Uroperitoneum in the Equine Neonate  ( 7-Nov-2001 )

S. S. Hyman
New Bolton Center, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, Pennsylvania, USA.

Introduction
Uroperitoneum has been recognized as a syndrome in foals for over 50 years [1,2]. Traditionally, it has been thought to present most frequently in the 24 - 36 hour old male foal during the post-parturient period [2-4]. Previous reports had a proportionately larger affected male than female population [2,3,5]. It was hypothesized that colts were more at risk due to their long narrow high resistance urethra that was less likely to allow bladder emptying, resulting in rupture of a full bladder during parturition when high pressures were applied focally or circumferentially around the bladder [1,6-8]. Rupture or disruption of any structure of the urinary tract can occur. Of the case studies reviewed, 8 of 16 foals with an identified site of rupture in one study, and 24 of 31 foals in another, had a bladder defect [4,9]. The dorsal wall of the bladder has been reported to be a frequent disruption site, with the ventral wall less likely to be involved [4,6,9]. The urachus appears to be the next most commonly affected structure. A few cases of ureteral and urethral defects have been reported [4,5]. Sepsis does not appear to favor one site over the others [9].

Pathophysiology
The exact pathophysiology of uroperitoneum is not fully understood. It was once thought that high pressures exerted on a full bladder during parturition was the main cause [1,2,6-8]. Others proposed that a full bladder and obstruction due to a partial umbilical cord torsion may be a cause of rupture during parturition [6,11]. Strenuous exercise and external trauma have also been seen as causes [6,9-11]. The lesions associated with the above causes typically have inflamed and hemorrhagic edges, consistent with trauma. A few reports exist where possible congenital bladder wall defects were proposed due to the smooth and non-inflamed edges of the tissue [6,10,12]. These claims, however, are disputed. Recently, more focus has been put focal infection and sepsis as factors leading to urinary tract rupture and uroperitoneum. Adams and Koterba [4] showed an association between septicemia, urinary tract infections and rupture of the urinary tract. This finding was supported by Kablack, et.al. In a retrospective study of 31 cases of uroperitoneum they found a relationship between neonatal illness and the development of uroperitoneum [9].

Clinical Signs
Clinical signs associated with uroperitoneum in the neonatal foal typically include straining to urinate, dribbling urine, and a stretched out stance [2]. At this point, the condition is often confused with colic due to meconium impaction of the gastrointestinal tract. Meconium impacted foals, however, usually have a roached back and stand with all four limbs underneath them as they strain to defecate [2,3]. Weakness, tachycardia, tachypnea, and not sucking well are also seen in uroperitoneum [2-4,6,9,14]. As the uroperitoneum progresses and fluid accumulates, a distended abdomen may be observed [2,3]. A fluid wave may be felt on ballottement of the abdomen [3]. Occasionally, scrotal accumulation of urine can occur [15]. With the recognition of sepsis as a contributing factor to the development of uroperitoneum, many foals also show signs of concurrent infection and sepsis, including fever, injected mucus membranes, diarrhea, and disease of other body systems.

Laboratory Findings
Laboratory findings are variable, depending on the duration of the uroperitoneum as well as the presence and severity of sepsis. However, some classic findings include hyperkalemia, hyponatremia, and hypochloremia [3-5]. These abnormalities arise from the equilibration of urine electrolytes and water with blood across the peritoneal membrane, allowing for loss of sodium and chloride and retention of potassium. The usual foal diet consists of milk that is high in potassium and low in sodium, perpetuating and exacerbating the electrolyte abnormalities. Those cases recognized very early in the clinical course of the disease may not have these classic signs. The time for equilibration of electrolytes and water across the peritoneal surface is not known in the horse, however, cases diagnosed may not have electrolyte abnormalities [9]. Foals who develop uroperitoneum while hospitalized, and receiving intravenous fluids, may not have electrolyte imbalances due to the
also possible. Tears within the bladder are readily seen and the empty bladder in a fluid-filled abdomen will collapse on itsel f, but usually cannot identify the defect. Contrast studies can help to further delineate the site of the lesion. Positive

Many diagnostic procedures exist to help confirm the diagnosis of uroperitoneum. Abdominocentesis with peritoneal to

Diagnostic Procedures

Many diagnostic procedures exist to help confirm the diagnosis of uroperitoneum. Abdominocentesis with peritoneal to

