CHRONIC POSTRENAL AZOTEMIA IN A DOG

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A six year old male, cryptorchid Labrador dog presented to the TVCC, CVAS, Bikaner with the symptoms of chronic vomiting, stranguria, progressive loss of body condition, dehydration. History revealed chronicity of more than two weeks. Hematology and serum biochemistry were indicative for chronic renal failure. The dog was treated with RL , 5% DNS , cefotaxime ,decadurabolin , lanthanium carbonate. ascorbic acid, methylcobalamin ,pyridoxine and nicotinamide. During three week of therapy, there was progressive worsening of renal function and ultimately leading to death of animal. Post mortem showed oversized testies possibly sertoli cell tumor, which possibly obstructing the urinary tract passage after the urinary bladder lead to post renal azotemia.
Keywords: Dog, Chronic Kidney Disease, Post renal azotemia

Introduction

When rupture or obstruction of the urinary tract prevents the normal collection and expulsion of urine from the body, the resulting azotemia is termed postrenal. Postrenal azotemia can coexist with prerenal and/or renal azotemia. Detection of postrenal azotemia requires attentiveness to the history and physical examination findings and to the results of specifically directed diagnostic tests. Prompt correction of postrenal causes of azotemia limits the potential for intrinsic renal damage and can contribute to a positive clinical outcome; therefore, postrenal azotemia should be investigated in all azotemic patients.(Fischer J *et al.*,2009).Accurate assessment of the origins of chronic azotemia i.e. pre- renal, renal, and postrenal is essential to the proper management of azotemic patients. However chronic renal failure (CRF) in dogs can be defined as azotemia of renal origin that is of more than 2 weeks of duration, which can affect 10% of dog population (Ahuja *et al.*, 2010).

Prerenal azotemia occurs when decreased renal perfusion results in a diminished glomerular filtration rate. Common causes of pre- renal azotemia include volume depletion, vascular collapse, thrombotic diseases and shock (cardiogenic, hemorrhagic, hypovolemic, or septic) (Julie *et al.*, 2009). Renal azotemia occurs when nephrons are directly damaged, most frequently by toxic, infectious, inflammatory, ischemic, or neoplastic processes. Postrenal azotemia is caused by urinary tract breach or obstruction or physical pressure. Any process distal to the renal tubules that interferes with the collection, containment, or excretion of urine can result in azotemia by preventing elimination of waste material in the urine, which can rapidly result in life-threatening fluid, electrolyte, and acid–base imbalances. Conditions causing prerenal and postrenal azotemia can also result in intrinsic renal damage if not identified and corrected. Postrenal azotemia can be acute or chronic, present communication is a case report of chronic postrenal azotemia in a dog.

Case history and clinical observations

A six year old male, cryptorchid Labrador dog presented to the TVCC, COVAS, Bikaner with the symptom of chronic vomiting, stranguria, progressive loss of body condition, dehydration. History revealed chronicity of more than two weeks. Hematology and serum biochemistry were indicative for chronic renal failure of stage 5 (Table-1).

Table- 1.Hematology and serum biochemistry of dog suffering from Azotemia

S. No	Parameters	Results on day 1	Results after 21 days
1.	Haemoglobin	9.2mg/dl	6.2 mg/dl
2.	WBC	17,500/cumm	12,200/ cumm
3.	Platelets	1.80lacs/cumm	1.6 lacs/ cumm
4.	TEC	1.57 million/cumm	1.23 million/cumm
5.	BUN	356.2	170.8 mg/dl
6.	S.Creatinine	9.2	13.8 mg/dl
7.	SGOT	85.1 U/L	80.80 U/L
8.	SGPT	155.2	160.4 U/L
9.	S.ALKP	232.2	80.2 U/L
10.	Serum Phosphorus	18mg/dl	12.00 mg/dl
11.	Serum Calcium	9.8 mg/dl	9.2 mg/dl

Treatment and discussion

Based on physical parameters of dehydration,intravenous fluid therapy was given with Ringer Lactate and Dextrose Normal Saline. Cefotaxime 25 mg /Kg Bwt.i/v bds, Decadurabolin @ 50 mg/week, i/m, and tab. Fosbait (Lanthanium carbonate) 500 mg, daily, PO was given to reduce blood urea nitrogen and phosphorus levels, respectively. Supportive therapy was done with ascorbic acid, 500mg/day, i/v, Neuroxin M (containing methylcobalamin 500 mcg,pyridoxine 50 mg and nicotinamide 50 mg/ml) @ 3 ml, i/v for seven days. During three week of therapy, there was progressive worsening of renal function(Table -1) but phosphorus level was reduced to 12 mg/dl due to use of tab.Fosbait and ultimately leading to death of animal. Post mortem showed oversized testis possibly sertoli cell tumor, which possibly obstructing the urinary tract passage after the urinary bladder lead to post renal azotemia in the present case. Gross examination of kidney was unremarkable. Post renal azotemia is caused by urinary tract breach or obstruction. Any process distal to the renal tubules that interferes with the collection, containment or excretion of the urine can result in azotemia by preventing elimination of waste material in the urine (Julie, 2009). Most lower urinary tract obstruction in dogs caused by either urolithiasis or neoplasia (Bjorling, 2003). Acquired obstruction can be due to intraluminal, extraluminal or intramural causes including urolith, nonmineralized material, trauma, inflammation, fibrosis, neoplasia and stricture etc. (Hardie and Kyles,2004). In cases of postrenal azotemia, the BUN and Creatinine ratio is more than 15(Drerhys,2012).

Conditions causing prerenal and postrenal azotemia can also result in intrinsic renal damage if not identified and corrected (Julie, 2009), which possibly happens in present case and leads to chronic renal failure (CRF). Destruction of nephrons decreases phosphorus filtration with a subsequent increase in serum phosphate, which stimulates PTH release from the parathyroid gland (Polzin, 2011). Hyperphosphatemia also decreases ionized calcium concentration, which stimulates PTH secretion. In a normal kidney and in early CRF, one effect of PTH is to decrease phosphate resorption in the proximal tubules so that more phosphate is excreted and serum phosphorus concentration is maintained within the normal range. However, as Chronic Kidney Disease (CKD) progresses and more nephrons become nonfunctional, a greater concentration of PTH is required to maintain serum phosphorus concentration and eventually hyperphosphatemia develops.

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