LOWER URINARY TRACT EMERGENCIES AFTER TRAUMA

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Introduction:
Lower urinary tract emergencies are common reasons for small animal patients to be presented to their veterinarians. The following pages will focus on the diagnosis and management of cats and dogs with lower urinary tract disruption after trauma. The principles described below may also aid in the management of other lower urinary tract emergencies as well.

Hyperkalemia:
The cause of death in dogs and cats with lower urinary tract disruption is NOT azotemia or the inability to urinate. The cause of death in these patients is hyperkalemia. As a result, the presence of hyperkalemia must be rapidly identified and life-saving treatment must be immediately instituted. Identification of hyperkalemia begins with the physical examination and is supported by historical findings (trauma in the case of urinary tract disruption). Animals with lower urinary tract disruption may demonstrate abdominal pain, presence of a fluid wave, and possibly inappropriate bradycardia (depending on when the trauma occurred and the presence of concurrent injuries).

Rapid supporting evidence for the presence of life threatening hyperkalemia may be acquired using the ECG. Electrocardiographic findings supportive of hyperkalemia include bradycardia, spiking or tenting of the “T” wave, prolongation of PR interval, flattening or absence of the “P” wave, and widening of the “QRS” complex. Hyperkalemia is definitively diagnosed through blood analysis. Always consider the effect of the hyperkalemia on the patient, rather than the absolute severity of the hyperkalemia when considering optimal management strategies. For example, a dog with a heart rate of 135 and normal perfusion and a potassium of 8.5mmol/L may not need treatment for hyperkalemia beyond fluid therapy while another animal with a potassium of 8.5mmol/L may demonstrate severe bradycardia and other ECG manifestations of hyperkalemia requiring emergency administration of a rapid bolus of IV fluids, calcium gluconate administration, as well as additional therapies described below.

Management of life threatening hyperkalemia due to lower urinary tract disruption does NOT begin with simply draining the abdomen of urine. This approach will take hours to be effective. Instead, the two most important therapies for life-threatening hyperkalemia include rapid infusion of 0.9%NaCl (or a balanced electrolyte solution such as Normosol-R or LRS) and administration of calcium gluconate. Saline administration will restore euvoolemia while diluting potassium concentrations and calcium gluconate will protect the heart from the effects of the hyperkalemia by restoring the potential difference between resting membrane potential and threshold potential. Please see Table 1 for a summary of the management of hyperkalemia.
**Table 1: Management of Life Threatening Hyperkalemia**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Onset Time</th>
<th>Dose</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid Therapy (0.9% NaCl).</td>
<td>Immediate</td>
<td>As needed for rapid volume expansion and rehydration.</td>
<td>Dilution is mechanism of action. Often delivered as a bolus.</td>
</tr>
<tr>
<td>Calcium Gluconate 10%</td>
<td>Immediate</td>
<td>20-60mg/Kg IV over 1-3min</td>
<td>Cardioprotective. Will not lower potassium concentration.</td>
</tr>
<tr>
<td>Regular Insulin and Dextrose</td>
<td>20-40min</td>
<td>0.1-0.25unit/Kg Regular Insulin and 0.5g/Kg Dextrose</td>
<td>Redistributes potassium intracellularly. Monitor blood glucose at 30 and 60min to assess for hypoglycemia. Alternatively, dextrose only can be administered IV relying on endogenous insulin release.</td>
</tr>
<tr>
<td>Sodium Bicarbonate</td>
<td>20-40min</td>
<td>0.1 x Body Weight (Kg) x Base Deficit over 20minutes</td>
<td>Redistributes potassium intracellularly. Rarely utilized unless life threatening metabolic acidosis (pH&lt;7.1) is also present.</td>
</tr>
<tr>
<td>Urinary Diversion</td>
<td>60min- Ongoing</td>
<td></td>
<td>Urinary catheterization (for obstruction and disruption) and placement of a multifenestrated abdominal catheter (for urinary disruption) will divert urine externally.</td>
</tr>
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**Lower Urinary Tract Disruption:**
Uroperitoneum must be considered as a differential diagnosis in all patients with abdominal pain after trauma and those that have historical findings consistent with urinary obstruction. Uroperitoneum results from disruption of the urinary system at any level (kidney, renal pelvis, ureter, bladder, urethra) although disruption of the bladder or urethra are by far the most common. Clinical signs commonly observed in dogs and cats with uroperitoneum include abdominal pain, abdominal distention, decreased or absent urination, lethargy and vomiting. Disruption of a ureter, kidney, or renal pelvis without disruption of the retroperitoneum may result in
uroretroperitoneum. Clinical signs commonly observed in dogs and cats with uroretroperitoneum include abdominal pain and distention and hematuria.

Dogs and cats with uroperitoneum may demonstrate bradycardia if hyperkalemia is present. Initial laboratory abnormalities may include acidosis, azotemia, and hyperkalemia. Lactic acidosis may be present due to concurrent perfusion abnormalities.

Uroperitoneum is confirmed based on abdominal fluid evaluation. Abdominal fluid may be collecting via routine abdominocentesis, four quadrant abdominocentesis, ultrasound-guided or abdominocentesis, or from samples acquired via placement of an abdominal drain. Uroperitoneum causes a chemical peritonitis resulting in a significant inflammatory response that is observed cytologically. Evaluation of the ratios of creatinine and potassium in the abdominal fluid to that in the blood (with identical sample handling) is useful in diagnosing the presence of uroperitoneum. A ratio of CREA_Abd to CREA_B of 2.0 or greater is highly supportive of uroperitoneum. A ratio of K_Abd to K_B of 1.4 or greater (dog) and 1.9 or greater (cat) is further supporting evidence for the presence of uroperitoneum. After confirmation is made, localization of the lesion(s) must be accomplished through retrograde urethrocystography and/or excretory urography. The author routinely performs an abdominal radiograph in these patients. If the retroperitoneum appears normal, the urinary tract injury is most likely bladder or urethra and retrograde urethrocystography is performed. If the retroperitoneum lacks detail or there is ventral deviation of the colon, then both retrograde urethrocystography and excretory urography are performed.

Surgical management of urinary tract disruption is specific to the underlying pathology. Animals that have a disrupted bladder usually undergo debridement of the edge of the perforation followed by single layer closure using a monofilament absorbable suture such as Monocryl (Ethicon Inc., Somerville, NJ) in an appositional pattern. A urinary catheter may be left in place for 24-48 hours (or until the patient is mobile and able to walk outside to urinate frequently) to prevent bladder distention. Proximal urethral disruption may be repaired in a similar fashion to bladder repair, however, it is recommended that a urinary catheter be placed to help ensure that the lumen of the urethra is not compromised significantly by the repair. This urinary catheter should remain in place for approximately 7 days to help prevent luminal narrowing associated with the healing process. Pelvic urethral disruption is more challenging and may be managed conservatively if a urinary catheter can be placed across the lesion and kept in place for 10-14 days. If this is not possible via a retrograde or antegrade (surgical or fluoroscopic) approach, then open surgical management will be necessary. A temporary procedure that would accomplish the goal of urinary diversion is placement of a surgical cystostomy tube while considering referral to a surgeon for exploration of the pelvis and primary repair of the lesion.

Suggested Reading