Urethral Obstruction

A PowerPage Presented By

Urethral obstruction is a life threatening emergency in dogs and cats and frequently involves important electrolyte and metabolic alterations with significant cardiovascular and renal implications. This is a common topic seen on boards as the presentation and treatment of these animals requires you to consider renal and cardiac physiology and to have knowledge of the relevant pharmacology to make proper decisions. This condition is not usually a diagnostic challenge but can be complex to manage; therefore, this page describes the key clinical features and management options.

Key Points

- **Most commonly** occurs in **male cats**. Rare in female cats. Occurs in male dogs more often than female dogs, likely due to the more complicated path and narrow lumen of the male urethra
- Can occur from physical obstruction (uroliths, neoplasia, mucosal plugs, stricture) or mechanical obstruction (severe urethral spasm)
- Hyperkalemia is the most important and life-threatening electrolyte disturbance
- Can have severe hypovolemia and cardiovascular compromise. May also present with azotemia, metabolic acidosis, hypocalcemia, and hyperphosphatemia

Key Clinical Features

Hyperkalemia:

- Occurs due to decreased renal potassium excretion and extracellular shift due to acidosis
    - Has effects on cell resting membrane potential, most significantly on cardiac myocytes, eventually making cells unable to depolarize
    - Causes classic ECG changes of:
        - **Bradycardia**, **tall tented T waves**, **widened QRS** complexes, **decreased to absent P** wave, prolonged P-R interval, shortened Q-T interval. (See www.vetgo.com/cardio/concepts/concsect.php?conceptkey=20115#20115)
        - Severity of ECG changes & clinical signs does not correlate well to magnitude of hyperkalemia

- **TREATMENT** OF HYPERKALEMIA:
    - IV fluids (e.g. 0.9% NaCl) will have dilutional effect to lower serum potassium
    - IV calcium gluconate (50-100 mg/kg) to restore the membrane potential and re-establish normal depolarization. **Does not lower K^+!**
    - IV dextrose and regular insulin to move potassium back into cells. Never give insulin alone, this can result in hypoglycemia and seizures!
    - Sodium bicarbonate (0.3 x base deficit x weight in kg…typically give 1/3-1/2 this amount)
        - This results in shifting potassium back into cells as pH increases

Metabolic Acidosis

- Occurs secondary to inability of the kidneys to excrete hydrogen ion, can be severe (ph< 7.0)
    - Results in respiratory compensation (increased RR and/or increased tidal volume)
    - Predisposes to cardiac arrhythmia, reduced catecholamine responsiveness and CNS depression

- **TREATMENT** OF METABOLIC ACIDOSIS
  - Re-establishing GFR by decompressing the bladder
  - IV fluid therapy
  - Sodium bicarbonate (0.3 x base deficit x weight in kg…typically give 1/3-1/2 this amount). Typically reserved for severe acidosis (pH < 7.1, HCO_3 < 10 mmol/L)

Azotemia/hyperphosphatemia

- Occurs due to decreased renal excretion of urea and other waste products
  - Contributes to CNS depression and other signs

- **TREATMENT** OF AZOTEMIA/hyperphosphatemia
  - Re-establishing GFR by decompressing the bladder/relieving obstruction
  - IV fluid diuresis

Hypocalcemia

- Can see severe ionized hypocalcemia secondary to hyperphosphatemia (law of mass action)
  - Further compromises neuromuscular excitation (can result in seizures) and cardiac contractility

- **TREATMENT** OF HYPOCALEMIA
  - IV calcium gluconate (50-100 mg/kg)

Initial Plan of Action and Treatment

- Tentative diagnosis is made on clinical presentation of straining to urinate, depression, and a distended, turgid bladder
- If possible, perform PCV/TP, blood gases with electrolytes, ECG, blood glucose and BUN, BP

1. Rehydration with potassium free fluids (0.9% NaCl) is essential *(There is some evidence that other isotonic crystalloids containing potassium may be just as effective but on boards, the best choice would be NaCl)*
2. Address life threatening electrolyte abnormalities by giving calcium, insulin and dextrose, and/or sodium bicarbonate
3. Relieve the bladder obstruction, typically by use of a urinary catheter to retropulse the obstruction back to the bladder. Appropriate sedation/analgesia is important to decrease risk of urethral trauma. Then, place an indwelling catheter to prevent reobstruction and to monitor urine output
4. Expect profound post-obstructive diuresis and monitor urine output and fluid rates. Urine production can exceed 50-100 ml/hr so important to keep up with IV fluids
5. Once patient is stabilized, identify and address the underlying cause of the obstruction, consider x-rays, abdominal ultrasound, urinalysis and urine culture
6. Continue appropriate analgesia and sedation
7. Urinary catheter left in place until azotemia, electrolyte abnormalities, and post-obstructive diuresis resolved. Observe 12-24 after catheter removal to ensure spontaneous urination

References and Links

Ettinger, Feldman, Textbook of Veterinary Internal Medicine, 5th ed pp. 94-97

Plunkett, Emergency Procedures for the Small Animal Veterinarian, 2nd ed pp. 224-227

On-line VIN Proceedings Notes

Canine and Feline Urethral Obstructions:
http://www.vin.com/Members/Proceedings/Proceedings.plx?CID=wvc2003&PID=pr03588&O=VIN

Urinary Obstruction: Can't Pee, Won't Pee:
http://www.vin.com/Members/Proceedings/Proceedings.plx?CID=bsava2007&PID=pr16412&O=VIN