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Ameliorative effect of hemodialysis in a dog having chronic kidney disease

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Abstract
A 4-year-old Labrador retriever bitch was presented to the clinics with a complaint of acute onset of vomiting, oliguria, lethargy and anorexia for two weeks. The case was diagnosed with chronic kidney disease (CKD).The animal was depressed with poor physical status. A thorough routine hemato-biochemical analysis showed severe Anemia (Hb-7.3%), elevated blood urea nitrogen (156 mg/dl), creatinine (5.8 mg/dl) and phosphorus (11.8 mmol/L). Medicinal treatment was given and hemodialysis was performed to treat against CKD. Hemodialysis with supportive therapy successfully treated with improved acute phase and kidney function.

Keywords: Dog, chronic kidney disease, hemodialysis

Introduction
Hemodialysis can be used for the treatment of acute or chronic kidney disease. The basic principle for hemodialysis is that it treats renal failure by removing uremic toxins by using the extracorporeal circulation of a patient's blood to exchange solute through an artificial kidney (hemodialyzer). Hemodialysis is effective in controlling azotemia, hyperphosphatemia and hyperkalemia, when performed on a daily or alternate day basis (DiBartola *et al*, 1985) [3]. In this case, hemodialysis with the supportive measures improved the kidney compensation and the physical as well as the physiological status of the uremic patient was markedly improved.

Case description, diagnosis and treatment
A 4-year-old female Labrador retriever having 40 kg body weight, was brought to the Veterinary Clinical Complex for treating against acute vomiting, anorexia, lethargy, oliguria and depression that was running more than a week. There was no history of taking abnormal foods or toxicants. The bitch was under well management, vaccinated, regularly dewormed as per the advice of a veterinary physician. Seven days before, the bitch had been examined by the veterinary physician. On clinical examination the bitch showed a pallor mucous membrane, an increased respiration rate (51 bpm) and heart rate (110 bpm). Rectal temperature was within normal range. Moderate dehydration was noted. Thoracic auscultation revealed cardiac murmur. According to the owner's description, the patient was suffering from heat and exercise intolerance since a week. A thorough hemato-biochemical profiles with urinalysis were considered for evaluation.

Low hemoglobin (7.3%), low red blood cell count ($3.4x10^6$ /µL), low packed cell volume (26%) with low concentration of platelet ($0.62x10^6$/µL) were noted in the complete blood count (CBC).In the serum biochemistry, elevated blood urea nitrogen (BUN, 156 mg/dl), creatinine (5.8 mg/dl), elevated alanine aminotransferase (SGPT, 94 IU/L), higher level of alkaline phosphatase (ALP, 178 IU/L), hypoalbuminemia (1.9 gm/dl) and hyperphosphatemia (11.8 mmol/L) were noted.

The initial treatment that was performed by the local veterinary physician consisting of oxygen support as and when required, administration of Ringer's lactate @ 50ml iv twice daily, dextrose 25% @ 50 ml iv twice daily, Inj. pantoprazole 40 iv once daily, Inj. ondansetron @ iv twice daily, inj. polybion @ 2 ml iv twice daily, sucralfate syrup @ 1 gm po, 8 hrs interval, Inj. ampicillin @ 22mg/kg b wt iv twice daily, with provision of salt free diet and oral aluminium hydroxide @ 200 mg, po, q6h with the meals.

On initial presentation, results of the physical examination were unremarkable. The dog was mildly dehydrated, oliguric and extremely depressed (Fig 1). Auscultation of thorax revealed normal lungs sound. Abdominal palpation was normal. The mean arterial blood pressure was 105 mmHg. Urine analysis revealed isosthenuric with specific gravity-1.010. Proteinuria, pyuria and bacteriuria were not present. Antibody titres to Leptospira *sp* were negative. Microscopical blood smear examination revealed absence of hemoprotozoa or rickettsia. A urine culture and fecal floatation examination report were negative. Abdominal radiographs showed no abnormalities. Abdominal ultrasonographs showed atrophied left kidney without cortico-medullary alteration and in right kidney, cortico-medullary demarcation was lost (Fig 2&3). An ECG was also done having no such abnormalities. A diagnosis of chronic kidney disease (CKD) was suspected, based on the duration of signs, significant azotemia, Ultrasonography report and the isosthenuric specific gravity of urine.

Initiation was taken with oxygen support for the animal to make it stable for the hemodialysis. After intravenous fluid administration for 24 hours, the dog gained 400 gm body weight, and clinically appeared overhydrated, the plasma potassium concentration had increased from 4.87 to 5.32 mmol/L. Inj. Erythropoietin (Eporise^{R}) at the dose rate of 100 U/kg b wt was administered subcutaneously. A urinary catheter was placed to monitor urine production. The urine production improved 150 ml to 260 ml in 24 hours after intravenous administration of mannitol (20%) at a dose rate of 1 gm/kg b wt and furosemide @ 2 mg/kg b wt. On second day, BUN and creatinine level were checked. But, because of the non-progressive condition with oliguria despite of fluid and diuretic therapy, hemodialysis was started on day 3.

