Uroabdomen is a condition commonly associated with abdominal or pelvic trauma that is seen in human and veterinary patients. Other etiologies of uroabdomen include urinary obstructive disorders (e.g., urolithiasis, feline lower urinary tract disease, neoplasia) and iatrogenic causes (e.g., urethral catheterization, aggressive bladder palpation, cystoscopy, cystocentesis). Uroabdomen results rapidly in life-threatening dehydration, metabolic acidosis, and electrolyte abnormalities. The first steps in treatment are fluid resuscitation and removal of urine from the abdomen. With early diagnosis and aggressive patient stabilization, this condition can be managed successfully.

Uroabdomen refers to the accumulation of free urine in the abdominal cavity caused by leakage from the kidneys, ureters, bladder, and/or urethra. The term uroabdomen includes urine accumulation in both the peritoneal and/or the retroperitoneal spaces. The veterinary literature is limited to individual case reports, a small number of retrospective studies, and one prospective study. The frequency of occurrence in veterinary patients appears to be low. Aumann and colleagues recently reported that uroperitoneum accounted for 0.1% of the hospital’s feline population. This article reviews the causes and pathophysiology of uroabdomen as well as discusses diagnosis and management of patients with this condition.

CAUSES

The causes of uroabdomen are believed to vary with species, gender, and age. It has been reported in humans, cattle, horses, dogs, and cats. In humans and small animals, uroabdomen is most frequently associated with bladder and urethral rupture caused by abdominal or pelvic trauma. As in humans, abdominal and pelvic trauma are the most common causes of uroabdomen in dogs and cats. In 1961, Meynard reported that in 7 of 9 dogs (77%) urinary bladder rupture was caused by a traumatic event. Burrows and colleagues reported that of the 40 small animal patients diagnosed with uroabdomen at the University of Pennsylvania between 1966 and 1971, 84.6% of the cases were caused by trauma. It was also reported that 42.3% had concurrent pelvic fractures. In 1982, Selcer reported that 16% of 100 dogs that had sus-
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Common Causes of Uroabdomen in Dogs and Cats

- Blunt abdominal or pelvic trauma
- Penetrating abdominal trauma
- Aggressive urethral catheterization
- Urinary tract obstruction
- Aggressive bladder palpation

Intraoperative findings of chemical peritonitis caused by uroabdomen include severe inflammation of the parietal and visceral peritoneum.

Figure 1—Intraoperative findings of chemical peritonitis caused by uroabdomen include severe inflammation of the parietal and visceral peritoneum.

PATHOPHYSIOLOGIC CONSEQUENCES

Regardless of the cause, uroabdomen results in profound dehydration, life-threatening hyperkalemia, severe azotemia, chemical peritonitis, and metabolic acidosis. The severity of the physiologic abnormalities depends on the duration of free urine within the abdomen, the site of injury, and the presence of concurrent disease. The pathophysiology of the abnormalities is multifactorial; however, the common denominator is retained free urine within the abdominal cavity. Urine is hyperosmolar as compared with extracellular fluid. The accumulation of the hyperosmolar urine in the abdominal cavity creates a concentration gradient across the peritoneum from the extracellular fluid compartment (ECF) to the abdominal cavity. Large molecules, such as creatinine, diffuse slowly and contribute to a persistent osmotic drive to shift water from the normal ECF to the abnormal abdominal fluid compartment. Small solutes present in the urine, such as urea, potassium, and other electrolytes, rapidly diffuse down their concentration gradient across the peritoneum into the ECF, resulting in an elevated concentration in the ECF. Sodium and chloride, normally present in higher concentrations in the ECF than in urine, diffuse into the abnormal abdominal fluid compartment, contributing to contraction of the ECF volume. These movements of solutes and water greatly contribute to the pathophysiology of uroabdomen. This concept is important in the diagnosis of uroabdomen.

Dehydration in patients with uroabdomen results from a combination of fluid shifts, fluid losses from vomiting, and a decrease in fluid intake. Both prerenal and postrenal factors contribute to the severe azotemia in uroabdomen patients. Dehydration leads to a decrease in glomerular filtration, resulting in a decrease in the excretion of urea and creatinine. Retention of urine within the abdomen with consequent accumulation of excretory products also results in a rise in serum urea and creatinine.

