Medical management of uroabdomen in cats

Cecilia Stilwell BVMS MRCVS, intern at Dick White Referrals, UK, discusses the treatment of the accumulation of urine within the abdominal cavity following injury to the urinary tract in cats

Uroabdomen is most commonly caused by trauma, with the bladder being the most common site of rupture. Urine in the abdominal cavity causes an azotaemia, metabolic and electrolyte derangements and cardiac disturbances, which can become life-threatening. Medical stabilisation is imperative before definitive surgical correction can be considered. The prognosis for these patients improves in the absence of concurrent injuries and with rapid stabilisation. It is, therefore, essential that the veterinarian and veterinary nurses work closely to identify and stabilise these patients quickly and effectively.

INTRODUCTION

Uroabdomen is a medical emergency which requires rapid recognition and implementation of therapy. Uroabdomen is the accumulation of urine within the peritoneal cavity, retroperitoneal cavity or both, secondary to rupture of the urinary tract. Uroperitoneum is most commonly encountered. This condition occurs following loss of integrity to the lower urinary tract (distal ureters, bladder or proximal urethra). In comparison, uroretroperitoneum will develop following injury to the kidney or proximal ureter.[1] When urine accumulates in the peritoneal or retroperitoneal cavities severe electrolyte and metabolic changes can occur. If left untreated these derangements will have harmful effects on the cardiac and renal function of the cat and can become life-threatening.

This review focuses of the common causes of uroabdomen in cats and discusses the approach to diagnosis and subsequent medical stabilisation of these cats.

WHAT ARE THE CAUSES OF UROABDOMEN?

There are numerous causes of uroabdomen, these can be traumatic or non-traumatic in origin (see Table 1). Uroabdomen in cats is most often associated with trauma, for example blunt or vehicle trauma. In these cases, the urinary bladder is most common site of rupture.[2,3]

Trauma
- » Blunt
- » Vehicles

Iatrogenic
- » Surgical (urogenital or abdominal)
- » Urethral catheterisation
- » Cystocentesis
- » Manual expression

Obstruction
- » Urolith in the kidney, ureter or urethra
- » Pelvic masses
- » Bladder neoplasia

Table 1: Causes of uroperitoneum.

Non-traumatic causes of a uroperitoneum include: obstruction (urolith, neoplasia), iatrogenic (manual bladder expression, cystocentesis, urethral catheterisation or during abdominal surgery), urogenital surgical complications (leakage from surgical site, dehiscence, obstruction secondary to post-operative swelling).[1,5,2,7] Iatrogenic urinary tract rupture is the most common non-traumatic cause of uroabdomen.[3]

HOW IS UROABDOMEN DIAGNOSED?

A diagnosis of uroabdomen is based on the history, clinical examination findings, laboratory evaluation and diagnostic imaging studies.

HISTORY

It is important to ascertain whether the cat has a history of trauma, feline lower urinary tract disease, urolithiasis, recent abdominal or urogenital surgery.

CLINICAL EXAMINATION

The severity of clinical signs will depend on the duration of the uroabdomen. The possibility of uroabdomen should be considered in any patient with a history of trauma.

The clinical signs include: lethargy, anorexia, vomiting, stranguria, haematuria, abdominal distension with a palpable fluid thrill and severe abdominal pain, which may present as aggression.[1,4,5] In severe cases, the cat may present in hypovolaemic shock. It is important to be aware that cats may present differently to dogs. Cats can be tachycardic or bradycardic with pale mucous membranes, weak pulses and cool extremities. They may also be tachypnoeic, hypothermic and show signs of generalised weakness and/or decreased mentation.[6]

Note that the presence of a palpable bladder and an ability to urinate does not exclude the possibility of a ruptured urinary tract and subsequent uroabdomen.[2]

LABORATORY

All cats with a suspected uroabdomen should have blood taken for analysis. The initial tests should include a complete blood count, electrolytes, serum biochemistry and acid-base analysis.[1,5,7] The most common abnormalities encountered with uroabdomen are hypovolaemia, raised urea and creatinine (azotaemia), metabolic acidosis and electrolyte disturbances (mild to marked hyperkalaemia, mild hyponatraemia, hyperphosphataemia, hypocalcaemia).[1,4,7]

If an abdominal effusion is identified, a sample of the effusion should be collected. An abdominocentesis can

be performed blind or under the guidance of ultrasound.[1] The biochemical and cytological characteristics of this fluid should be examined. A definitive diagnosis of uroabdomen is made by comparing the creatinine and potassium levels within the serum to the abdominal fluid. In cats, a 2:1 ratio of abdominal fluid creatinine to serum creatinine and 1.9:1 ratio for abdominal fluid potassium to serum potassium is considered diagnostic for uroabdomen.[1, 2] On cytology, given urine is a chemical irritant, a non-septic neutrophilic inflammatory process is often encountered. Cats will have an increased risk of septic peritonitis if they had bacteriuria, from a pre-existing urinary tract infection. It is therefore important to evaluate the slide closely for evidence of bacteria.[7] Uroabdomen can be confirmed if the concentration of creatinine in the abdominal effusion is ≥2 times the serum creatinine concentration and the concentration of potassium in the abdominal effusion is >1.9 times the serum potassium concentration.

