Max's Mighty Mystery

Sarah A. Baker
Mississippi State University
College of Veterinary Medicine
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Advisor:
Sarah Castaldo, DVM

Introduction:

Uroabdomen results from the rupture of the urinary tract with subsequent accumulation of urine in the peritoneal cavity, retroperitoneal cavity, or both. It is most often associated with vehicular or blunt trauma to either the abdomen or pelvis. The urinary bladder is the most common site of rupture and occurs due to a rise in intraperitoneal pressure caused by the traumatic event. Males cannot easily adapt to these increases in pressure due to their long, narrow urethra and thus are at an increased risk of rupture. Patients with pelvic fractures are also at an increased risk of uroabdomen due to deformation of the pelvic canal in addition to possible lacerations of the bladder or urethra from sharp bone fragments. (Grimes, 2018; Reineke, 2018)

Urethral rupture secondary to external trauma is another cause of uroabdomen; however, it is much less common than bladder ruptures in both dogs and cats. This is due to the mobility of the ureters as well as protection from the retroperitoneal space, dorsal body wall musculature, and peritoneal organs. If a urethral rupture does occur it is most often in the proximal portion. Nontraumatic causes of uroabdomen in dogs and cats include spontaneous bladder rupture secondary to urethral obstruction, bladder neoplasia, genitourinary tract surgery, accidental injury to the urinary tract during abdominal surgery, or iatrogenic causes from manual bladder expression, cystocentesis or urethral catheterization. (Stafford, 2013).

Diagnosis of uroabdomen is based on history and physical exam findings along with laboratory evaluation and imaging studies. Initial diagnostic testing performed for all animals suspected to have urinary tract trauma typically include a CBC and measurement of serum electrolyte concentrations, a serum biochemistry profile, and acid-base status. Since vehicular or blunt trauma is the most common cause of uroabdomen, the urinary tract is often overlooked as

most patients present with other life-threatening conditions such as hypovolemic shock, pulmonary contusions, traumatic brain injuries, and fractures. Some dogs and cats that present after motor vehicular trauma show no signs of urinary tract disruption while others show nonspecific signs of vomiting, anorexia, weakness and lethargy secondary to progressive azotemia, hyperkalemia and metabolic acidosis, in addition to hypovolemic shock.. Patients may also show signs of abdominal pain with or without signs consistent with urinary tract trauma. Physical exam typically reveals a palpable fluid wave in the abdomen as well as bruising of the inguinal region or perineum. Patients can have hematuria and display signs of stranguria, dysuria, or anuria and the urinary bladder may or may not be palpable. The most common abnormalities associated with uroabdomen are azotemia, metabolic acidosis, and electrolyte abnormalities that include hyponatremia, hyperphosphatemia, and hyperkalemia. Azotemia and hyponatremia often develop within 24 hours whereas hyperkalemia may not develop for 48 hours or more after initial onset. (Stafford, 2013; Colopy, 2016)

If abdominal effusion is noted on abdominal palpation, a sample of the effusion should be collected by abdominocentesis and analyzed. Urine within the peritoneal cavity can appear as a transudate, modified transudate, or exudate, with variation caused by hemorrhage or inflammatory cells. To confirm that the effusion is originating from the urinary tract, the ratios of creatinine and potassium in the abdominal effusion to peripheral blood can be compared. Rule outs for abdominal effusion include hemoperitoneum, septic peritonitis, bile peritonitis, and uroperitoneum. (Stafford, 2013)

Treatment of uroabdomen is dependent on the location and severity of the urinary tract disruption. Though uroabdomen is a medical emergency, surgical treatment is often required for

definitive repair of the urinary tract defect. Aggressive IV fluid administration, urinary diversion, and treatment specifically targeting hyperkalemia are often necessary to stabilize these patients prior to surgery. (Grimes, 2018) The overall prognosis for small animals with uroabdomen varies. Factors impacting prognosis include the site and severity of the urinary tract insult, presence of other injuries, resolution of electrolyte and acid-base derangements during stabilization, restoration of adequate renal perfusion and function, and healing of the injured site of the urinary tract without complication. (Colopy, 2016)