Under normal conditions, the kidney maintains an acid–base balance by excreting titratable and fixed acid. In patients with uroabdomen, hydrogen ions are retained in the urine as it accumulates in the abdomen and is reabsorbed through the peritoneal capillaries. As a result, the body’s buffer system is depleted, leading to metabolic acidosis. Hypovolemic shock, secondary to severe, untreated dehydration, can result in poor tissue perfusion and ultimately lead to the production of lactic acid, which further contributes to the patient’s acidemia.

As urine accumulates in the abdomen, a chemical peritonitis develops (Figure 1). Chemical peritonitis results in functional ileus and abdominal pain. Peritonitis also contributes to the fluid shift from the extracellular spaces into the abdominal cavity. Septic peritonitis, although uncommon, can occur in patients with a pre-existing UT infection or secondary to a penetrating wound. Peritonitis results in an increased loss of albumin into the abdominal cavity, leading to decreased oncotic pressure, which also contributes to abdominal effusion.

DIAGNOSIS

A suspected diagnosis of uroabdomen can be made based on history, physical examination findings, blood work, and survey radiography. Analysis of abdominal fluid and simultaneous comparison of creatinine and potassium concentrations in the abdominal fluid with
those in the serum are essential for confirming a diagnosis of uroabdomen. Positive-contrast studies of the UT are also necessary to confirm the diagnosis and localize the site of the lesion.

Patient history will often include recent abdominal or pelvic trauma or urethral catheterization. Clients frequently report an acute onset of hematuria and/or dysuria, leading to anuria. A progressive onset of lethargy, anorexia, and vomiting is also commonly reported. It is important to note that the ability to urinate is variable. Patients with UT disruption and uroabdomen may be able to void grossly normal urine for several hours after the initial insult. Uroabdomen, therefore, cannot be ruled out based on the patient’s ability to urinate.

Common physical examination findings are dehydration, progressive mental depression, abdominal pain, hypothermia, and evidence of external trauma. Hypovolemic shock may be present in cases of trauma or in the later stages of uroabdomen as dehydration worsens. Thorough abdominal palpation may reveal disruption of the abdominal musculature or herniation. Extravasation of urine into the subcutaneous tissue over the abdominal or perineal region causes an intense inflammatory reaction that results in pain and swelling of the affected area. The bladder may be palpable on initial physical examination and the clinician should not rule out uroabdomen based on this finding.

The abdomen is often distended in the later stages as urine accumulates. Abdominal pain resulting from chemical peritonitis and/or abdominal trauma is a common physical examination finding.

Complete blood cell count and serum chemistry abnormalities depend on the duration between the initial insult and presentation. The complete blood cell count will commonly reveal a normal to elevated hematocrit and leukocytosis due primarily to a mature neutrophilia. In one study, 25% of the cases reported neutrophilia with a left shift, which was attributed to inflammation from trauma or peritonitis. Common serum chemistry abnormalities include increases in the concentration of blood urea nitrogen, creatinine, albumin (total protein), and potassium. Azotemia and hyperkalemia are mild to moderate in the early stages but become more severe as urine accumulates in the abdomen. Venous blood gas analysis may reveal metabolic acidosis, which may be mild or even absent in the acute stages of uroabdomen.

Survey radiographs may suggest intraabdominal fluid accumulation; however, they are not definitive for uroabdomen nor will they clarify the site of disruption. As urine accumulates and peritonitis develops, serosal detail of intraabdominal structures is decreased or absent. Functional ileus is apparent as the urine irritates the serosal surfaces. Widening of or changes in the density of the retroperitoneal space (retroperitoneal streaking) may indicate the presence of blood or urine within this space. The bladder may be visible and/or possibly displaced. Again, this does not rule out bladder leakage from rupture or avulsion. The presence of pelvic fractures on survey radiography should alert clinicians to the possibility of bladder or urethral trauma.

