NEPHROTIC SYNDROME IN A DOG – A CASE REPORT

Usha N. Pillai, P.P. Kanaran, V.R. Ambily, Elso John and K.M. Jayakumar
Department of Clinical Veterinary Medicine, College of Veterinary & Animal Sciences
Mannuthy, Thrissur, Kerala

Nephrotic syndrome (NS) is defined as the concurrent presence of hypoalbuminemia, proteinuria, hyperchlosterolemia and extravascular fluid accumulation (Abrass, 1997). Nephrotic syndrome is well recognized in dogs (Grauer *et al*., 1992). The present paper deals with a case of nephrotic syndrome in a dog.

Material and Methods

A three year old female Labrador dog was presented to the Veterinary College hospital with the history of reduced food intake, polydipsia, polyuria, and edema around the eyes and submandibular region for about one month. Edema gradually subsides during the evening hours. Clinical data were within normal range except slight congestion of visible mucous membranes. Urinalysis revealed severe proteinuria (++++) with a specific gravity of 1.015. Samples were collected on the day of admission for analysis of haemato-biochemical parameters viz. complete haemogram, serum total protein, albumin, cholesterol, BUN, creatinine and urine protein creatinine ratio using standard procedure. Ultrasonographic examination revealed dilated renal pelvis and ureters. Abdominal fluid also could be detected on ultrasonogram. Haematobiochemical studies revealed leucocytosis (17500/mm^3) mild thrombocytosis (418000/mm^3) hypoproteneuria (3.40 g%), hypoalbuminemia (1.4g%) hypercholesteraemia (408 mg%) with normal serum creatinine (0.8 mg%) and BUN (9 mg%) urine protein creatinine ratio of >3.5 mg/mmol).

Treatment and Discussion

Based on clinical findings and haematobiochemical studies the condition was diagnosed as nephrotic syndrome. The animal was treated with aspirin, enlapril @ 0.5 mg/kg body weight twice daily and lasilactone @ 2 mg/kg body weight daily. Albumin hypercatabolism, down regulated synthesis and compartmental redistribution contribute hypoalbuminemia associated with glomerular disease (Abrass, 1997). Fluid extra vasation is likely multifactorial in origin and thus serum albumin concentration is correlated with, but is not the only determinant of NS. The cause of hyper cholesterotemia in human patient with NS in unclear. Low dose aspirin therapy is essential to control thronboembolism (Relford and Lees, 1996).

NS is a serious complication of kidney disease in dog, usually resulting from glomerulonephritis or renal amyloidosis. The resultant kidney disease or kidney failure allows excessive protein loss through the urine. Through rare, the syndrome is life threatening and potentially fatal (Cook and Cowgill, 1996).

References

Ralford, R.L. and Lers, G.E. 1996. Nephrotic syndrome in dogs. Diagnosis and treatment. *Compend Contin. Edu. Pract. Vet*. **18**(3): 279-293.

Cook, A.K. and Cowgill, L.D. 1996. Clinical and pathological features to protein losing glomerular disease in the dog – a review of 37 cases. *J. Am. Anim. Hosp. Assoc*. **32**(4): 313-322.

Abrass, C.K. 1997. Clinical spectrum and complications of the nephrotic syndrome. *J.Invest. Med*. **45**: 143-153.

Gauer, G.F., Frisbic, D.D., Synder, P.S. 1992. Treatment of membronoproliferative glomerular nephritis and nephrotic syndrome in a dog with a thromboxane synthesis inhibitor. *J. Vet. Intern. Med*. **6**: 77-81.