DIAGNOSTIC IMAGING

Following initial stabilisation and laboratory evaluation, abdominal imaging can be performed to identify the location of urinary tract injury. The possible imaging modalities include: abdominal radiographs, abdominal ultrasound and computed tomography.[7] The modality of choice will depend on cost, availability and experience. In veterinary medicine, radiography is often the first choice. Survey radiographs of the abdomen are taken first (see Figure 1). These may reveal a generalised loss of serosal detail, suggestive of an abdominal effusion. Occasionally, free gas may also be present within the abdominal cavity. This is more commonly associated with distal urinary tract rupture, at the level of the urethra. The survey radiographs may also reveal the presence of uroliths within the urinary tract (see Figure 2).

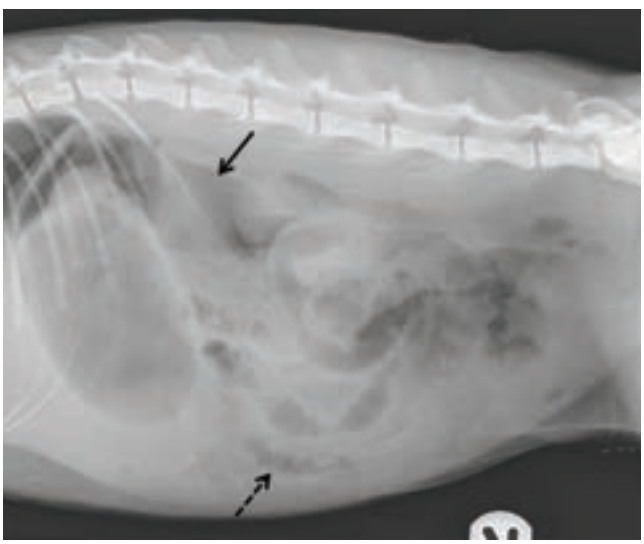

Figure 1: Right lateral survey abdominal radiograph of a cat. The generalised loss of serosal detail is consistent with a peritoneal effusion. The small intestine is corrugated with a mildly gas-distended lumen (dotted black arrow). Corrugation can be seen with peritonitis. There is also free gas within the abdomen (black arrow).

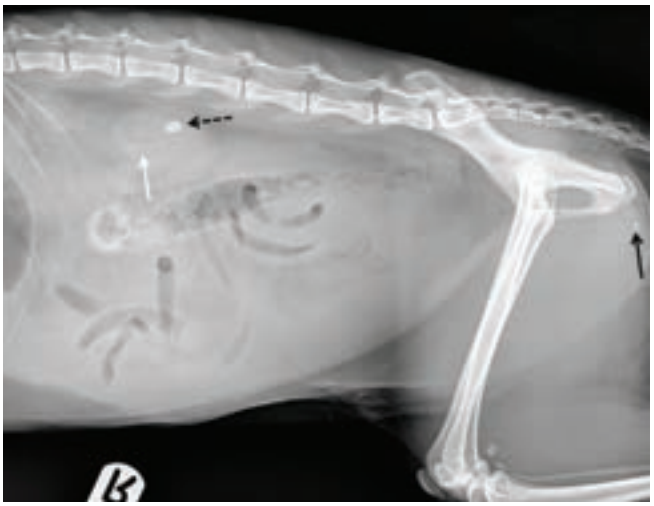

Figure 2: Right lateral survey abdominal radiograph of a cat. There is reduced serosal detail, consistent with peritoneal effusion. A nephrolith is superimposed over the left kidney/left ureter (dotted black arrow). Small nephroliths/ calcifications are superimposed over the more cranially located right kidney (white arrow). There is also a urethrolith present in the distal urethra (black arrow).