History and Presentation:

Max, an approximately 10-year old male intact German Shepherd, presented to MSU-CVM emergency service on 10/25/19 for acute abdominal pain and possible gastrointestinal obstruction. The owners stated that Max was usually an energetic dog, but starting late the night of 10/24/19, he began to act lethargic and depressed. When lifted to be put on the bed, Max was reactive to the pressure applied to his abdomen. The owners also noticed that Max was not urinating as much as he normally does. He was taken to his referral veterinarian on 10/25 where he was sedated with ketamine and diazolam. Radiographs and bloodwork were performed. Bloodwork revealed a mild azotemia (BUN 44, Creatinine 3.3). Abdominal radiographs revealed gas dilated intestinal loops and fecal matter in the colon. 8mg of Zofran was administered at 12pm. Around 1-2pm, Max vomited a large amount of darkly colored liquid material. He was then referred to MSU-CVM. He lives with one other black and tan female intact German Shepherd and he is primarily an outdoor dog; however, he sleeps inside. He is up to date on vaccinations and is on Bravecto for flea and ticks. He is not on any heartworm prevention. He

was bitten by a copperhead last year but does not have any previously diagnosed medical conditions.

On presentation, Max was depressed and responsive. His vital parameters were within normal limits with a temperature of 101.9 F, heart rate of 112 beats per minute and a respiratory rate of panting. He had strong synchronous femoral pulses and no murmurs or arrhythmias were heard on auscultation. His mucous membranes were pink and moist and he was hypersalivating. His capillary refill time was mildly increased at 1 second. He was approximately 6% dehydrated with a prolonged skin tent and mildly sunken eyes. On AFAST, free abdominal fluid was noted and he was very painful on palpation of his abdomen. He was hypertensive with a blood pressure of 164/136 (MAP: 145). Differentials at the time included GI or urinary tract rupture, urinary tract obstruction, GI obstruction/foreign body, biliary rupture/obstruction, and neoplasia.

Diagnostic Approach:

After a thorough physical exam, an abdominocentesis was performed using the 4-quadrant technique which revealed a small amount of red-tinged fluid. Glucose and lactate values of the abdominal fluid were compared to the values of the peripheral blood. Lactate values were 1.4 mmol/L for both the abdominal fluid and peripheral blood sample. Glucose measurements were 113 mg/dL in the peripheral blood and 180 mg/dL in the abdominal fluid. A Serum Chemistry was performed and revealed azotemia (BUN of 81 mg/dL and a Creatinine of 5.44 mg/dL), hyperkalemia (6.82 mmol/L), hyperglobulinemia (5.0 g/dL) , increased anion gap (21 mmol/L), increased Total Bilirubin (1.1 mg/dL), high CK (422 U/L), and a low CO2 (17.9 mEq/L). A CBC was performed revealing a lymphopoenia (177.0 /uL), increased MCHC (38.4

g/dL), and neutrophilia (16480.0 /uL). His PCV and total protein were within normal limits at 36% and his 6.0. Abdominal radiographs were performed and showed evidence of peritoneal effusion as well as gas dilated small intestines. He was started on a Fentanyl CRI, Cerenia, Pantoprazole and fluids at 1.5x maintenance overnight. Later that night, Max's respiratory rate increased, and a 3mcg/kg bolus of fentanyl was administered for pain. He was then transferred to the internal medicine service the morning of 10/26.

