin, 2000). Similarly, glomerular and interstitial nephritis have been associated with babesiosis in some of the cases in this study. Glomerulonephritis in which there were protein and renal casts and epithelial cells in urine sediment were commonly observed in both complicated and uncomplicated babesiosis (Lobetti and Jacobson, 2001). Babesia species are also known to cause

anaemia which can lead to tissue hypoxia and subsequent renal damage (Irwin, 2010). Systemic inflammatory responses to canine babesiosis have also been reported to cause renal damage (Schetters, *et al*., 2009). Most of the observations in this study were evaluated at postmortem examination and information on the haematological and biochemical parameters were not available. There is dearth of information on the prevalence of glomerular and interstitial nephritis that lead to renal failure in dogs in this study area. However, this present study revealed high prevalence of the condition and might serve as baseline information for future study. Leptospirosis and other bacterial organisms are the leading causes of renal failure in dogs in the study area. The gross and histopathological findings in this study correlated well and presents a classical cases of chronic and acute glomerulonephritis in dogs.

REFERENCES

BABER, P. (2000). Diagnosis and management of chronic renal failure. *In Practice*, 25: 306-331

BEIDUN, H., KRISTIANAAN, TAIGA, J.J. and HOGAL, J. (2007). Renal histomorphology in dogs with pyometra and control dogs, and long term clinical outcome with respect to signs of kidney diseae. *Acta Veterinarian Scandinavica*, 49: 15.

COOK, S.M. (1993). Renal failure attributable to atrophic glomerulopathy in four related Rottweiler. *Journal of American Veterinary Medicine Association*, 1:202 (1): 107-109.

COURSER, W.G. and SALANT, D.J. (1982). Immunopathogenesis of Glomerular Capillary Wall Injury in Nephritic States, in Contemporary Issues in Nephrology. Nephrotic Syndrome, Myers, B.D., Brenner, B.M., Stein, J.H. (eds), Churchill Livingstone, New York, Vol 9: Pp. 47.

CYNTHIA, M.K. (2005). Chronic Kidney Disease In: The Merck Veterinary Manual. Cynthia, M.K. (ed), 9th Edition, Merck and Co., Ink White House Station, N.J., USA, Pp. 1267-1272.

DAVID, J.S., STEPHEN, A., CHRISTINE, D., NEVA, J.C., GERALD, C.G., IRWIN, D.F., HELMUTT, G.R. and JOHN, E.D. (1985). Influence of antigen distribution on the mediation of immunological glomerular injury. *Kidney International*, 27: 938-950.

DONALD, M.M. (2010). Urinary system: In Pathologic Basis of Veterinary Diseases. Donald, M.M. (ed) 5th Edition, Elsevier publishing Company, Illinois, pp 510-610.

DUGOLD, F.M., Terrence, C., Adrian, P.S. Cattell, V. (1986). Canine chronic renal diseases: prevalence and types of glomerulonephritis in dogs. *Kidney International*, 29 (6): 1144-1151.

FAINE, S. (1965). Silver staining of Spirochaete in single tissue section. Journal of Clinical Pathology, 18: 381-382.

FINCO, D.R., BROWN, S.A., CROWELL, W.A. (1994). Effect of aging and dietary protein intake on uninephrectomized geriatric dogs dogs. *American Journal of Veterinary Research*, 55: 1282-1290.

GRAUER, G.F. (2009). Glomerulopathies In: Nelson, R.W., Couto, C.G. (eds). Small Animal Internal Medicine, 4th edition, St. Louis (MO): Mosby (Elsevier) Pp. 637-644.

HORNEY, B.S. and STOJANOVIC, V. (2013). Protein-losing nephropathy associated with Borelia burgdorferi. Seropositivity in a soft coated Wheaten ferrier: Response to

therapy. *Canadian Veterinary Journal*, 54 (4): 392-396.

IRWIN, P.J. (2010). Canine babesiosis. *Veterinary Clinics of North American Small Animal Practice*, 40 (6): 1141-1156.

JACOB, F., POLZIN, D.J., OSBOME, C.A. (2005). Evaluation of the association between initial proteinuria and morbidity rate or death in dogs with naturally occurring chronic renal failure. *Journal America Veterinary Association*, 226 (3): 393-400.

LOBETTI, R.G. and JACOBSON, L.S. (2001). Renal involvement in dogs with babesiosis. *Journal of South Africa Veterinary Association*, 72 (1): 23-28.