Fluid analysis of the abdominal fluid is crucial in confirming a diagnosis of uroabdomen. The fluid obtained often does not resemble urine and is grossly serosanguineous. Cytologic analysis usually reveals the fluid to be a transudate, but it can also be an exudate if septic peritonitis is present. Fluid can be obtained by performing an abdominal paracentesis with a 20- to 22-gauge needle, a 19- to 21-gauge butterfly, or an over-the-needle intravenous catheter. With the patient in lateral recumbency, the site should be clipped and prepared aseptically to avoid introducing bacteria into the abdominal cavity. The needle is placed just to the right of ventral midline, caudal to the umbilicus and cranial to the bladder. If fluid is not easily obtained or the source of the fluid is questionable, a four-quadrant abdominal paracentesis can be performed. If the abdominal paracentesis is negative...
but suspicion of uroabdomen is strong, a diagnostic peritoneal lavage can be performed. This procedure is useful in obtaining samples when the volume of abdominal fluid is small. A thorough description of this procedure is recommended.

A definitive diagnosis of uroabdomen is made by simultaneous measurement of the creatinine and potassium concentration in the abdominal fluid and the patient’s serum. In patients with uroabdomen, the creatinine concentration of the abdominal fluid will be slightly to markedly higher than the creatinine concentration of the serum. This has been a consistent finding in dogs, foals, and cats. The retrospective study performed by Aumann and colleagues reported a mean abdominal fluid:serum creatinine ratio of 2:1 (range, 1.1:1 to 4:1) and a mean abdominal fluid:serum potassium ratio of 1.9:1 (range, 1.2:1 to 2.4:1) in cats. A recent retrospective study evaluated the abdominal fluid: peripheral blood creatinine and potassium ratios in 13 dogs with uroperitoneum. This study showed 85% of dogs with uroperitoneum had an abdominal fluid:serum creatinine ratio greater than 2:1 (mean, 5.1:1), and 100% of the dogs had an abdominal fluid:serum potassium ratio greater than 1.4:1 (mean, 2.5:1). All dogs with uroperitoneum in this study had an abdominal fluid creatinine concentration that was at least four times normal serum levels. Urea can also be measured and compared; however, it is a small molecule and rapidly equilibrates between the peritoneal fluid and the blood. Burrows reported urea concentrations of the abdominal fluid and the blood to be equal within 45 hours, the time at which abdominal fluid was obtained in this study. It can be concluded that unless urea is measured shortly after the inciting cause of uroabdomen, it is not as reliable an indicator as creatinine and potassium.

Positive-contrast radiography is the most sensitive method to diagnose urine leakage and localize the site in dogs, cats, and humans. Contrast studies should be performed only when the patient is well hydrated and metabolic derangements have been stabilized. Prolonged renal excretion of contrast media has been reported to cause renal damage in humans; therefore, excretory urography is contraindicated in dehydrated or azotemic patients. The preferred methods are excretory urography for suspected upper UT lesions and retrograde urethrocystography for lower UT lesions. The location of fluid pooling on survey radiography or ultrasonography can be instrumental to the clinician in deciding which contrast study to perform. Retroperitoneal fluid may indicate a disruption in the kidney, ureter, or distal urethra, whereas peritoneal fluid may indicate a disruption in the distal ureter, bladder, or proximal urethra.

Because injuries to the lower UT are reported to be more common in small animals, cystography and retrograde urethrocystography are generally performed first. Positive-contrast cystography is preferred over double-contrast cystography when evaluating the integrity of the bladder because it is more accurate and safer. In a study of 14 dogs with experimentally induced bladder ruptures, both imaging methods were performed. Double-contrast cystogram revealed 72% of the ruptures, whereas positive contrast revealed 100%. In patients with urinary bladder rupture, a positive-contrast cystography will reveal free contrast media in the abdominal cavity. (Figure 2)

False-negative results are most often caused by an inadequate amount of contrast agent. Positive-contrast retrograde urethrocystography is used to evaluate the urethra. In patients with urethral trauma,
Steps To Treat Patients with Uroabdomen

- Restore plasma volume
- Correct dehydration and replace ongoing fluid losses
- Treat hyperkalemia
- Place a peritoneal drainage catheter
- Place a urethral catheter
- Manage patient pain
- Provide nutritional support
- Surgically correct the UT defect