On the morning of 10/26/19, Max was sedated with Dexmedetomidine and repeat abdominal radiographs were performed showing peritoneal effusion as well as dilated small intestines which could be due to either enteritis, peritonitis, or less likely an unidentified small intestinal mechanical obstruction. An abdominal ultrasound was also performed and showed a large amount of anechoic free fluid diffusely throughout the abdomen. The urinary bladder wall was diffusely thickened and irregularly marginated, measuring up to 1.47 cm in thickness. There were multiple striated, smoothly marginated, hypoechoic regions within the urinary bladder wall as well as a single irregularly shaped, smoothly marginated nodule confluent with the dorsal urinary bladder wall. A new sample of abdominal fluid was obtained, as well as a urine sample from the bladder, and a FNA of the bladder wall. The fluid BUN and Creatinine were compared to Max's blood BUN and Creatinine from the night before. The BUN and Creatinine in the fluid (158 mg/dL and 23.5 mg/dL) were significantly higher than that in the blood (81 mg/dL and 5.44 mg/dL). At this time, given his history, physical exam, bloodwork, radiographic and ultrasonographic findings as well as fluid analysis from abdominocentesis, uroabdomen was suspected. To determine the source of the uroabdomen, further diagnostics were performed.

Max was put under General Anesthesia and a cystogram was performed which revealed positive contrast extending from the apex of the urinary bladder with a progressive increase in contrast within the peritoneal space. The cystogram also revealed undulating margins of the urinary bladder which may be due to either cystitis or neoplasia. A diagnosis of uroabdomen due to a urinary bladder rupture was then made. An iSTAT was performed prior to surgery and showed azotemia (BUN 91 mg/dL; creatinine 5.5 mg/dL), mild hypochloremia (107.1 mmol/L), moderate hyperkalemia (5.52 mmol/L), increased anion gap (19.9 mmol/L), and a decreased PO2 (56.7 mmHg). Max was then prepped and taken to surgery.

Pathophysiology:

When urine leaks from the urinary system into the abdomen, both potassium and creatinine from the leaked urine will accumulate in the abdominal effusion. The peritoneal lining can reabsorb potassium more efficiently than creatinine, resulting in peripheral hyperkalemia. In order to remove some of the circulating potassium, the kidneys respond by concentrating it in the urine. However, due to the urinary disruption, this potassium rich urine leaks into the abdomen and creates a higher potassium concentration in the abdominal effusion than the peripheral blood. This increase in potassium causes an increase in cell membrane excitability which can result in life threatening arrythmias progressing from bradycardia to ventricular fibrillation or asystole. The creatinine concentration of the abdominal effusion will be higher than the peripheral creatinine concentration because creatinine cannot be readily absorbed across the peritoneal membrane so it remains stuck in the abdominal effusion. Creatinine can cause the movement of water from the intracellular and intravascular spaces into the peritoneal cavity. This movement of water leads to dehydration and hypovolemia. The resulting dehydration and hypovolemia will

decrease the glomerular filtration rate, further limiting the excretion of potassium, creatinine, urea and phosphorus. A metabolic acidosis may be present secondary to both hypovolemia and from a failure of the kidneys to excrete hydrogen ions. Hydrogen ions accumulating in the abdominal cavity are reabsorbed into the circulation, eventually depleting the blood buffering capabilities. The presence of urine in the abdomen is irritating leading to a chemical peritonitis and signs of abdominal pain. If the urine is infected, a septic peritonitis may be identified. (Reineke, 2018; Colopy 2016).

Effusion creatinine > 4X that of the serum creatinine's upper reference limit, an effusion creatinine that is 2X or > 2X that of the serum creatinine, and an effusion potassium that is greater than 1.4X that of the serum potassium are all supportive of a uroperitoneum. If 2 or more of these tests are identified in the patient, these combined tests offer 100% sensitivity and specificity for diagnosing a uroperitoneum. (Cornell University; Colopy, 2016)

Treatment and Management options:

Max was taken to surgery on 10/26/19 for a cystotomy to repair his ruptured bladder. Max was placed in dorsal recumbency, clipped, and aseptically prepped with 4% chlorahexidine scrub. An incision was then made over the ventral abdomen from the caudal umbilicus extending caudally to the pubis with a #10 scalpel blade. Dissection with Metzenbaum scissors was used to expose the linea alba. The abdominal wall was elevated and using a #15 scalpel blade, a small stab incision was made in the linea alba to open the abdomen. The incision was extended and moistened laparotomy sponges were placed along the edges of the incision. Balfour retractors were used to retract the abdominal wall and provide better visualization. The bladder was identified and a stay suture was placed at the apex. An incision was then made on the ventral

