International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Impact Factor (2012): 3.358

Microfilariosis in Dogs – An Emerging Cause for Renal Failure

Ambily, V.R[1], Usha. N. Pillai[2]

[1]Assistant Professor, Department of Clinical Veterinary Medicine,
College of Veterinary and Animal Sciences, Mannuthy, Thrissur, Kerala,, India

[2]Associate Professor, Department of Clinical Veterinary Medicine,
College of Veterinary and Animal Sciences, Mannuthy, Thrissur, Kerala, India

Abstract: ***Filariasis is a disease of great public health importance in the country. It can remain asymptomatic for years. Since Kerala is an endemic focus of brugian filariosis, we have to consider the role of natural filarial infections as one of the possible cause for increasing incidence of renal failure in human beings and dogs. The present study was carried out to elucidate the possible role of microfilariae in renal diseases in dogs. Hundred dogs above 6 months of age presented to Veterinary college hospital with clinical signs suggestive of microfilariosis were utilised for the study. Animals screened positive for microfilaria by wet film examination were subjected to detailed study and treatment trials. Serum samples were collected from all the animals, blood urea nitrogen (BUN) and creatinine were analysed and compared with healthy controls. Urine samples of microfilaraemic and six healthy controls were analysed for specific gravity, bile pigments and protein ,urine protein creatinine ratio, γ glutamyl transferase, alkaline phosphatase (ALP) and N-Acetyl-β-D-Glucosaminidase (NAG). Ultrasonographic evaluation of kidneys of a few affected animals revealed hyperechoic areas in medullary region and renal pelvis with corresponding reduction in corticomedullary delineation indicating advanced renal damage. Results of biochemical and urinalysis revealed elevated levels of BUN, creatinine, urine protein creatinine ratio, NAG, ALP, proteinuria with low specific gravity. Two animals died during the course of treatment were subjected to postmortem examination and lesions indicating chronic interstitial nephritis were observed. Results of ultrasonographic evaluation, biochemical and urinalysis and histopathological evaluation were highly correlated to suggest an impending renal insult. Nephrotoxic changes could be reversed if treatment instituted at an early stage. This renal pathology in canine microfilariosis suggested the involvement of toxic and immunological effects of filarial parasites in the pathogenesis of the disease.***

Keywords: Microfilaria, Dirofilaria, Brugia, Renal failure, Ultrasonography, NAG

1. Introduction

Filariasis is a disease of great public health importance.It can remain asymptomatic for years. Since Kerala is an endemic focus of brugian filariosis, we have to consider the role of natural filarial infections as one of the possible cause for increasing incidence of renal failure in human beings and dogs.The present study was carried out to elucidate the possible role of microfilariae in renal diseases in dogs.

The World Health Organization has initiated the global programme to eliminate lymphatic filariosis (GPELF) by the year 2020 by Mass Drug Administration (MDA). Acccording to Showketh Ali *et al* (2007) the performance of the first round of MDA programme launched in Kerala during the year 2005 was poor and main reasons cited for poor performance were inadequate public awareness, fear of drug reactions etc. Canine filariosis is mainly caused by *Dirofilaria repens*, *Dirofilaria immitis*, *Dipetalonema reconditum*, *Dipetalonema grassi*, *Dipetalonema dracunculoides, Brugia pahangi* etc Recent work conducted at the Department of Clinical Veterinary Medicine, College of Veterinary and Animal sciences,Mannuthy detected human filarial parasite *Brugia malayi* in canines. In addition to the reasons cited, presence of this parasite in canine population may also contribute to the poor performance of MDA programme. Recent surveys conducted by Kanaran (2007) revealed the increased incidence of renal failure in canine population. Langharnrner *et al.* (1997) demonstrated and characterized renal disorders in asymptomatic human microfilaraemic carriers from *Brugia malayi* endemic areas of Kerala. Literature on microfilariasis in human beings revealed that brugian filariosis is one of the major cause for renal failure in human beings which might be due to the toxic and immunological effects of the parasite.

2. Materials and Methods

Dogs above 6 months of age brought to the Veterinary College Hospital, Mannuthy, with clinical signs suggestive of filariosis viz., anorexia, fever, oedema of limb and scrotum, conjunctivitis and lymphangitis were utilized for the present study. Animals screened positive for microfilariae by wet film examination were subjected to detailed clinical examination and clinical procedures like Ultrasonography. Pre and post treatment (7 days after treatment) serum samples were collected from all these animals, blood urea nitrogen and creatinine were analysed and compared with healthy controls. Urine samples of microfilaraemic and six healthy controls were analysed for specific gravity, bile pigments and protein, urine protein creatinine ratio,γ glutamyl transferase, alkaline phosphatase (ALP) and N-acetyl-β-D-Glucosaminidase (NAG) and the data were statistically analysed. All the animals were treated with microfilaricidal drugs. Two animals died during the course of treatment were subjected to post mortem and histopathological examination using standard procedures.

