NEPHROTIC SYNDROME IN A SIAMESE CAT

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Nephrotic syndrome is a relatively rare end-stage renal disease of cats, defined as the combination of four hallmark clinical signs:

1) significant protein loss in urine (proteinuria)
2) low serum albumin (hypoalbuminemia)
3) edema and/or other abnormal fluid accumulation (Ascites)
4) elevated blood cholesterol level (hyperlipidemia, particularly hypercholester-olemia)

The average age of onset in cats varies, but middle-aged and old cats are most commonly affected. In most cases, a proliferative glomerulonephritis is an underlying cause of nephrotic syndrome (Bishop S.A. *et al.,* 1992). There appears to a sex predilection in cats with 75% of cases occurring in males.

Animals with proteinuria, hypoalbuminemia, and hyperlipidemia without edema or ascites are considered to have a **nephrotic tendency**. Dogs and cats are less likely to develop overt dependent edema or ascites as compared with human beings. Development of edema or ascites requires sodium retention leading to fluid overload in addition to the hypoalbuminemia; it may be that domestic species are less prone to sodium retention than are humans. It should be remembered that nephrotic tendency results from a loss of glomerular permselectivity not glomerular permeability. Azotemia is **not** a requisite clinical component of nephrotic tendency (Katherine M. James, 1998).

Common causes of nephrotic syndrome in cats include: Glomerulonephritis (White J.D. *et al.,*2008 and Cavana P. *et al.,*2008); Amyloidosis; Chronic interstitial nephritis (Bishop S.A. *et al.,*1992[a]); Feline infectiousperitonitis;Renal lymphosarcoma; Potassium depletion nephropathy.

Nephrotic syndrome is a set of above mentioned clinical signs that may develop as a secondary to the above effects of glomerular injury.

A 5year old castrated male Siamese cat was brought to the Faculty Clinics with the history of subcutaneous oedema of face, body and hind legs (Fig.1), vomition and anorexia since a week. The cat was depressed, emaciated and inactive and had dry and lustreless coat, regular and predominantly costal respiration, pendulous abdomen, polyuria, pale mucous membrane, slightly sunken eye balls with dry cornea, and regular, hard and incompressible pulse. Examination of heart revealed decrease in point of maximum impulse and mild heart sounds, but no adventitious heart sounds. Lungs were almost normal. Palpation and percussion of abdomen indicated presence of fluid thrill. Radiograph of Chest and abdomen showed important nephroitic lesions (Fig.2). Urine analysis revealed massive proteinuria (0.7 g/dl) and epithelial casts. There was normocytic anaemia with normal leucogram. Biochemical analysis of blood/serum indicated elevated blood urea (160 mg/dl) and serum creatinine level (7.5 mg/dl), hypoalbuminaemia (2.1 g/dl) and normal levels of total serum protein (6.7 g/dl) and cholesterol (175 mg/dl).

The Cat was treated with Ampicillin 6 mg/kg bid, 5% Dextrose @25mg/kg iv daily, Vitamin – E 100 IU per day, B-Complex 0.5 ml i/m daily and Furosemide 2 mg/kg daily were given for a weak. For proteinuria and Hypertension Enalapril @ 0.1 - 0.5 mg/kg q 24 hrs was administered. For Hypercoagulability treatment, Aspirin

0.5 mg/kg (q 72 hrs) was also administered. Dietary sodium restriction and high-quality, restricted-quantity protein diets were recommended as referred by Grauer G.F. and DiBartola S.P.(1995). The cat could not be saved even after all efforts and died on 10th day.

Fig.1:The classic appearance of a nephrotic Syndrome cat with facial oedema and pot-bellied appearance

Fig.2 Radiograph of the Cat with nephrotic syndrome

Postmortem examination revealed, about 1 litre of straw coloured fluid in the abdominal cavity, congested and oedematous lungs, hard and rounded border liver and enlarged and brownish colour kidneys containing multiple haemorrhagic areas in cortex and severe congestion at cortico-medullary junction. The histopathological changes of membranous glomerulonephrit are microthrombi in glomerular capillaries, haemorrhagic necrosis and infarction indicates a vascular pathology as a probable cause. Membranous nephropathies are generally associated with heavy proteinuria and frequently an insidiously progressive course leading to renal failure. Proteinuria, presence of urinary casts, elevated blood urea and serum creatinine indicated renal failure.

Very few studies of companion animal patients are published. When an underlying disease is found, correction of that disorder is often the most critical aspect of therapy. Unfortunately, such as underlying disorder is rarely found. Supportive care for patients with renal failure, hypertension, ascites, or edema is always indicated.

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