Short Communication

Therapeutic Management of Chronic Renal Failure in a Dog

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Abstract

A case of chronic renal failure was diagnosed in an eight year old Labrador dog on the basis of history, clinical signs, physical examination, hematology, plasma biochemistry, followed by confirmation with the help of ultrasonography and urinalysis. Urinalysis on the day of presentation revealed turbid straw colored urine, decreased specific gravity, trace amount of non-haemolysed blood, alkaline pH, marked proteinuria and pyuria with high inflammatory cells and epithelial cast. Basing on the plasma creatinine value, the case was diagnosed as terminal stage 4 chronic renal failure. The patient was treated with fluid therapy, antibiotic, antacid, antiemetic, calcium supplement, multivitamin, and erythropoietin. Though the prognosis in case of end stage chronic renal failure is guarded, nevertheless, it took 30 days to bring back the animal from stage 4 to stage 2 with clinical signs of improvement and it was followed up to 5 months without any further complication.

Key words: Chronic Renal Failure, Hematology, Plasma Biochemistry, Ultrasonography, Urinalysis

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Introduction

Chronic renal failure (CRF) is defined as progressive impairment in the kidney's ability to concentrate urine, excrete nitrogenous waste and maintain electrolyte homeostasis due to loss of nephrons over a period of months to years (Nelson and Couto, 2014). It is caused due to different reasons like congenital and familial diseases, infectious and inflammatory disorders, amyloidosis, neoplasia of the kidneys, nephrotoxic

substances and hyperthyroidism etc. CRF is a common problem in aged dogs and is associated with significant morbidity and mortality (Nelson and Couto, 2014). Clinically CRF is manifested by depression, polyuria, polydipsia, weakness, anorexia, vomition, diarrhoea, pale mucous membrane, oral ulceration, halitosis along with high blood urea nitrogen (BUN) and creatinine (Nelson and Couto, 2014). The present report communicates a case of CRF with its clinical presentation, diagnosis, and its medical management in a senile dog.

An 8-year-old male Labrador dog weighing 34 kg was presented with the history of anorexia, vomiting and oliguria for last 5 days. Careful collection of past history revealed cystitis approx. 5 months back and treated for the same with tablet Augmentin, herbal renoprotectives and chlorpheniramine maleate with clinical improvement as per the owner. On physical examination, rectal temperature was 99.8° F, pale conjuctival mucus membrane, pain on palpation of bladder and kidney and pulse rate of 54 bits/min. Ultrasonography of kidney revealed hyper echoic kidney (left and right) cortex (Fig. 1), indistinct cortico-medullary junction, thickened and hyper echoic urinary bladder wall.

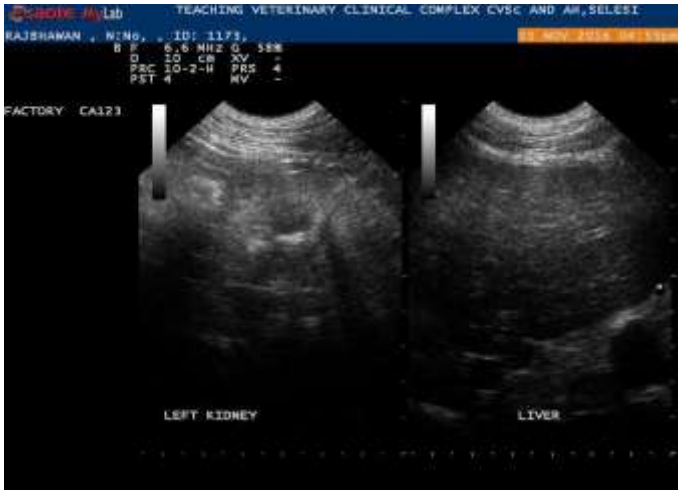

Fig.1: Renal ultrasonogram revealing hyperechoic cortex (compared to liver) and indistinct cortico-medullary junction

Splenic and hepatic ultrasonogram revealed splenomegaly and hepatitis, respectively. So, ultrasound gave the impression of cystitis, nephritis, splenomegaly and hepatitis. The radiography of abdomen revealed small size liver, enlarged spleen and gas filled intestine. Urinalysis (Table 1) with the help of commercially available urinalysis reagent strip on the day of presentation (day 0) revealed turbid straw colored urine, decreased specific gravity, trace amount of non-haemolysed blood, alkaline pH, marked proteinuria, and marked leucocyte count (125cells/µl).