TREATMENT

The following treatment recommendations are based on a synopsis of the reviewed literature and our experience. Regardless of the site of injury or the cause of uroabdomen, the first goal of treatment is patient stabilization. Isotonic replacement fluids, such as lactated Ringer’s solution, Normosol-R®, or 0.9% NaCl should be used for initial resuscitation. Treatment of hypovolemic shock, if present, is the first order of fluid therapy. This is accomplished with an initial bolus of isotonic crystalloid fluids as mentioned. The historically reported shock dose is 90 ml/kg/hr in dogs and 55 ml/kg/hr in cats. The resuscitation volume should be tailored to meet the needs of the individual patient. The authors commonly administer a bolus of one third to one half of the shock dose, reassess the patient’s clinical response, and repeat the bolus, if needed. In patients with severe hyperkalemia in which serum potassium levels exceed 8 mEq/L or cardiac extravasation of the contrast media will be seen at the site of disruption1,7,16,18 (Figure 3). Excretory urography is the contrast procedure of choice if the area in question is the cranial retroperitoneal space.7 Extravasation of the contrast media within the renal capsule is seen in patients with renal parenchymal injury in which the capsule is intact.7 Accumulation of contrast material within the retroperitoneal space occurs with injury to the ureter or renal parenchyma with capsule disruption. The traumatized ureter, proximal to the site of injury and the renal pelvis, will often be dilated.7 Contrast material will be seen within the peritoneal cavity if the site of ureter injury is distal.6,7 Côté and colleagues21 recently described the use of ultrasonographic contrast cystography to diagnose urinary bladder rupture. They described a technique that involves infusion of the contrast agent (microbubble saline solution) through a urinary catheter while visualizing the procedure sonographically. Rupture of the urinary bladder was diagnosed when microbubbles were seen in the fluid surrounding the urinary bladder immediately after infusion of the contrast into the urinary catheter. The diagnosis was confirmed in both dogs with positive-contrast radiography and at surgery.21
Conduction disturbances are present, immediate therapy should be instituted.

The electrocardiogram is a useful tool in the detection of cardiac conduction disturbances in patients suspected to have hyperkalemia. The characteristic electrocardiographic changes seen when serum potassium levels exceed 7 to 8 mEq/L 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. Recommended therapy includes a bolus or constant-rate infusion of calcium gluconate, insulin with a dextrose constant-rate infusion, or sodium bicarbonate.

The patient’s dehydration is corrected over a period of 6 to 24 hours. Appropriate pain management must be instituted as well. Opioids, such as morphine, oxymorphone hydrochloride, hydromorphone, fentanyl citrate, or butorphanol, are commonly used to manage pain in our uroabdomen patients.

After fluid resuscitation, drainage of urine from the abdomen should be established. Continuous passive drainage of the urine is necessary for stabilization and allows effective diuresis to occur. Percutaneous placement of a peritoneal drainage catheter results in rapid removal of accumulated urine. Peritoneal dialysis/drainage catheters with multiple fenestrations are commercially available (Figure 4). Trocar chest tubes are effective in achieving passive drainage if peritoneal dialysis catheters are not available. The use of balloon-tipped catheters and latex Penrose® drains has also been reported. Placement of the catheter can often be performed using a narcotic sedative and local anesthesia. The recommended site of placement is 2 to 3 cm caudal to the umbilicus, on midline, or just to the right of midline. The abdomen is clipped and prepared aseptically. A small stab incision is made in the skin at the level of the umbilicus. The catheter and trocar/stylet are tunneled caudally under the subcutaneous tissue for 2 to 3 cm, and the catheter is placed through the abdominal wall. Once the abdominal wall is entered, the trocar/stylet is backed out 1 to 2 cm, and the catheter is directed caudally (Figure 5). The trocar/stylet is removed and the catheter is connected to a sterile, closed collection system. The catheter is secured to the body with sutures and covered with a sterile pad and light bandage (Figure 6).

