THERAPEUTIC MANAGEMENT OF ACUTE KIDNEY INJURY IN ADOG

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A 3 years old Golden Retriever dog presented to VCC, Nagpur Veterinary College, Nagpur with anorexia, vomiting and lethargy. Clinical examination revealed mild dehydration and splenomegaly. Blood analysis revealed slight neutrophilia, rise in serum creatinine, and BUN levels. The dog had history of treatment with meloxicam and paracetamol combination injection for last 4-5 days. Based upon history and blood analysis the case was diagnosed as a case of Acute Renal Failure. Therapeutic management with fluid therapy, reno-protectant drugs, and symptomatic treatment was instituted. The dog showed satisfactory recovery and reduction in azotaemia within 3 weeks of treatment.
Keywords: Acute Renal Failure, Anorexia, Azotaemia.

In veterinary medicine, acute kidney injury is a prevalent diagnosis that is linked to significant morbidity and mortality (Rimer *et al.,* 2022). The overall case fatality rate for dogs managed for AKI is still as high as 45% to 60% despite improvements in AKI management and greater accessibility to renal replacement medicines or haemodialysis (Legatti *et al*., 2018; Ross, 2011). Several factors are involved in the pathophysiology of AKI in dogs. The most common etiologies include ischemia, inflammation, exposure to nephrotoxins, and infectious diseases (Legatti *et al*., 2018; Segev, 2011). Nephrotoxins include Heavy metals, Organic compounds, Ethylene glycol, Lily plant, Grapes or raisins, Envenomation (eg, snake, bee, wasp, bull ants) , Melamine/cyanuric acid and drugs like Aminoglycoside antibiotics, Cisplatin, Amphotericin B, Non-steroidal anti-inflammatory drugs and Radiographic contrast agents (Ross, 2011), treatment of osteoarthritis and perioperative pain, NSAIDs and selective cyclooxygenase (COX) inhibitors are frequently utilised in veterinary medicine (Forsyth, 2000).

Lethargy, anorexia, vomiting, diarrhea, anuria, polyuria, and polydipsia are some of the non-specific clinical symptoms that are most frequently associated with AKI. These clinical symptoms result from an accumulation of uremic toxins, other body organs involvement, as well as comorbidities and complications (Rimer *et al.,* 2022). There is no specific treatment for NSAID-induced AKI; only supportive care is available. Management with gastric lavage and fluid diuresis (to prevent decreased renal blood flow and to prevent hypotension) is advised when dogs consume potentially toxic doses (Poortinga and Hungerford, 1998).

Materials and Methods

A 3-year-old Golden Retriever dog presented to VCC, Nagpur Veterinary College, Nagpur with anorexia, vomiting, reduced urine output, and lethargy. Clinical examination revealed normal temperature (102.1^{o}F) ,mild dehydration and splenomegaly. The dog had history of treatment with meloxicam and paracetamol combination injection for the last 4-5 days.The Complete Blood Count revealed slight neutrophilia (76.3%), and the concentration of serum urea and creatinine were determined to be 155.5 mg/dl, 8.4 mg/dl ,respectively (Table-1).

The clinical presentation, taken together with the background and laboratory test results, indicated a diagnosis of Acute Renal Failure. Aggressive fluid therapy with Ringer's Lactate crystalloid and Dextrose Normal Saline is instituted. Furosemide @ 2 mg/kgiv q 8hrs, Ceftriaxone @ 25 mg /Kg iv q 12hrs, Antiemetic ondansetron @ 0.2 mg/kg iv q 12 hrs, Proton Pump Inhibitor @ 1mg/kg slow iv given. In addition, Tab.

Sevelamer 400mg with meals as Phosphorus binding agent, ACE inhibitor Tab. Enalapril 0.5 mg/kg and Syrup Rennecare 7.5 ml orally 12hrs prescribed. The improvement in blood analytes showed in Table-2.