Treatment

Uroperitoneum should be treated as a medical emergency. Initial treatment should be directed at stabilizing the patient and correcting any electrolyte and acid-base abnormalities and providing fluid volume replacement. 0.9% or 0.45% saline should be used until laboratory data are available so that additional potassium is not provided to a potentially hyperkalemic patient. This will also help to slowly correct the hyponatremia and hypochloremia. Potassium levels >5.5 mEq/L can be life threatening and should be addressed quickly. The hyperkalemia can be managed by peritoneal drainage to help remove the source of the potassium. This can be performed with teat canulas, Foley catheters, large gauge (16g or 14g) intravenous catheters, or in the author's experience, human peritoneal dialysis catheters work extremely well and are less likely to be fully blocked with omentum and fibrin due to their multiple fenestrations. Fluid replacement should match the amount of fluid removed to prevent acute hypotension due to expansion of previously collapsed capillary beds. Abdominal drainage will also help ventilation and decrease the work of breathing by decreasing pressure on the diaphragm. Serum potassium levels can be decreased preoperatively by this technique. We have been successful with this technique in our hospital. Use of intravenous Ca++ gluconate, glucose, sodium bicarbonate, and insulin will also decrease potassium levels, however, it must be remembered that the potassium has only been driven into the cells and one therapy is discontinued, as total body potassium is still increased, it can once again equilibrate out into the serum causing increased potassium levels. Hyponatremia should also be slowly corrected. The central nervous system maintains its own sodium homeostasis even in the face of whole body depletion. Rapid administration of sodium (i.e. hypertonic saline) can lead to an intracellular influx of sodium and water that can cause cellular edema and neurological signs. If hyponatremia has been of several days duration, rapid correction can result in central pontine lesions, as with time, the brain will adjust intracellular osmolytes to match the extracellular conitions.
Blood cultures should be obtained prior to administration of preoperative antimicrobials. Broad-spectrum coverage such as that provided by penicillin and amikacin or ceftiofur sodium is recommended until culture results are available. Aminoglycosides should be used cautiously in foals with decreased renal function and serum peak and trough levels should be monitored. Foals with failure of passive transfer should be treated with adequate volumes of intravenous plasma. Once the metabolic abnormalities have been addressed, surgical management can be considered. Most cases require surgical correction [2-5,9,15]. One case of medical management of a small dorsal tear using an indwelling Foley catheter was described by Lavoie [14]. Often the timing choice for surgical intervention is difficult due to the patient instability and the anesthetic risks associated with the frequently present hyperkalemia and hyponatremia. Most foals are weak, dehydrated, acidotic, and hyperkalemic. However, if the preoperative medical stabilization is properly accomplished, the anesthetic risks can be minimized. The ability to mask down these foals with inhalant anesthesia, as well as safer agents such as isoflurane, decreases risks. Surgical repair techniques have been first described in 1958 by DuPlessis [2]. Since then many other techniques for repair of ruptured bladder have been presented [5,18-20]. Briefly, a midline or paramedian incision is made. The bladder defect is identified and closed with absorbable suture in an inverting pattern making sure to place the sutures in healthy tissue. The abdomen should be lavaged, especially if cytology of peritoneal fluid suggests infection. Urinary catheters and peritoneal drains can be placed on surgeon preference. Body wall and skin closure is routine. It is now usual for the internal umbilical remnant to be removed at the time of surgery. Laparoscopic repair of a bladder defect was successfully performed by Edwards, Ducharme, and Hackett [13], however, the possibility of urolith formation from non-absorbable laparoscopic sutures may be a contraindication for this technique [13]. Repair of other urinary tract defects (ureters, urethras) are described in the literature [18-20].

Complications associated with the primary urinary tract defect as well as those of concurrent disease can occur. Recurrence of the urinary tract rupture can occur. Dysrhythmias from electrolyte abnormalities are commonly seen. Hypotension from hyperkalemia and hypocalcemia may also be present. Other non-urinary tract complications may be just as serious. Sepsis, hypoxemia, pneumonia, peritonitis, and acute respiratory distress syndrome all complicate the management of uroperitoneum. One case was complicated by a hole in the diaphragm that allowed peritoneal fluid into the thorax and caused pulmonary edema [3].

**Prognosis**

Prognosis is closely associated with concurrent illness, especially septicemia. Uncomplicated uroperitoneum from a defect in the bladder has a good prognosis [2,4,5,9]. If the location of the lesion is other than the bladder, the prognosis is historically not as favorable. Richardson and Kohn reported only 1 of 10 survivors with lesions found in the urachus or ureters [5]. This may have been due to concurrent septicemia, which was not factored into risk factors for survival in that study. Foals with septicemia have a much poorer prognosis. Adams and Koterba reported a 50% long-term survival rate in 18 foals on their study. Seven of the nine non-survivors had bacterial or fungal infections [4]. Kablack, et al. [9] found an association between death and sepsis, increased serum sodium and chloride, decreased potassium, and an increased peritoneal to serum creatinine ratio. The unusual association with normal electrolyte concentrations is likely due to foals being hospitalized for conditions such as sepsis and receiving intravenous fluid therapy.

**References**


All rights reserved. This document is available on-line at www.ivis.org. Document No. A0415.1101.