Indwelling catheterization of the urinary bladder is recommended to keep the bladder decompressed and reduce urine flow into the abdominal cavity in patients with bladder and proximal urethral injury. In patients in which the urethra is traumatized and a catheter cannot be placed, prepubic tube cystostomy can be used to achieve temporary urinary diversion. The cystostomy tube can be placed surgically or percutaneously. A balloon-tipped Foley catheter and a commercially available percutaneous prepubic cystostomy catheter are used for cystostomy tubes.
placed surgically and percutaneously, respectively. Both techniques and catheter types are thoroughly described by McLoughlin.\textsuperscript{16} This catheter should also be connected to a sterile, closed collection system (Figure 7). The fluid from both collection systems is measured and recorded frequently. The volume of fluids infused and drained is monitored closely. If an appropriate volume of fluid is not collected, the patency of the catheters should be checked by aggressively flushing the catheter. Omental obstruction of the peritoneal drainage catheter is a common complication.\textsuperscript{16,18,24} If the catheters are patent and/or the patient’s uremia is not resolving, the possibilities of urine in the retroperitoneal space or oliguric/anuric renal failure must be investigated immediately.

The presence of free urine within the abdominal cavity is not an indication for an immediate exploratory laparotomy as long as drainage is established.\textsuperscript{25} Only after the patient is stabilized should definitive repair of the UT injury be considered. Definitive surgical management is based on the location of the leakage site and can usually be performed within 24 to 48 hours. Delays in surgical correction can be longer based on the patient’s response to initial medical management.\textsuperscript{1,18,19,25}

Treatment options for ureteral trauma include ureteronephrectomy, placement of a ureteral catheter (stent), ureteral anastomosis, or reimplantation of the ureter into the bladder (neoureterocystostomy).\textsuperscript{15,25,26} Reimplantation of the ureter and ureteral anastomosis in small animals is technically difficult and often requires magnification of the operative field.\textsuperscript{26} Avulsion of the ureter from the renal pelvis usually requires nephrectomy.\textsuperscript{16,18,25,26} Midureter lacerations can be sutured, but stricture formation is a common complication, especially in cats.\textsuperscript{18,25,26} Longitudinal spatulating of the ends of the ureter may be performed to increase the circumference of the ureteral anastomosis. Placement of a stent across the surgical site to decrease the contact of urine with the suture line may reduce the chance of stricture formation.\textsuperscript{25,26} Distal ureter trauma is best repaired by reimplanting the ureter into the bladder.\textsuperscript{16} If the ureter length is shortened due to the injury or surgical debriedment, tension can be alleviated by freeing the kidney from its retroperitoneal attachments and moving it caudally, or drawing the bladder cranially and securing it to the fascia of the sublumbar musculature.\textsuperscript{26}

Surgical repair of the bladder historically has been the treatment of choice. Identification of the site, debriding of the edges, and routine closure of the defect are the most common recommendations.\textsuperscript{1,11,16,18,22,25} Osborne and colleagues\textsuperscript{27} advocated the consideration of medical management of feline patients with iatrogenic urinary bladder rupture. In carefully selected cases, the use of an indwelling urinary catheter and a closed collection system to maintain the bladder in a contracted state may allow the disrupted site to seal.\textsuperscript{27} It was also reported that three of the 14 dogs with surgically induced bladder rupture spontaneously sealed within 45 hours without treatment.\textsuperscript{3} It is the authors’ experience that rapid, definitive repair of the urinary bladder rupture and shorter hospitalization time are achieved with surgical correction.

Complications of traumatic urethral injury include stricture formation and urinary incontinence.\textsuperscript{15,16} Urine contact with tissues over a period greater than 12 to 24 hours will result in inflammation, edema, and cellulitis, which delays healing and promotes fibrosis.\textsuperscript{10,16,26} As with the ureters, the consequences of urine contact can ultimately result in stricture formation and obstruction at the injury site. Placement of a transurethral catheter (urethral stenting) or other means of urinary diversion, such as tube cystostomy, is necessary to protect the lesion from urine.\textsuperscript{10,11,16,25} With regard to its diameter, care must be taken when choosing the size of the transurethral catheter because overdistention of the urethra can have a deleterious effect on healing. Noncircumferential defects in the urethra have been reported to heal when urinary diversion techniques are used.\textsuperscript{10,16,25} The urethral catheter should remain in place for 2 to 4 weeks, depending on the size of the urethral defect.\textsuperscript{1,16,18} Complete disruption of the urethra must be corrected by anastomosis or urethroscopy.\textsuperscript{16,18,25} Urinary diversion techniques have also been recommended after urethral anastomosis to decrease the occurrence of complications.\textsuperscript{10}

The decision to treat the uroabdomen patient surgically or conservatively should be based on the location
and severity of the UT injury, the condition of the patient at presentation, and the patient’s response to initial stabilization.\textsuperscript{1,16,25} The presence and severity of concurrent injuries, predisposing factors (e.g., neoplasia), and financial constraints of the owner must also be considered when formulating a treatment plan.