Positive-contrast radiography is subsequently used to identify the exact site of urine leakage. To aid interpretation of the urogenital tract it is advisable to perform an enema to evacuate the colon. A retrourethrocystogram (see Figure 3) is useful for highlighting leaks within the lower urinary tract (urethra, urinary bladder) and is often the method of choice in the majority of clinical settings. On the other hand, intravenous urography will allow identification of a

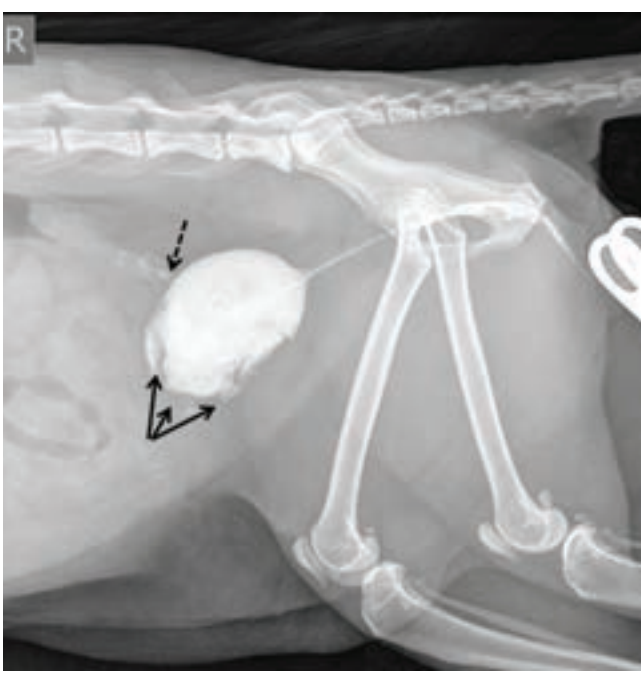

Figure 3: Retrograde positive contrast urethrocystogram – right lateral radiograph of the caudal abdomen of a cat. The cranial wall of the urinary bladder appears thickened with multiple heterogeneous filling defects, indicating severe bladder wall thickening (black arrows). There is loss of serosal detail within the abdomen, consistent with peritoneal effusion. A ruptured bladder is confirmed by the single jet of contrast leakage present on the dorsal aspect of the bladder at the level of L6 (dashed black arrow).

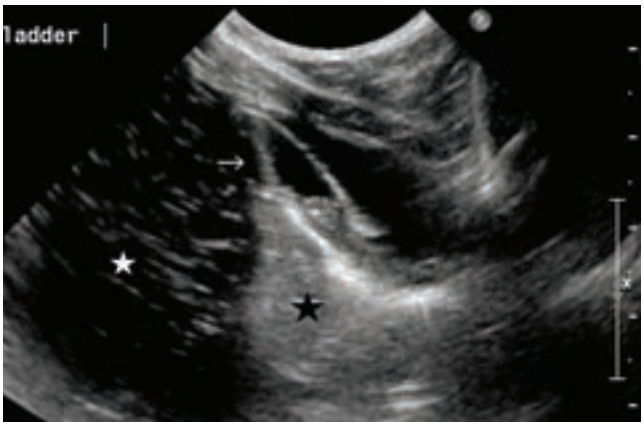

Figure 4: Abdominal ultrasound of the bladder in a cat. There is a marked abdominal effusion (white star) and a small bladder (black star). Aggitated sterile saline was instilled into the bladder via a urinary catheter. The saline exits the cranial pole of the bladder and enters the abdominal cavity, confirming the presence of a urinary bladder tear (white arrow).

leak at any point within the urinary tract.[1,7] It is important to be aware that the cat must be adequately hydrated before an enema or intravenous urography can be performed. Ultrasonography allows identification of an abdominal effusion and assessment of the architecture of the kidneys, bladder and other structures. It can be difficult to locate the site of rupture on ultrasound.[7] Identification of a bladder rupture can be improved by instilling an agitated sterile saline solution, containing numerous microbubbles, into the bladder via a urinary catheter (see Figure 4).[7]

HOW DO YOU STABILISE THESE CATS?

Surgical exploration and repair of the urinary tract may be the definitive treatment for some cats with uroabdomen. However, before the patient is suitable for surgery, it is imperative that they are cardiovascularly stable and the metabolic and electrolyte disturbances have been addressed.[1,6] The approach to stabilising these patients is summarised in Table 2.