surface of the body of the bladder as dorsal incisions are thought to predispose to complications as a result of decreased visualization and the anatomic proximity of the ureter. (Appel, 2012) Pool suction was then used to remove the urine. The bladder was inverted to examine the mucosal surface. Nodules were noted diffusely throughout the bladder so a full thickness biopsy was taken, encompassing the nodules. The bladder was then flushed with warm saline multiple times and closed using a Cushing pattern and 3-0 PDS. A leak test was performed with 3-5 ml of sterile saline and no leak was identified. The abdomen was then lavaged with 1 liter of warm saline. The abdomen was closed in three layers. The abdominal wall was closed with 2-0 PDS in a simple continuous pattern. The subcutaneous tissue was closed with 2-0 PDS in a simple continuous pattern. The skin was closed with 3-0 PDS in an intradermal pattern. A tefla was placed over the incision and 3 sure sites were placed to hold the tefla in place and protect the incision. Max was given a dose of Baytril pre-op and was maintained on LRS, NaCl, Fentanyl, and Lidocaine intra-op. During surgery, he became increasingly acidotic and was given 22 mEq of sodium bicarbonate. There were no other complications during surgery and recovery from anesthesia was uneventful. A urinary catheter was placed during recovery and was emptied and measured Q4h. Post-operatively, he was maintained on a Fentanyl CRI at 3 mcg/kg, LRS at 142.5 mLs/hr, Baytril 10 mg/kg IV Q24h, Cerenia 1 mg/kg IV Q24h, Unasyn 30 mg/kg IV Q8h, and Pantoprazole 1 mg/kg IV Q12h. He had a pain score of 4/24 following surgery. He remained stable throughout the night of surgery and the next day and was urinating normally. His pain levels continued to decrease so his Fentanyl was discontinued on 10/28 and he was switched to Tylenol 4 (2 mg/kg) and Gabapentin (9 mg/kg) orally every 8 hours. His fluids were also discontinued that same day. On 10/29, his results from urine culture showed growth of Citrobacter freundii and his Unasyn was discontinued. His Cerenia and Pantoprazole were also

discontinued that same day and he was switched to oral Baytril. His urinary catheter was pulled on 10/30. Afterwards, he was urinating normally and showed no signs of straining.

Case Outcome:

Max was discharged from MSU-CVM on October 31, 2019. He was sent home with Tylenol 4 and Gabapentin for pain control and inflammation. Due to his positive urine culture of Citrobacter freundii, he was sent home with Baytril. Max's owners were instructed to keep Max confined and prohibited from exercise for 2 weeks to allow his incision to heal. He was to wear an E-collar at all times and owners were instructed to keep his incision dry for the next 10-14 days. His incision was to be rechecked in 7-10 days and at that time a urine culture would be performed.

Biopsy of the bladder wall revealed that the underlying lamina propria, muscularis, and serosal layers were markedly expanded by increased amounts of hemorrhage and the mucosal epithelium was multifocally ulcerated (Multifocal ulcerative cystitis with transmural hemorrhage). Multifocally the lamina propria were also expanded by occasional loose aggregates of neutrophils and fewer lymphocytes (Multifocal Suppurative perivascular inflammation). The underlying cause for his ruptured bladder is unclear; however, potential rule outs based on the changes appreciated may include traumatic rupture or rupture secondary to an obstruction.

Conclusion:

Uroabdomen is a life-threatening condition that requires rapid diagnosis and stabilization of electrolytes (mainly hyperkalemia), acid-base disturbances, and azotemia before any consideration for advanced diagnostics, anesthesia, and surgery are considered. Once the patient

is deemed stable, diagnostics to determine the location of urinary tract disruption followed by surgical repair can be performed. Close monitoring in the postoperative period is important to document improvement or resolution of laboratory abnormalities as well as overall patient well-being. In addition, it is important to monitor for postoperative complications such as ongoing urine leakage from dehiscence of the surgical site, stricture formation, urinary incontinence, or progression to urosepsis. (Stafford, 2013).

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