The dog was premedicated with atropine sulphate @ 0.04 mg/kg b wt subcutaneously. No anesthesia was induced as the dog was very much cooperative. A 40 cm dual lumen dialysis catheter was placed in the right jugular vein. Mean arterial blood pressure was maintained between 90 and 120 mmHg throughout the procedure. A one-hour dialysis treatment was performed (Fig 4). The post dialysis BUN, creatinine values improved to 85 mg/dl, 3.9 mg/dl respectively and Hb value was 7.1g/dl. Mannitol @ 0.25 g/kg b wt was given for follow up treatment. On day 6, hemodialysis was again performed and the post dialysis BUN, creatinine and Hb were 32.1 mg/dl, 1.8 mg/dl and 9.2 g/dl respectively. Urine production was improved. The dog was in complete rest and was under the follow up medicinal treatment like capsule CK Reno @ 2 caps bdpc, Rinonadyl @ 3 caps daily, phosclear powder @ 8 scoop with food in three divided dosage, Inj. erythropoietin once in a week with multivitamin syrup.

Discussion

CKD can be caused by a prerenal cause, such as severe dehydration or decreased perfusion due to cardiac diseases, renal causes such as toxic insult, infectious agent, ischemic event or a sequel to a systemic disease, or a post renal cause, such as a ureteral or urethral obstruction (Cowgill and Francey, 2005) [1]. The anemia was attributed to gastrointestinal bleeding or the decreased erythrocytic life span that occurs in CKD due to a malfunctioning of the membrane $Na^{+}K^{+}$ATPase pump and impaired regeneration of reduced glutathione need to prevent Hb oxidation (Polzin *et al*, 2005) [2]. In CKD patient, there is high probability that the patient's ability to synthesize and secrete endogenous erythropoietin is impaired, consideration should be given to administration of an erythropoietin stimulating agent early in the course of hospitalization (Cowgill *et al*. 1998) [7]. Injection Eporise^{R} is a man -made version of human erythropoietin that stimulates the bone marrow to produce red blood cells. Intravenous fluid therapy was considered immediately to correct uremia and electrolyte imbalance as a primary aim of the therapeutic plan.CK-Reno is a composite rug of several herbal ingredients with some essential amino acids that help to reduce oxidative stress, inflammation, controls glomerular hypertension, reduces creatinine, BUN and improve kidney function. Rinonadyl contains probiotics that eliminates uremic toxins. Phosclear contains aluminium hydroxide that acts as a phosphorus binding agent that helps to prevent hyperphosphatemia thus prevents further kidney damage. Hypokalemia is a common manifestation of CKD, that the dogs' benefit from potassium supplementation through the intravenous fluids (Polzin, 2011).

It is generally seen that once the patient's hydration status is normalized, the plasma or serum creatinine concentration usually decreases by at least 1 mg/dl per day. But within 48 hours of fluid therapy, azotemia did not improve, the plasma BUN was 134, and creatinine had increased from 3.9 to 4.2. Hemodialysis was chosen as the preferred treatment in this case because the medicinal therapy did not resolve the azotemia within 2 days.

Dialysis should be initiated when the clinical azotemia with fluid and electrolyte imbalance and oliguria or anuria cannot be managed effectively with medicinal therapy alone. If hemodialysis is not available, peritoneal dialysis is an alternative therapy that can be used to manage CKD. Hemodialysis is a technically sophisticated therapy that is used to remove uremic toxins and correct the electrolytes, hydrogen ion and fluid imbalances associated with renal failure (Grauer, 2009) [6]. After two successive hemodialysis, the BUN and creatinine values improved significantly. Simultaneously, oral medications, scientific management and fluid therapy considerably improved the physical condition of the patient. The owner was advised to check her patient's hematobiochemical valuation weekly and remained under the routine observation of the doctors.

The purpose of treatment in CKD patient is to prevent and manage the complications and subsequent effects of renal damage. The progressive renal tissue damage in CKD patient can be well managed by proper therapeutic measures, renal replacement therapy and dietary schedule. The hemodialysis with medicinal therapy can extend the quality life in dogs and cats. In this case, hemodialysis, which is a part of renal replacement therapy remained successful in composing the physical status of the patient as well as kidney function remained stabilized for maintaining quality life.