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Table 1: Changes in urinalysis pattern on different days of therapy

Parameters	Day 0	Day 15	Day 30	Reference
Color	Straw colored	Straw colored	Transparent	Amber
Transparency	Turbid/Cloudy	Turbid	Clear	Clear
P^H	8	7.5	6.5	5.5-7.5
Specific gravity	1.005	1.01	1.025	1.015-1.050
Glucose	-ve	-ve	- ve	-ve
Bilirubin	-ve	Small (+)	-ve	0 to 1+
Ketone	-ve	Trace (5mg/dl)	-ve	-ve
Blood	Non haemolysed trace	Non haemolysed moderate	-ve	-ve
Protein (mg/dl)	++++ ve (≥ 2000mg/dl)	+++ve (300mg/dl)	+(30 mg/dl)	0-30mg/dl
Urobilinogen	-ve	-ve	-ve	-ve to weak +
Nitrite	-ve	-ve	-ve	-ve
Leucocyte	125 cells/µl	70 cells/µl	-ve	-ve

Microscopic examination of urine sediment revealed intact RBCs, high inflammatory cells (mononuclear cells) and epithelial cells (Fig. 2).

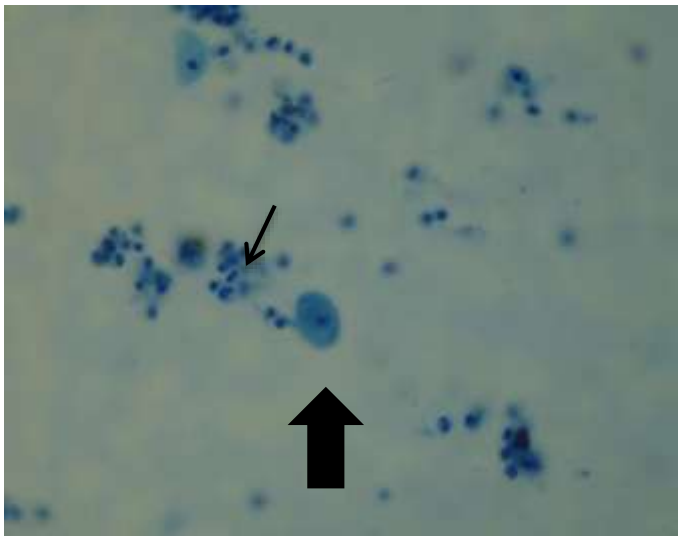

Fig. 2: Urine sediment showing epithelial cell (thick arrow) and mononuclear cells (thin arrow) (0.5% Methylene blue stain, 40X).

Haematology (Table 2) revealed anaemia, monocytosis with lymphocytic neutropenia. Plasma biochemistry revealed hypoalbuminemia, hyperglobulinemia, increase in the activity of aspartate aminotransferase (AST), azotaemia, hyperphosphataemia and hypocalcaemia. So, on the basis of history, clinical signs, haematology, plasma biochemistry, urinalysis, ultrasonography and radiography, the case was diagnosed as CRF.