3. Results and Discussion

Out of hundread animals screened for microfilaria by wet film examination, circulating microfilaria could be detected

in 80% of dogs. Staining of blood smear with giemsa (1:10) demonstrated that 16 out of 80 dogs were positive for sheathed microfilaria and remaining were nonsheathed.

Ultrasonogram of kidneys revealed hyperechoic areas in medullary region and renal pelvis and also echogenicity of cortex was increased with decreased cortical thickness and corresponding reduction in corticomedullary delineation. These observations are indicative of renal parenchymal diseases in dogs as suggested by Walter *et al.* (1987).

Mean blood urea nitrogen concentration of healthy control and day 1 and 7 of animals were 16.29±3.46, 33.84±5.10 and 27.84±4.82 mg/dl respectively.Mean serum creatinine value of healthy controls and day1 and 7 of microfilaraemic dogs were 0.33± 0.09, 2.35±0.77 and 1.32±.31 mg/dl respectively.Pre treatment serum biochemical values of microfilaraemic animals showed a statistically significant increase in BUN and creatinine when compared to healthy controls. These findings were in agreement with Ananda and D'souza (2006) and Hashem and Badawy (2008). This might be due to severe kidney dysfunction and intravascular haemolysis associated with the infection as opined by Kitagawa *et al*. (1989). Immune mediated glomerular nephritis, glomerulo sclerosis (Grauer *et al*, 1989) chronic interstitial nephritis and amyloidosis (Rawlings, 1986) were observed in dogs infected with *Dirofilaria immitis* and this might have contributed to the elevated BUN and serum creatinine in the present instance also.

Qualitative urinalysis using dipstick revealed the presence of urine protein (4+) with a mean specific gravity of 1.010. Similar observations were made by Forterre *et al.* (2004) and Raila *et al.* (2007) in dogs with chronic renal disease. Proteinuria with reduced specific gravity suggested renal involvement. Microalbuminuria was an important indicator of early renal damage (Langston, 2004) and was always associated with underlying systemic disease The presence of kidney disease was indicated by a significantly elevated proteinuria in humans with lymphatic filariasis from *Brugia malayi* endemic areas of Kerala (Langharnrner *et al.*, 1997).

The urinalysis of microfilaraemic dogs revealed significant increase in NAG, γ GT and ALP when compared to nonmicrofilaraemic dogs. N-acetyl-β-D-glucosaminidase (NAG) and γ-glutamyl transpeptidase (GGT) are renal tubular enzymes which are primarily located in the lysosomes and brush border respectively of the proximal convoluted tubule and these enzymes were released into the urine as a result of renal tubular injury (Clemo, 1998). Increases in urine NAG and GGT indices allow for earlier detection of renal tubular damage in dogs as reported by Brunker *et al.* (2009). The measurement of activity of renal tubular enzymes like NAG, GGT and alkaline phosphatase were more sensitive for detection of acute renal damage than the current standard veterinary diagnostic tests like assessment of serum creatinine, blood urea nitrogen (BUN) and urine specific gravity (Cowgill and Francey, 2005). Increased urinary NAG activity was observed by Kanaran (2007) in dogs with chronic renal failure and Langharnrner *et al.* (1997) in human brugian filariasis patients from Kerala.

On autopsy and histopathological examination, the kidneys revealed vacuolation, necrosis and predominantly hyalinization and atrophy of glomeruli with the presence of dense granular deposits adjacent to the glomeruli, diffuse tubular necrosis with desquamation of tubular epithelium and fibrous tissue proliferation in the interstitium indicating chronic interstitial nephritis. This is consistant with the findings of Kamalu (1991) in dogs infected with *Dirofilaria repens* and Shirota *et al.* (1979) in *Dirofilaria immitis* infected dogs. The renal tubules with hyaline cast indicating the presence of proteinuria. Persistant proteinuria, being a marker of renal disease was associated with progressive glomerular and tubulointerstitial lesions resulting in loss of nephrons as observed by Grauer (2005) in dirofilariasis.

To conclude elevated levels of BUN, creatinine, urine protein creatinine ratio, NAG, ALP, proteinuria with low specific gravity confirmed the renal involvement in microfilaraemic dogs irrespective of the type of microfilaria involved in the disease process. Renal pathology in canine microfilariosis suggested the involvement of toxic and immunological effects of these parasites in the pathogenesis of the disease as suggested by Kamalu (1991). Post treatment biochemical values and ultrasonographic observations indicated that nephrotoxic changes could be reversed if treatment instituted at an early stage. Early diagnosis of this condition is facilitated by the estimation of urinary enzymes such as NAG, GGT and ALP.