MACDOUGALL, D.F., COOK, T., STEWARD, A.P., CATTEL, V. (1986). Canine chronic renal disease: prevalence and types of glomerulonephritis in dog. *Kidney International*, 29 (6): 1144-1151.

MSHELBWALA, F.M., OMOTAINSE, S.O., AJAYI, L.O., OLANIYI, M.O., ADEBIYI, A.A., TAKEET, M.I., ADEBAYO, O.O. and KASALI, O.B. (2015). Canine trypanosomosis: Clinical observation and morphological pathology associated with natural infection of Trypanosoma congolense in exotic breeds of dog in Abeokuta, Nigeria. *Nigerian Journal of Parasitology*, 36 (2): 85-90.

OGUNSESAN, D., OYEDEPO, J., OATES, J., ADEOFUN, C.O., IKEMEH, R. and BERGL, R. (2011). GIS-Supported Survey of Low-land Rain Forest in South-western Nigeria. Proceedings of the Environmental Management Conference, Federal University of Agriculture, Abeokuta, Nigeria. Pp. 1-14.

O´NEILL, D.G., ELLIOTT, J., church, D.B., MCHREEN, P.D., THOMSON, P.C. and BRODBELT, D.C. (2013). Chronic kidney disease in dogs in United Kingdom. Veterinary practices, prevalence, risk factors and survival. *Journal of Veterinary Internal Medicine*, 27 (4): 814-821.

RADOSTITS, O.M., BLOOD, D.C. and GRAY, C.C. (991). Trypanosomosis in Animals. Veterinary Medicine, 8th Edition. Bailliere Tindal, London, Pp. 1209-1220.

ROSETTI, C., BIBIANA, N.V., NOEMI, P., JULIO, C.C. (2004). Comparism of three diagnostic methods for the detection of Leptospires in the kidneys of wild house mice (Mus musculus). Pesq. Vet. Bras., 24 (1): 6-10.

SCHETTERS, J.P., KLEUSKENS, J.A., Van De CROMMERT, J., De LEEURO, P.W.G., FINIZIO, A.L. and GOVENFLOT, A. (2009). Systemic inflammatory responses in dogs experimentally infected with Babesia canis; a haematological study. *Veterinary Parasitology*, 162 (1): 7-15.

SMARTS, P.M.Y., MAYER, G., MADDARA, G.G.J. and DUCHA, L. (2016). Urinary marker in healthy young and aged dogs and dogs with chronic kidney disease. *Journal of Veterinary Internal Medicine*, 24 (1): 65-72.

SYME, H.M., MARKWELL, P.J., PTEIFFER, D. (2006). Survival of cats with naturally occurring chronic renal failure is related to severity of proteinuria. *Journal of Veterinary Internal Medicine*, 29 (3): 528-535.

SHELLY, L. V. (2011). Glomerular Disease. Topics in Companion Animals, 26 (3): 128-134.

THIRUNAVUKKARASA, P.S., RAO, V.V., SRINIVASAN, S.R., NAMBI, A.P., DHUNAPALAN, P. (2004). Trypanosomosis in dogs in dogs. *Indian Journal of Veterinary Medicine* , 24: 117.

URQUHART, G. M., ARMOUR, J., DUNCAN, J.L., DUNN, A.M. and JENNINGS, F.W. (2002). Clinical aspect of Trypanosomosis in dogs. Veterinary Parasitology, 2nd edition. Blackwell Science Co. UK. Pp. 217.

VADEN, S.L. (2010). Glomerular disease In: Ettinger, S.J., Feldman, E.C. (eds). Textbook of Veterinary Internal Medicine, 7th edition, St. Louis (MO): Saunders (Elsevier). Pp. 2021-2036.

VADEN, S.L. and BROWN C.A. (2009). Glomerular disease In: Bonagura, J.D., Twedt, D.C. (eds), Kirk`s Current Veterinary Therapy XIV. St. Louis (MO): Saunders (Elsevier) Pp. 863-868.

VAN VELTHUYSEN, M.I.E. and FLORQUIN, S. (2000). Glomerulopathy associated with parasitic infections. *Clinical Microbiology*, 13 (1): 55-66.

WILLIAM, G.C. (1985). Mechanism of glomerular injury in immune-complex disease. *Kidney International*, 28: 569 - 583.