CONCLUSION

Uroabdomen in small animals is a condition commonly associated with abdominal and pelvic trauma. Uroabdomen should be considered in patients with a history of trauma, urinary obstruction, or urethral catheterization. A diagnosis can be obtained by simultaneous measurement of creatinine and potassium of the serum and abdominal fluid, coupled with contrast radiography of the UT. Emergency management of these patients should always include aggressive fluid therapy and drainage of urine from the abdomen. Definitive repair should not be attempted until patient stabilization is achieved. Repair can be delayed for a number of days as long as continuous abdominal drainage and urethral catheterization have been accomplished.\textsuperscript{1,16,25} Various mortality rates have been reported in both veterinary and human patients. A mortality rate of 11% to 44%,\textsuperscript{6,28} 42.3% to 56.2%,\textsuperscript{1} and 38.4%\textsuperscript{2} have been reported in humans, dogs, and cats, respectively. Concurrent injuries, location of the UT disruption, and a delay in diagnosis and treatment have been repeatedly shown to greatly increase the mortality rate in patients with uroabdomen.\textsuperscript{1,6,12,16} With early diagnosis, aggressive emergency therapy, and patient stabilization, uroabdomen can be managed successfully.

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1. Which of the following abnormalities contribute to dehydration in patients with uroabdomen?
   a. vomiting and decreased fluid intake
   b. peritonitis
   c. shifting of extracellular fluid into the abdominal cavity
   d. all of the above

2. Which of the following is not indicated in the initial treatment of uroabdomen in animals with severe uremia?
   a. exploratory laparotomy
   b. rehydration and continued fluid therapy
   c. removal of urine from the abdominal cavity
   d. pain management

3. A definitive diagnosis of uroabdomen is made by
   a. an excretory urethrogram, resulting in extravasation of contrast material.
   b. a positive diagnostic peritoneal lavage, yielding a serosanguineous fluid.
   c. an abdominal fluid:serum creatinine ratio greater than 1.
   d. a and c

4. Which statement regarding uroabdomen patients is false?
   a. Both prerenal and postrenal factors contribute to the severe azotemia seen in uroabdomen patients.
   b. A patient that urinates grossly normal urine most likely does not have a disruption in the UT.
   c. Abdominal pain is a consistent physical examination finding in patients with uroabdomen.
   d. Septic peritonitis is a potential complication in patients with a preexisting UT infection.

5. Which solutes diffuse from the abdominal cavity in animals with uroabdomen?
   a. urea, creatinine, chloride, and potassium
   b. creatinine, sodium, and urea
   c. sodium, hydrogen, potassium, and creatinine
   d. potassium, urea, and creatinine

6. Which of the following is not a common cause of uroabdomen in dogs and cats?
   a. motor vehicle trauma
   b. urinary outflow obstruction
   c. transitional cell carcinoma
   d. traumatic urethral catheterization

7. Comparison of abdominal fluid and serum concentrations of ________ is the most reliable laboratory method to confirm the presence of uroabdomen.
   a. hydrogen
   b. creatinine and potassium
   c. urea
   d. sodium

8. Survey abdominal radiography of patients with uroabdomen may reveal which of the following?
   a. retroperitoneal streaking
   b. poor serosal detail of intraabdominal structures
   c. generalized ileus
   d. any combination of the above

9. The ________ is the most common site of UT disruption in dogs and cats.
   a. distal ureter
   b. urinary bladder
   c. renal pelvis
   d. proximal urethra

10. Which statement regarding the peritoneal drainage catheter is false?
    a. After its placement, the catheter is connected to a sterile, closed collection system.
    b. If the catheter is not draining the abdomen effectively, the first step is to flush the catheter to check its patency.
    c. After placement, the catheter is capped and manually aspirated once every 2 to 4 hours.
    d. Once the site of leakage is definitively repaired, the catheter is removed.

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