References

[1] Ananda KJ and D'souza PE.(2006). Haemato-biochemical changes in dogs infected with microfilariosis caused by *Dirofilaria repens*. *Indian J. Vet. Med.* 26:139-140.

[2] Brunker JD, Ponzio NM and Payton ME. (2009). Indices of urine N-acetyl-β-D-glucosaminidase and γ-glutamyl transpeptidase activities in clinically normal adult dogs. *Am. J. Vet. Res.* 70: 297-301.

[3] Clemo FAS (1998). Urinary enzyme evaluation of nephrotoxicity in the dog. *Toxicol. Pathol.* 26:29–32.

[4] Grauer GF, Culham CA, Dubielzig RR, Longhefer SL and Grieve R B. (1989). Experimental *Dirofilaria immitis*-associated glomerulonephritis induced in part by in situ formation of immune complexes in the glomerular capillary wall. *J. Parasitol.* 75: 585-593.

[5] Grauer GF.(2005). Canine glomerulonephritis: new thoughts on proteinuria and treatment. *J. Sm. Anim. Pract.* 46: 469-478.

[6] Hashem M and Badawy A.(2008). Haematological and biochemical studies on filariasis of dogs. *The Internet J. Vet. Med.* 4 : 1-12.

[7] Forterre S, Raila J and Schweigert FJ. (2004). Protein profiling of urine from dogs with renal disease using Protein Chip analysis. *J. Vet. Diagn. Invest.*16: 271–277.

[8] Kanaran PP.(2007). Clinico-biochemical and ultrasonographic evaluation of renal failure in

[9] dogs. *M.V.Sc thesis.* Kerala Agricultural University, Thrissur. 139p.

[10] Kamalu BP. (1991). Canine filariasis caused by *Dirofilaria repens* in South eastern Nigeria. *Vet. Parasitol.* 40: 335-338.

[11] Kitagawa H, Sasaki Y and Ishihara K.(1989).Clinical studies on canine dirofilarial haemaglobinurea: measured and calculated serum osmolatities and osmolar gap. *Jap. J. Vet. Sci.* 51(4): 703-710.
[12] Langharnrner J, Birkl HW and Zahner H.(1997). Renal disease in lymphatic filariasis: evidence for tubular and glomerular disorders at various stages of the infection *Trop. Med. Int. Hlth.* 9: 875-884.
[13] Langston C. (2004). Microalbuminuria in Cats. *J. Am. Anim. Hosp. Assoc* . 40:251-254
[14] Raila J, Aupperle H, Raila G, Schoon HA and Schweigert FJ. (2007). Renal pathology and urinary protein excretion in a 14 month old Bernese Mountain dog with chronic renal failure. *J. Vet. Med.* 54:131-135.
[15] Rawlings CA.(1986).Heartworm Disease in Dogs and Cats. W.B. Saunders Co., Philadelphia, p. 207.
[16] Showkathali MK, Rajendran R, Regu KI, Mohanan MK, Dhariwal AC and Lal S.(2007). Study on the factors affecting the MDA programme in Kerala State. *J. Commun. Dis*. 39: 51-56.
[17] Shirota K, Takahashi R, Fujiwara K and Hasegawa A.(1979). Canine interstitial nephritis with special reference to glomerular lesions and filariasis. *Nippon Juigaku Zasshi.* 41:119-129.
[18] Walter PA, Feeney DA, Johnston GR and O'Leary TP (1987). Ultrasonographic Evaluation of Renal Parenchymal Diseases in the Dog: 32 cases (1981-1986). *J. Am. Vet. Med. Assoc.* 191:999-1007.

Author Profile

Dr Ambily. V. R, a post graduate in Clinical Veterinary Medicine, presently working as Assistant Professor, Department of Clinical Veterinary Medicine, College of Veterinary and Animal Sciences, Mannuthy. Kerala Veterinary and Animal Sciences University.Awarded Young Scientist Award in the year 2010 for outstanding research in Veterinary Medicine.

Dr Usha Narayana Pillai, a PhD holder in Clinical Veterinary Medicine, working as Associate Professor, Department of Clinical Veterinary Medicine, College of Veterinary and Animal Sciences, Mannuthy. Kerala Veterinary and Animal Sciences university.Awarded Smt. Ava Roy Gold medal in the year 2013 for outstanding research and teaching excellence in the field of Veterinary Medicine. Received lot of recognitions and awards for research activities.