TABLE-1. HAEMATOLOGICAL AND BIOCHEMICAL EVALUATION

Test	Observed Values	Test	Observed Values
Haemoglobin	14.6 gm/dl	BUN	155.5mg/dl
TLC	8.0 $x10^3$	Sr. Creatinine	8.6 mg/dl
RBC	5.50 x 10^6	Total Protein	6.47 gm/dl
PCV	43.4 %	Albumin	3.2gm/dl
Neutrophil	76.3%	Globulin	3.27gm/dl
Lymphocyte	14.8%	Sodium	146.6mmol/L
Monocyte	8.9%	Potassium	4.01 mmol/L
Eosinophil	1.3%	SGPT	22.3 IU/L
MCV	79 um^3	SGOT	19IU/L
MCH	26.5 ^{pg}	SAP	153 IU/L
MCHC	33.5 g/dl	Chloride	103.5IU/L
Platelets	348 x $10^3/mm^3$	Calcium	9.2mg/dl
RDW	13.2 %	Phosphorus	5.3 mg/dl
MPV	7.5 $microm^3$		

TABLE-2. CHANGES IN BLOOD ANALYTES DURINGTREATMENT

Test	Day 0	Day 1	Day 2	Day 3	Day 4	Day 10	Day 17
BUN (mg/dl)	155.5	145.7	123.4	86.4	62.2	24.8	14.2
Sr.Creatinine (mg/dl)	8.6	8.4	6.8	5.4	4.44	2.5	1.5
Sodium (mmol/L)	146.6	142.2	151.4	151	149.6	148.1	151.4
Potassium (mmol/L)	4.01	3.73	3.46	3.43	3.37	4.9	4.66
Chloride (mmol/L)	103.5	105	109	105.4	103.6	101.2	99.8
Phosphorus (mg/dl)	5.3	7.1	6.0	4.74	4.6	4.6	5.0

Results and Discussion

The diagnosis of this case was established according to the patient history, clinical signs, haematological and biochemical evaluation. Paracetamol is a commonly used analgesic and antipyretic drug in humans and is toxic to dogs when used at higher doses. Some dogs are less tolerant to this drug than others and may cause fulminant liver failure or kidney failure. Paracetamol has been shown to promote hepatocyte apoptosis but the exact mechanism of paracetamol-induced renal failure is unclear as also reported by Corina *et al.*, 2004. Acute renal failure is treated with specific medication for the underlying cause, as well as supportive care tailored based on the stage of acute renal failure andthe animal's fluid, electrolyte, and acid-base status**.** The cornerstone of acute renal failure treatment is fluid administration, which promotes urine production, normalizes internal fluid balance, and corrects renal blood flow problems. Therefore, in this case, treatment was instituted with aggressive fluid therapy and symptomatic drugs were administered. As the neutrophilia was evident in the CBC broad-spectrum antibiotic was given. Vomiting can be a significant problem in animals with ARF, because uraemia results in increased gastric acid secretion, therefore drugs that inhibit gastric acid production were indicated. Here in this case proton pump inhibitor pantoprazole was given to inhibit

hyperacidity and a 5-HT3 receptor antagonist Ondansetron given to control vomiting.

References

Corina, Lorz, Pilar, Justo, Ana, Sanz, Dolores, Subirá, Jesús, Egido and Alberto, Ortiz. (2004), Paracetamol-Induced Renal Tubular Injury: A Role for ER Stress. *JASN*, **15**(2): 380-389;

Forsyth, S.F., Guilford, W.G. and Pfeiffer, D.U. (2000). Effect of NSAID administration on creatinine clearance in healthy dogs undergoing anaesthesia and surgery. *J. Small Anim. Pract.*, **41**(12): 547–550.

Legatti, SAM, El-dib, R. and Leggati, E., (2018). Acute kidney injury in cats and dogs: a proportional meta-analysis of case series studies. *PloSOne*, **13**: 1-18.

Poortinga, E.W., Hungerford, L.L. (1998). A case-control study of acute ibuprofen toxicity in dogs. *Prev. Vet. Med*., **35**(2): 115–124.

Rimer, D., Chen, H., Bar, Nathan, M., and Segev, G. (2022). Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome. *Journal of Veterinary Internal Medicine*, **36**(2): 609-618.

Ross, L. (2011). Acute kidney injury in dogs and cats. *Veterinary Clinics: Small Animal Practice*, **41**(1): 1-14.

Segev, G. (2011). Outcome prediction of acute kidney injury in dogs and cats. *Isr. J. Vet. Med*., **66**: 82-88.