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Acute Post Renal Azotemia in a Cat

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ABSTRACT

Clinical, laboratory, ultrasonographic and radiologic examination of the 3-year-old-male-cat with anorexia, inactivity, abdominal distension, heamaturia and anuria in the last few days admitted to the the Selcuk University Veterinary Faculty Animal Hospital was made. Physical and laboratory examinations were made. As a result of examinations severe azotemia and penile urethral calculi were detected. The stone in the penile urethra was removed by lubricating the urethral lumen without any surgical intervention. Azotemia was corrected with supportive treatment. After the clinical relieve the patient was discharged from the hospital. In this case report, clinical, laboratory, ultrasonographic and radiologic findings and the treatment protocol of the cat were presented. In conclusion, it was emphasized that postrenal azotemia due to urethral obstruction in our case is common in domestic animals. If it is not diagnosed and corrected rapidly it can be life-threatening. In treatment, adequeta fluid treatment is essential and also ultrasonographic and radiological examinations are very important in diagnosis.

1. INTRODUCTION

Rapid and accurate assessment of the source of azotemia (prerenal, renal and / or postrenal) is essential for the appropriate treatment of azotemic patients. Prerenal azotemia develops due to decreased glomerular filtration rate (GFR) which is associated with reduced renal perfusion, fluid loss, vascular collapse, thrombotic diseases and shock (cardiogenic, hemorrhagic, septic or hypovolemic). Renal azotemia develops due to direct damage to nephrons by toxic, inflammatory, infectious, ischemic or neoplastic causes. Postrenal azotemia develops due to obstruction of the urinary system or traumatic ruptures (Fischer et al., 2009). Azotemia develops if urinary collection, retention and excretion are prevented due to any mechanism from distal to renal tubules. When elimination of waste materials in the urine is prevented, life-threatening fluid, electrolyte and acid-base imbalances occur (Bjorling, 2003).

Obstruction of the lower urinary tract (LUTS) is a common urological condition that causes severe azotemia in cats. LUTS obstruction is usually detected by physical examination and painful palpation of the bladder and imaging techniques. Most LUTS obstructions occur in cats due to uroliths and urethral mucocrystalline plugs (Westropp et al., 2005).

Calcium oxalate (CaOx) and ammonium urate stones are the most common uroliths in urethral obstruction due to their relatively small sizes predisposition to multiple formation (Fischer et al., 2009). The incidence of CaOx uroliths has increased significantly over the last 10 years. This mineral is the component of more than 90% of the analyzed nephrolites and uroliths (Bartges et al., 2004). Many factors can influence the formation of CaOx stones. These are; saturation of calculogenic minerals in urine, crystal clustering, crystallization and urinary factors that trigger the growth of these crystals (Bartges and Kirk, 2006).

When an azotemia is identified, anamnesis and physical examination are often helpful in determining initial evidence that the cause may be postrenal. Anorexia, weight loss, lethargy, stagnation, uremic or stinky breath are common with uremic symptoms in patients with severe postrenal azotemia (Fischer, 2005). Clinicopathological sequelae due to postrenal nitrogenemia vary from mild to fatal. As the kidneys are the main organs that remove phosphate, potassium, and other soluble substances as well as nitrogenous components early disorders such as increase in blood urea nitrogen (BUN), creatinine, phosphorus and if there is a complete obstruction hyperkalemia. In complete obstructions, normal excretion of hydrogen is prevented and metabolic acidosis develops which is characterized by low bicarbonate level, negative base deficit and low pH in blood biochemical profile (Fischer et al., 2009). Urinary analysis findings vary in cats with postrenal azotemia. Since intrinsic renal damage does not always develop, urine may be diluted or concentrated. Hematuria is common in cats with LUTS obstruction (Weisse et al., 2002).

LUTS obstruction causes increased pressure in the bladder and proximal urethra. If this pressure is too strong or if it is not relieved for a long time, it may enter the upper urinary system. Urethral or trigonal mucosal damage and edema may develop in the area of obstruction. This type of damage stimulates cell infiltration by inflammatory leukocytes. Urethral damage due to obstruction, inflammation, submucosal hemorrhage, perivascular aggregation of inflammatory cells and if the dissolution of the obstruction fails for more than 10 hours it may cause urothelial necrosis of the bladder and systemic infection. In addition, excessive distension of the bladder causes hypoxic damage in tight junctions between detrusor myocytes. This neuromuscular damage causes the formation of postobstructive detrusor atony (Seki et al., 1992).

