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HOW DO I APPROACH A PATIENT WITH UROABDOMEN: FROM THE INITIAL DIAGNOSIS TO PATIENT'S STABILISATION

Uroabdomen is a common cause of acute abdomen in canine and feline patients. It results from urine accumulation in the abdominal cavity or retroperitoneal space (or both) secondary to a rupture in the urinary tract. Uroabdomen needs to be promptly diagnosed and adequately managed, as if unrecognised, it might result in life-threatening electrolyte alterations, metabolic acidosis and hypovolaemia.

Uroabdomen in dogs and cats is most often associated to road traffic accident and secondary blunt trauma to the abdomen and pelvis. The bladder (or urethra especially in male dogs) is usually the most common site of rupture. Other nontraumatic reported causes of uroabdomen include: spontaneous bladder rupture secondary to urethral obstruction, bladder neoplasia, manual bladder expression, cystocentesis, urethral catheterization, genitourinary tract surgery, or accidental injury to the urinary tract during abdominal surgery.

Independently from the underlying cause, uroabdomen results in severe dehydration and hypovolaemia from fluid loss in the abdomen, life-threatening hyperkalaemia, metabolic acidosis, severe azotaemia and chemical peritonitis. Dehydration and hypovolaemia in patients with uroabdomen results from a combination of fluid shift in the abdomen, vomiting and anorexia/adypsia. Fluid loss in the abdominal cavity occurs because urine that accumulates in the abdomen is hyperosmolar compared to the extracellular fluid compartment (ECF). This causes a shift of fluid and electrolytes from the ECF to the abdominal cavity via the peritoneal membrane. Solute like natrium and chloride are more concentrated in the ECF compared to the abdominal effusion, so following their concentration gradient, they will accumulate in the abdominal effusion; solute like creatinine, urea and potassium are more concentrated in the abdominal effusion compared to the ECF so they will slowly equilibrate with time and their concentration in the ECF increase, leading to azotaemia and hyperkalaemia.

A suspected diagnosis of uroabdomen can be made based on patient history, physical examination findings, blood work and acid base results and survey radiography. In dogs and cats that suffered trauma often the signs of urinary tract rupture are overlooked due to the presence of concomitant major injury to the cardiovascular and respiratory tract. In these patients suspect of urinary tract rupture/uroabdomen should be considered if there is severe bruising of the ventral abdominal wall or of the inguinal region, if the patient shows signs of pollakiuria/stranguria, haematuria, decreased urinary production of oliguria, severe abdominal pain and distension and in a patient that develops azotaemia and hyperkalaemia after a trauma. Physical examination of the patient with uroabdomen might reveal tachycardia, bradycardia or arrhythmias, severe abdominal pain and a possible positive ondulation. Remember that a palpable bladder (also after trauma) as well as the presence of urinary production doesn't rule out the presence of uroabdomen. Loss of serosal details on survey abdominal radiographs may suggest intraabdominal fluid accumulation; however, survey radiographs are not specific for uroabdomen nor will they clarify the site of disruption. If available, the use of ultrasonography (Abdomen Focused Assessment Sonography for Trauma A-FAST) provides a much more sensitive and rapid means of detecting peritoneal fluid and perform an abdominocentesis. Abdominal effusion collected via abdominocentesis (blind or ultrasound guided) should always be analysed (haematocrit, total solid, creatinine and potassium) and the values of creatinine and potassium in the abdominal effusion compared with those in the serum. In cats a mean abdominal fluid:serum creatinine ratio of 2:1 and a mean abdominal fluid:serum potassium ratio of 1.9:1 has been reported to be diagnostic for uroperitoneum. A retrospective study evaluated the abdominal fluid: peripheral blood creatinine and potassium ratios in dogs showed that 85% of dogs with uroperitoneum had an abdominal fluid:serum creatinine ratio greater than 2:1 and 100% of the dogs had an abdominal fluid:serum potassium ratio greater than 1.4:1 . Positive contrast studies of the urinary tract are also necessary to confirm the diagnosis and localize the site of the lesion, however they should be performed only in a cardiovascular stable patient when the metabolic derangements have been corrected. Because injuries to the lower UT are reported to be more common in small animals, cystography and retrograde urethrography are generally performed first. Excretory urography should be performed if there is a suspicion of fluid accumulation in the retroperitoneal space , to better evaluate the integrity of the kidneys and ureters. The initial treatment of the patient with uroabdomen should focus on fluid resuscitation, adequate analgesia, correcting electrolytes and metabolic derangements and on obtaining urinary diversion. Hypovolaemic shock should be treated with the use of isotonic balanced crystalloid infusion. A buffered solution like ringer