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Table 2: Changes in haemato-biochemical parameters on different days of therapy

Parameter	Day 0	Day 15	Day 30	Reference Range
Hb (g/dl)	9	8.7	14.6	18-Dec
Packed cell volume (%)	30	28.7	42	37-55
Total erythrocyte count (x 10^6/$mm^{3)}$	4.19	3.96	5.98	5.5-8.5
Total leukocyte count (x 10^3/$mm^{3)}$	6.63	7.76	15.31	17-Jun
Platelet (x 10^3/$mm^{3)}$	185	205	210	200-500
Differential leukocyte count				
N (%)	46.5	62.8	76.7	51-84
L (%)	33.1	23.2	14.9	Aug-38
M (%)	15.4	9.2	3.4	9-Jan
E (%)	5	4.8	5	0-9
Plasma Biochemistry				
Total Protein (g/dl)	6.8	6.8	6.6	5.4-7.5
Albumin (g/dl)	1.2	2.2	3.2	2.3-3.1
Globulin (g/dl)	5.6	4.6	3.4	2.4-4.4
Total bilirubin (mg/dl)	0.8	0.8	0.3	0-0.3
Direct bilirubin (mg/dl)	0.2	0.2	0.2	0-0.4
Alanine aminotransferase (U/L)	30.8	30.8	38	10-109
Alkaline phosphatase (U/L)	97.4	77.4	91.2	1-114
AST (U/L)	91.7	21.7	24.5	13-15
BUN (mg/dl)	140	120.3	64.3	28-Aug
Creatinine (mg/dl)	11	7.5	2	0.5-1.7
Ca (mg/dl)	3.9	2.9	8.5	9.1-11.7
P (mg/dl)	12.3	11.1	3.4	2.9-5.3

Results and Discussion

The patient was given Inj. Dextrose 10% @ 10 ml/kg I/V, Inj RL @ 10 ml/kg I/V, Inj Amoxirum forte @ 10 mg/kg I/V BID, Inj Rumeric (multi-vitamin) 2 ml I/V OD, Inj Metoclopramide @ 0.2 mg/kg BD for an initial period of 15 days followed by tab. Calcium Sandoz (625mg) – @ 15 mg/kg PO TID and Cap. Omeprazole -D @ 1 mg/kg PO OD for 30 days and Inj. Zyrop 2000 IU (Human recombinant erythropoietin) @ 50 IU/kg I/V three times a week. Fluid therapy was given to provide energy and electrolytes. Antibiotic (chosen on the basis of antibiotic sensitivity test), multivitamin and antiemetic were given to check secondary bacterial invasion, to boost immunity and to check vomition, respectively. Tab. Calcium sandoz and Cap. Omeprazole -D was given to check hyperphosphatemia and gastric acidity, respectively. Zyrop was given to enhance erythropoiesis. Diuretic was not used to avoid dehydration and prerenal azotemia (Nelson and Couto, 2014).

Basing on creatinine value, the case was diagnosed as terminal stage 4 chronic kidney failure underscoring uraemia with associated clinical signs of vomiting, lethargy, anorexia, and dehydration. Pale conjuctival mucus membrane was due to the presence of a non-regenerative, normocytic, normochromic anaemia (Kahn, 2010). On urinalysis (by urinalysis reagent strip), turbid colored urine was indicative of high

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epithelial casts, leukocytes and haematuria. Decreased specific gravity was due to uraemia causing lack of concentration of urine by kidneys, alkaline pH of urine might be attributed to the metabolic alkalosis or presence of urease positive bacteria in the urine. Marked proteinuria and high leucocyte count were indicative of cystitis as well as nephritis (Nelson and Couto, 2014). Ultrasonographic evidence was in accordance with earlier report (Kavitha *et al.,* 2013). Lymphocytic neutropenia was indicative of long standing disease. Plasma hypoalbuminemia was due to urinary loss of albumin, hyperglobulinemia might be due to inflammation. Azotaemia, hyperphosphataemia and hypocalcaemia could be due to a reduction of functional renal mass (Nelson and Couto, 2014). Though the prognosis in case of end stage CRF is guarded, nevertheless, it took 30 days to bring back the animal from stage 4 to stage 2 with clinical signs of improvement and the patient remained stable at stage 2 for the next 5 months without any further complication.

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