CARDIOVASCULAR STABILISATION

When the urine enters the abdominal cavity the presence of large osmotically active particles, such as creatinine, draws water from the intracellular and intravascular fluid into the abdomen. This can result in significant volume depletion and haemodynamic compromise.[1]

Intravenous fluid therapy should be started with urgency in any patient presenting with signs of hypovolaemia and perfusion abnormalities. Oxygen supplementation may also be beneficial at this stage. Isotonic crystalloids, such as Hartmann's or 0.9% saline, have been found to be safe and effective. A balanced electrolyte solution such as Hartmann's has been considered to improve the acid-base status more rapidly than 0.9% saline.[7,8] The typical crystalloid shock rate for cats is 40-60ml/kg. However, due to the risk of fluid overload, it is now recommended that cats receive crystalloid fluid boluses in increments of one quarter of the total shock volume over 15-20 minutes. The clinical status of the cat should be revaluated before further boluses are administered in order to determine their ongoing fluid requirements. Clinical parameters to assess include the patient's mentation, capillary refill time, heart rate, blood pressure and urinary output.[7]

Cardiovascular stabilisation
- » Gain intravenous access
- » Intravenous fluid therapy
- » Provide oxygen as required

Hyperkalaemia
- » Elimination: promote potassium diuresis with intravenous fluid therapy and actively drain urine from the bladder and peritoneal cavity.
- » Redistribution: shift potassium from the extracellular space to the intracellular space. With dextrose, insulin and dextrose, sodium bicarbonate or 2-adrenergic agonists.
- » Antagonism: Antagonise the cardiotoxic effects of hyperkalaemia with 10% calcium carbonate.

Metabolic acidosis
- » Intravenous fluid therapy
- » Sodium bicarbonate may be considered in severe cases

Urine drainage
- » Catheterise the urinary bladder
- » Peritoneal fluid drainage
- » Peritoneal dialysis may be considered in severely ill patients

Pain management
- » Opioids – methadone or buprenorphine
- » Adjunctive analgesia –local anaesthesia, gabapentin, ketamine continuous rate infusion

Table 2: Summary of the initial stabilisation of uroabdomen.

ELECTROLYTE DISTURBANCES

Hyperkalaemia causes decreased cell membrane excitability. Clinically, cats may present with generalised skeletal muscle weakness and/or potentially life-threatening cardiac rhythm disturbances.[6] It is therefore, advisable to perform an electrocardiogram (ECG) on these patients.[7] Characteristic ECG abnormalities seen with a mild to moderate hyperkalaemia include bradycardia and tall, tented T waves. In the event of severe hyperkalaemia (>8.5mmol/L) there may be absent P waves, atrial standstill, ventricular tachyarrhythmias and eventually ventricular asystole.[6,7,9]

There are three approaches to the medical management of hyperkalaemia. Management focuses on antagonising the cardiotoxic effects of hyperkalaemia, followed by reducing the serum potassium levels through elimination and redistribution of potassium.[7]

ELIMINATION

This is best achieved through the administration of intravenous fluid therapy with crystalloids. By replenishing the fluid deficit the glomerular filtration rate will improve and increase the excretion of the potassium.[1] For cats with mild-to-moderate hyperkalaemia (5.5-7.5 mmol/L), fluid therapy alone is often sufficient.[7] Loop diuretics can also further increase the urinary excretion of potassium; however, they should not be used until the cat is

rehydrated, and therefore, are often not indicated in the initial stabilisation.[10]

REDISTRIBUTION OF POTASSIUM

This involves shifting potassium from the extracellular space to the intracellular space. Options include: a 5-10% dextrose infusion, regular insulin combined with a dextrose infusion, sodium bicarbonate or β2-adrenergic agonists, such as terbutaline.[7,10,11] Administration of dextrose will result in the release of endogenous insulin, this will promote the movement of glucose and potassium into the intracellular space. In reality, the exogenous administration of dextrose does not cause a significant rise in endogenous insulin. Therefore, more often, a dose of regular insulin is administered along with a continuous rate infusion (CRI) of dextrose to prevent hypoglycaemia.[9] Sodium bicarbonate or β2-adrenergic agonists are typically only considered if the cat is unresponsive to dextrose and insulin therapy [4,7]

POTASSIUM ANTAGONISM

Ten per cent calcium gluconate can be given intravenously to antagonise the cardiotoxic effect of the potassium on the myocardium. This medication typically works for 30-60 minutes. Whilst it does not reduce the potassium levels it allows time for the therapy aimed at reducing serum potassium to become effective.[7,9]

Calcium gluconate will antagonise the cardiotoxic effect of hyperkalaemia. Adjunctive therapy is required to lower the serum potassium.