lactate, Hartman's solution and/or Sterofundin Iso is the preferred solution for initial resuscitation by the author as it is more balanced and although containing minimal amount of potassium, will facilitate volume expansion and resolution of the metabolic acidosis when compared to the use of NaCl 0.9% . The fluid should be administered in boluses titrated to the patient's clinical status and the severity of the hypoperfusion. Adequate analgesia should be addressed in the initial phase of the resuscitation with the use of a pure μ opioid (methadone, sulfentanil, morphine) and the therapy titrated till the patients is more comfortable. An initial dose of 0.1-0.2mg/kg methadone followed by a constant rate infusion of sulfentanil (0.3-0.5ugr/kg/hours) is the preferred approach of the author. Further multimodal analgesia could be considered with the use of constants rate infusion of ketamine and in dogs of lidocaine. A non steroidal anti-inflammatory drugs should never be used as initial analgesic in an cardiovascular and metabolic unstable patients.

In patients with bradycardia or with elevated potassium an ECG should always be performed to evaluate any ECG changes associated to hyperkalemia. Most common ECG changes include peaked T waves, widening of the QRS complex, flattening and eventual loss of the P wave, merging of the QRS complex with the T wave, atrial standstill, ventricular fibrillation, or asystole. Strategies to treat hyperkalemia should consider drug therapy to shift potassium intracellularly, reducing myocardial sensitivity to potassium, and promoting urinary output. If the patients present ECG changes associated with hyperkalemia, to protect myocardial function while initiating other therapy to lower potassium, calcium boro gluconate (10% solution at 0.5-1ml/kg) may be given slowly intravenously (15-30 minutes). It is important to remember that this treatment doesn't lower serum potassium concentration, but makes the myocardium less sensitive to the effects of hyperkalemia until other therapy is initiated. Treatment to lower serum potassium , besides volume expansion with fluid therapy and stimulated glomerular filtration rate and potassium excretion via the urine, consist in facilitate an intracellular shift of potassium from the ECF to the ICF compartment. It includes the administration of intravenous regular insulin at 0.1-0.2 IU/kg, followed by intravenous bolus of dextrose at 1 gram for each unit of insulin administered. Because the insulin often works longer than the bolus of glucose it is important in these patients to monitor the glucose regularly and to maintain them on a isotonic crystalloid solution containing 5% glucose. The administration of sodium bicarbonate at 2-3mEq/kg in 30 min- 1 hour could be used to cause a mild metabolic alkalosis that will allow the potassium to shift from the plasmatic compartment into the intracellular space.

After fluid resuscitation and initial stabilisation of hyperkalaemia and metabolic abnormalities, drainage of urine from the abdomen and urinary diversion should be established. This is often obtained with a percutaneous placement of a peritoneal drainage catheter to allow removal of urine from the abdominal cavity and with the placement, when possible, of an indwelling urinary catheter to keep the bladder empty and decrease the possibility of more urine accumulating in the abdominal cavity. If a urethral catheter cannot be passed due to obstruction or damage to the urethra the placement of a cystostomy tube should be considered . Different catheters are available for peritoneal drainage; they can often be placed with mild opioid sedation and local anaesthesia.

It is important to remember that the presence of urine in the abdominal cavity is not an indication for an immediate exploratory laparotomy as long as drainage and urinary diversion is established. The repair of urinary tract damage should be considered only when the patient is stabilized.

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