Case Presentation

In the anamnesis, it was learned that the cat was not neutered, fed with commercial dry food, had no history of urinary tract infection, and had no appetite, stagnation, and unable to urinate for the last 3 days. Physical examination revealed dehydration, hypothermia (35.8 ºC), bradipnea (32) and bradycardia (64), abdominal tension, pain and hematuria (Figure 1A). Following physical examination, blood gas (Radiometer ABL 90 Automatic analyzer, Model 5700, 74351, USA), hemogram (MS4 CFE 279, France), serum biochemistry (BT 3000 Plus, Biotecnical Inc. SPA, Rome, Italy), urine dipstick (URIT) -31, China) and sediment (Olympus Cx21i light microscope, x40 magnification) analysis, ultrasonographic and radiological examinations were performed. In addition, cystocentesis was performed with a 22G needle from the collum region of the urinary bladder for dipstick analysis and decompression. Blood gas analysis showed metabolic acidosis, severe hyperkalemia and hypochloremia, and hemogram showed leukocytosis and polycythemia (Table 1).

On the ultrasonographic examination, thickening of the bladder wall (0.42 cm) (Figure 2) and urethral calculi (Figure 3) were detected.

Urine dipstick analysis showed leukocyturia and proteinuria (Table 3).

Table 1. Blood gases and haemogram findings

Blood gases	Value	Referance range	Haemogram	Value	Reference range
pH	7.185	7.35 – 7.45	WBC (m/mm^3)	21.64	5.0 – 19.0
K (mmol/L)	8.3	3.4 – 5.6	RBC (m/mm^3)	15.64	4.0 – 9.0
Lactate (mmol/L)	6.8	0 - 2	MCV (fl)	43.9	35.5 – 55.0
Hct (%)	54.1	29 - 48	Hct (%)	68.6	24.0 – 45.0
BE(ecf) (mmol/L)	-8.1	-4 - 4	MCH (pg)	11.7	16.0 – 24.0
BE (B) (mmol/L)	-8.8	-4 - 4	MCHC (g/dl)	26.6	28.0 – 40.0
HCO_3 (P,st) (mmol/L)	16.1	19 - 24	RDW	12.1	8.0 – 12.0
HCO_3 (P) (mmol/L)	20.1	19 - 24	THR # (m/mm^3)	183	120 - 500

pH: Potential hydrogen. K: Potassium. Hct: Hematocrit. BE: Base excess. HCO_3: Bicarbonate.

Serum biochemistry revealed severe azotemia and hyperphosphatemia (Table 2).

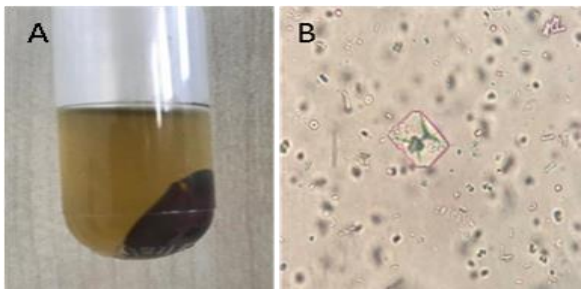

Figure 1. Haematuria (A) and CaOx crystals in urine sediment

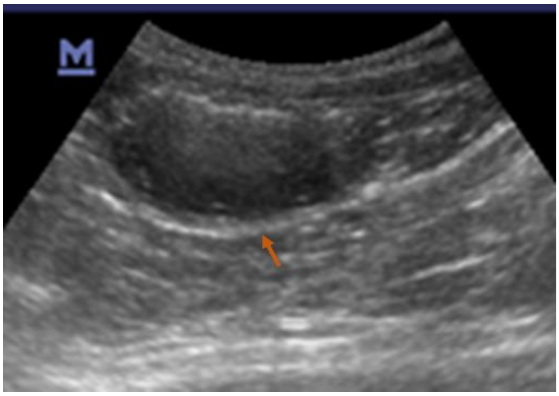

Figure 2. Thickening of the bladder wall (Orange arrow, 0.42 cm)