METABOLIC ACIDOSIS

Impaired clearance of hydrogen ions by the kidney leads to metabolic acidosis.[4] Intravenous fluid therapy is the first line therapy for the treatment of metabolic acidosis. As mentioned before, Hartmann's may rectify the imbalance faster than 0.9% saline as the lactate is metabolised to bicarbonate which acts as an alkalinising agent.[8] If the cat is severely acidotic, pH <7 or HCO3- is <12 and the acid-base status is not improving with fluid therapy, then sodium bicarbonate may be considered. It is important to give small, incremental doses, rechecking the blood gas values after each dose to avoid over supplementation and subsequent alkalosis.[12]

URINE DRAINAGE

A urinary catheter should be placed into the bladder. This enables the urine output to be monitored more accurately. Regular drainage will also keep the bladder decompressed. This helps to reduce the hydrostatic pressure within the bladder, subsequently reducing urine leakage into the abdomen. Urinary catheter placement and bladder decompression will also allow some small tears within the bladder and urethra to heal without surgical intervention.[1,5,7,13]

Drainage of the urine residing within the peritoneal cavity using a needle or peritoneal catheter has also been found to improve patient stabilisation. The catheter or needle can be aseptically placed in a conscious cat using a local anaesthetic block.[1,2,5,7] Peritoneal dialysis may be considered in cats with a severe azotaemia, hyperkalaemia and acid-base derangements, which are not responsive to the intravenous fluid diuresis and pharmacological intervention mentioned.[5,14]

PAIN MANAGEMENT

Analgesia should not be overlooked as the chemical peritonitis caused by urine in the abdominal cavity can be very painful. Opioids such as methadone and buprenorphine can be used. Occasionally a multimodal analgesic approach is required. In addition to the opioids, a ketamine CRI, gabapentin or local anaesthesia can be considered. It is important to be aware that ketamine is excreted unchanged in the urine. Therefore until the uroabdomen is drained ketamine can be continuously reabsorbed, resulting in prolonged sedation and anaesthesia.[7]

In the presence of hypovolaemia and azotaemia the use of nonsteroidal anti-inflammatories is not recommended. In addition, α2-adrenergic agonists such as dexmedetomidine should be avoided. The bradycardia, peripheral vasoconstriction and decreased cardiac output seen with α2-adrenergic agonists may exacerbate the cardio-toxic effects of the hyperkalaemia.[7]

WHAT IS THE PROGNOSIS?

The prognosis for cats with uroabdomen depends on a number of factors. These include: the presence of concurrent conditions such as musculoskeletal injury, cause and location of the rupture, severity of metabolic disturbances and the management of these cases.[2,3,5] The prognosis is improved for cats which sustained iatrogenic injuries, had no concurrent conditions or injuries and in cats which received early diagnosis with rapid management and stabilisation.[3,5,7,15]

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READER QUESTIONS AND ANSWERS

1: WHICH ONE OF THE FOLLOWING OPTIONS CONFIRMS THE PRESENCE OF UROABDOMEN IN CATS?

A The potassium in the abdominal effusion is ≥1.9 times the serum potassium.
B The creatinine in the abdominal effusion is ≥2 times the serum creatinine. The glucose in the abdominal effusion is ≥1.9 times the serum glucose.
C The serum potassium is ≥1.9 times the potassium in the abdominal effusion. The serum creatinine is ≥2 times the creatinine in the abdominal effusion.
D The potassium in the abdominal effusion is ≥1.9 times the serum potassium. The creatinine in the abdominal effusion is ≥2 times the serum creatinine.

2: WHICH ONE OF THE FOLLOWING OPTIONS BEST DESCRIBES THE CYTOLOGICAL APPEARANCE OF A NON-SEPTIC UROABDOMEN?

A Neutrophilic inflammation
B Acellular
C Neutrophilic inflammation with intracellular bacteria
D Granulomatous inflammation

3: WHICH ONE OF THE FOLLOWING CAN BE USED TO ANTAGONISE THE CARDIOTOXIC EFFECTS OF HYPERKALAEMIA?

A Intravenous fluid therapy
B Calcium gluconate
C Sodium bicarbonate
D Insulin and dextrose

4: WHICH ONE OF THE FOLLOWING OPTIONS IS THE MOST APPROPRIATE FIRST LINE TREATMENT FOR METABOLIC ACIDOSIS?

A Promote potassium redistribution
B Sodium bicarbonate
C Intravenous fluid therapy
D Dextrose

5: WHICH ONE OF THE FOLLOWING OPTIONS IS THE FIRST STEP IN STABILISING A CAT WITH UROABDOMEN?

A Urine drainage
B Redistribution of potassium
C Cardiovascular stabilisation
D Diagnostic imaging

ANSWERS: 1: D; 2: A; 3: B; 4: C; 5: C