Table 1: Hematobiochemical evaluation before and after two successive hemodialysis treatment

Parameters	Before treatment	After two successive hemodialysis	Reference range
Hb%	7.3	9.2	12.1-20.3
RBC	3.4/lac (x 10^6)	5.9	6.5-12.5
PCV (hematocrit)	26%	40.4	37-54
MCV	72.6 fL	67.8	66-75
MCH	21.7 pg	20.1	22-27
MCHC	28 g/L	30.7	34-36
Platelet count	0.62/ lac (x 10^6)	0.89	1-6
WBC (TC)	9100/cu.mm	13,500	5,000-14,000
Neutrophils	62%	65	62-80
Lymphocytes	32%	30	10-28
Monocytes	04%	03	03-09
Eosinophil	02%	04	02-14
Basophil	00%	00	00-01
ESR (1st hr)	32 mm/1st hr	18	6-10
Blood Urea Nitrogen (BUN)	156 mg/dl	32.1	6.8-25.9
Serum creatinine	5.8 mg/dl	1.8	0.4-1.6
Serum SGOT (AST)	37 IU/L	57	12-37
Serum SGPT (ALT)	91 IU/L	89	17-69
Alkaline phosphatase (ALP)	178 IU/L	94	15-130
Serum phosphorus	11.8 mmol/L	2.2	0.68-2.03
Glucose (Fasting)	91 mg/dl	98	55-102
Serum bilirubin	0.15 mg/dl	0.3	0.0-0.4
Total serum protein	6.7 gm/dl	5.8	5.4-7.1
Albumin	1.9 gm/dl	2.7	2.6-4.7
Globulin	4.8 gm/dl	3.1	1.6-3.7
Serum sodium	142 mEq/L	146	140-150
Serum potassium	4.87 mEq/L	5.6	5.4-7.1
Serum chloride	127 mEq/L	118	109-120

Table 2: Concentration of blood urea nitrogen (BUN) and creatinine before and after treatment

Evaluated on	BUN (gm/dl)	Creatinine (gm/dl)
Day 1	156	5.8
Day 2	154	5.6
Day 3 (post dialysis)	85	3.9
Day 6 (post dialysis)	32.1	1.8

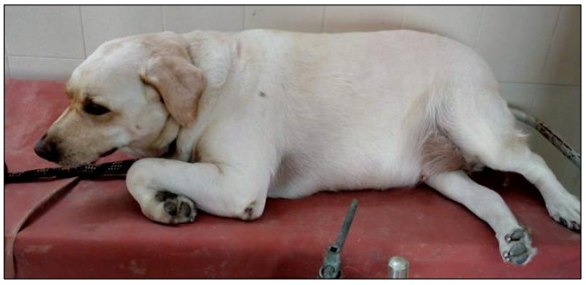

Fig 1: The dull, depressed, anorectic ARF patient

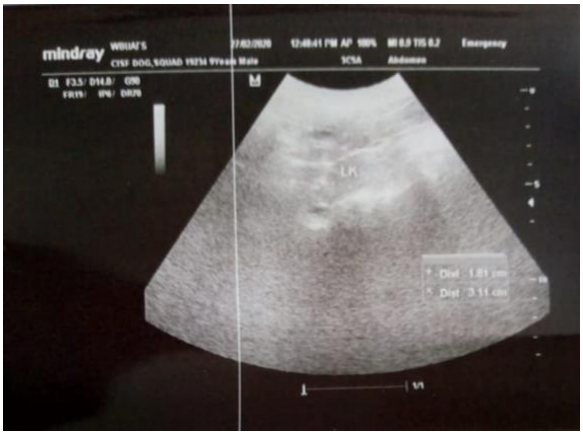

Fig 2: USG showing atrophied Left Kidney

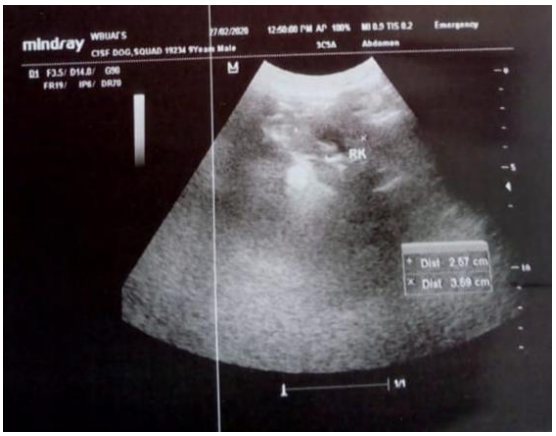

Fig 3: USG showing cortico-medullary demarcation lost in right kidney

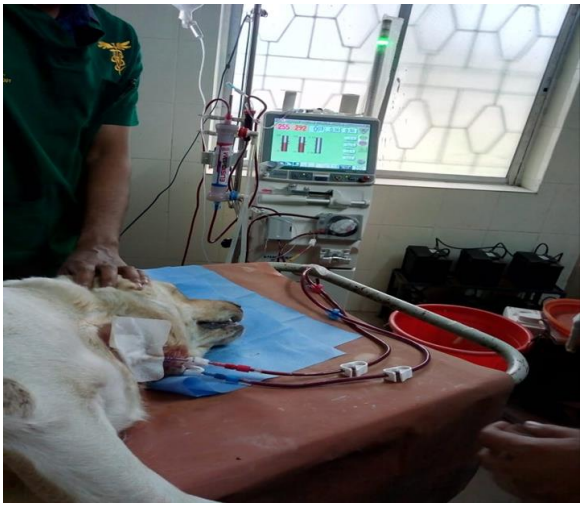

Fig 4: Performing Hemodialysis in CKD patient

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