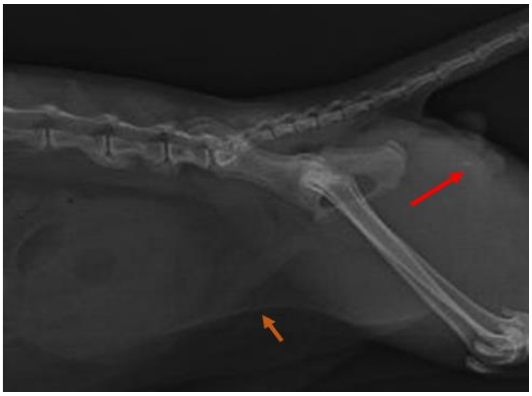

Figure 3. Urethral calculi (Red arrow) and distention in bladder (Orange arrow)

Table 2. Serum biochemistry findings before treatment

Parameters	Referance range	Values Before Treatment
BUN	14 – 36	**117.4 mg/dL** ↑
Creatinine	0.6 – 2.4	**5.0 mg/dL** ↑
Amylase	100 – 1200	1014 U/L
Glucose	64 – 170	77 mg/dL
Magnesium	1.5 – 2.5	2.2 mg/dL
LDH	20 – 500	350 U/L
Phosphorus	2.4 – 8.2	**13.1 mg/dL** ↑
Cholesterol	75 – 220	175 mg/dL
Albumin	2.5 – 3.9	2.6 g/dL
Protein	5.2 – 8.8	3 g/dL

BUN: Blood urea nitrogen. LDH: Lactate dehydrogenase.

Following decompressive cystocentesis, the urethral lumen was lubricated with 0.9% NaCl isotonic solution at body temperature and the stone was removed without urethrostomy. To correct the azotemia, the cat was hospitalized for 3 days and treated with 0.9% NaCl isotonic solution, 60 ml / kg, IV, sodium bicarbonate (Carbotek, Teknovet®) IV, vitamin-amino acid supplement (Duphalyte, Zoetis®) 5 ml / kg, IV, ranitidine (Ragacid, VEM Drug®) 4 mg / kg, IV, meloxicam (Maxicam, Sanovel®) 0.3 mg / kg, SC, enroflaksasin (Baytril-K 5%, Bayer®) 5 mg / kg kg, IV, nitrofurantoin (Pyeloseptyl, Biofarma®) 4 mg / kg, PO was administered. After the treatment biochemical analysis was performed again and it was determined that the azotemia parameters returned to normal limits (Table 4). The patient was discharged after improvement of clinical findings.

Table 3. Urine dipstick analysis

Urine dipstick analysis	
Leukocyte	+1 cell/uL
Ketone	- mmol/L
Nitrit	-
Urobilinogen	Normal
Bilirubin	0 umol/L
Glucose	+/- mmol/L
Protein	+3 >3 g/L
Specific gravity	1.020
pH	5.0
Blood	+2 80 cell/uL

pH: Potential hydrogen
Urinary sediment analysis revealed CaOx crystals (Figure 1B).

Table 4. Serum biochemistry findings after treatment

Parameters	**Referance range**	**Values After Treatment**
BUN	14 – 36	25.4 mg/dL
Creatinine	0.6 – 2.4	2.2 mg/dL
Amylase	100 – 1200	423 U/L
Glucose	64 – 170	84 mg/dL
Magnesium	1.5 – 2.5	2.3 mg/dL
LDH	20 – 500	344 U/L
Phosphorus	2.4 – 8.2	6.1 mg/dL
Cholesterol	75 – 220	189 mg/dL
Albumin	2.5 – 3.9	2.7 g/dL
Protein	5.2 – 8.8	3.2 g/dL

BUN: Blood urea nitrogen. LDH: Lactate dehydrogenase.

4. DISCUSSION AND CONCLUSION

Acute postrenal azotemia is a common urological condition requiring immediate intervention. It can be fatal especially if it is not diagnosed and corrected early in cats. The most common causes of postrenal azotemia in small animals are ureteral or urethral obstruction and traumatic urinary system ruptures. Azotemia due to postrenal causes can be treated when intrinsic damage does not occur in the kidneys (Ross et al., 1999). As a result, when azotemia is detected, anamnesis, physical examination and laboratory findings should be supported by diagnostic imaging such as x-ray and USG. In this way, the source of the postrenal azotemia in the urinary system can be easily identified and with appropriate fluid treatment azotemia can be corrected.

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