Uroperitoneum in a preparturient mare

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Abstract

A 12-year pregnant Thoroughbred mare at ~ 340 days of pregnancy was presented for evaluation of lethargy, anorexia, and colic signs. Reproductive examination was unremarkable; however, uroperitoneum was diagnosed via transabdominal ultrasonography, serum biochemistry, and peritoneal fluid analysis. Mare was medically stabilized for 24 hours. To promote fetal maturation, 80 mg dexamethasone was given prior to Caesarean section and exploratory celiotomy. A ventral urinary bladder wall rupture and multiple descending colon mesenteric rents were identified and repaired. Foal was delivered alive, treated for neonatal maladjustment syndrome, and discharged after 14 days. Seven days after surgery, severe complications of the mare led to its abrupt decline in health and sudden death.

Keywords: Mare, uroperitoneum, bladder rupture, caesarean section, dexamethasone

Background

Uroperitoneum is an insidious and potentially fatal disease process in horses.[1,2] Although most commonly diagnosed in foals,[1,3] uroperitoneum secondary to bladder rupture is uncommon in adult horses and reportedly associated with parturition,[2,4-7] urolithiasis,[8,9] or trauma.[10] Clinical signs typically present from 48 to 72 hours after rupture and consist of lethargy, anorexia, variable urination (anuria, pollakiuria, or stranguria), tachycardia, tachypnea, abdominal distension, or cardiac arrest.[3] Hematologic findings often include azotemia, hyperkalemia, hyponatremia, hypochloremia, and metabolic acidosis.[3] This report describes uroperitoneum secondary to urinary bladder rupture in a preparturient mare, that to the authors' knowledge, has not previously been reported. This case underscores the importance of including uroperitoneum after urinary bladder rupture as a differential for late-term pregnant mares and highlights the possibility for safe use of maternal dexamethasone treatment after 335 days of pregnancy to hasten fetal maturity prior to Caesarean section.

Case presentation

A multiparous 12-year Thoroughbred mare ~ at 340 days of pregnancy was referred to Iowa State University's Lloyd Veterinary Medical Center for evaluation of a 24-hours history of lethargy, decreased appetite, and persistent colic. At presentation, mare exhibited muscle fasciculations and weight shifting. She was tachycardic (72 beats/minute), tachypneic (48 breaths/minute), and afebrile with a normal rectal temperature (37.9°C). There was marked mammary gland development, with a slightly sticky, opaque mammary secretion. Remainder of the physical examination was unremarkable.

A complete blood count identified an elevated hematocrit (51.7%; reference range [RR]: 34 - 45%), and a mild leukocytosis (12.41 × 10^3/µl; RR: 5.0 - 11.0 × 10^3/µl) characterized by moderate mature neutrophilia (10.9 × 10^3/µl; RR: 2.1 - 6.7 × 10^3/µl). Serum biochemistry included marked azotemia (BUN 47 mg/dl and creatinine 9.8 mg/dl; RR: 14 - 21 mg/dl and 1-2.1 mg/dl, respectively), elevated muscle enzymes (CK 1198 iu/l and AST 680 iu/l; RR: 74 - 426 iu/l and 100 - 465 iu/l, respectively), hyponatremia (135 meq/l; RR: 137 - 145 meq/l), hypochloremia (94.0 meq/l; RR: 102 - 114 meq/l), hyperglycemia (148 mg/dl; RR: 80 - 113 mg/dl), and hyperbilirubinemia (7.48 mg/dl; RR: 0.7 - 2.4 mg/dl). Venous blood gas analysis had alkalemia (pH 7.458), decreased ionized calcium (1.36 mmol/l; RR: 1.4 - 1.8 mmol/l), mild hyponatremia (133.3 mmol/l; RR: 137 - 145 mmol/l), and hypochloremia (94 mmol/l; RR: 102 - 114 mmol/l).

Transcutaneous transabdominal ultrasonography of caudoventral abdomen revealed a single live fetus in anterior presentation, with a normal fetal heartrate (73 beats/minutes) and anechoic fetal fluids of subjectively normal volume. Mare's cranioventral abdomen contained ~ 10 - 15 cm of hypoechoic free peritoneal fluid. Remaining ultrasonographic

evaluation was unremarkable. Neither placental separation nor edema was evident on transrectal ultrasonography. Combined thickness of the uterus and placenta (0.99 cm) was within normal limits. Mammary secretions contained 200 ppm calcium (stall-side calcium carbonate test [FoalWatch™ Titrets®, CHEMetrics Inc., Midland, VA]).

Peritoneal abdominocentesis yielded ~ 10 ml of large volume of mildly serosanguinous fluid with increased BUN (70 mg/dl), creatinine (30 mg/dl), and potassium (8.7 meq/l), and decreased sodium (128.0 meq/l) and chloride (92.0 meq/l). Diagnosis was uroperitoneum secondary to suspected urinary bladder rupture.

Treatment

Mare

Mare was hospitalized (day 1) for stabilization prior to exploratory laparotomy and elective Caesarean section. A Foley urinary catheter (Jorgensen Labs, Loveland, CO) and 32-French right cranioventral abdominal drain (Teleflex, Morrisville, NC) were placed to facilitate urine drainage. Intravenous fluid therapy (Ringer's lactate solution [Lactated Ringer's Injection USP, Hospira Inc., Lake Forest, IL]) was initiated at a rate of 1.5 ml/kg/hour. A constant rate infusion ([CRI]; 0.04 mg/kg/minute) of lidocaine (VetOne® Lidocaine 2%, Sparhawk Laboratories Inc., Lenexa, KS) was also initiated. Mare was given intravenously flunixin meglumine ([1.1 mg/kg every 12 hours]; VetOne® Prevail, MWI Animal Health, Boise, ID). To promote fetal maturation, 80 mg of dexamethasone (VetOne® Dexamethasone Injection 2 mg/ml, Bimeda-MTC Animal Health Inc., Cambridge, Ontario, Canada) was given intramuscularly to the mare.

At 24 hours, transcutaneous transabdominal ultrasonography revealed the fetus to be tachycardic (130 beats/minute), consistent with fetal stress.[11] General anesthesia was induced with intravenous ketamine ([3.8 mg/kg]; KETASET® Ketamine HCl Injection, Zoetis Inc., Parsippany, NJ) and intravenous propofol ([0.5 mg/kg]; PropoFlo™ Propofol Injectable Emulsion, Zoetis Inc.) and was maintained on isoflurane inhalant (IsoSol Isoflurane USP, Vedco Inc., St. Joseph, MO). A live filly was delivered via Caesarean section.[12] A 5 cm ventral urinary bladder defect was identified and cystorrhaphy performed.[13] Two 8 - 10 cm and 2 (3 - 5 cm) descending colon mesenteric rents were identified and repaired. A 32-French abdominal drain was placed cranial to the ventral midline incision followed by routine ventral midline incisional closure.

Mild ataxia was noted during anesthetic recovery, and upon standing, mare became pyrexic (39.2 °C), tachycardic, and tachypneic. Repeat serum biochemistry revealed markedly elevated muscle enzymes (CK 64,894 iu/l and AST 1728 iu/l). Urine was collected via urinary catheter, and pigmenturia observed. Mare was treated with external cooling techniques and a single 0.1 mg/kg dose of intravenous morphine (Morphine Sulfate Injection USP, Hospira Inc., Lake Forest, IL). Intravenous fluid therapy (Ringer's lactate [3 ml/kg/hour]) was initiated and lidocaine CRI was reinitiated (0.04 mg/kg/minute). Penicillin G procaine ([2.2×10^4 iu/kg every 12 hours]; VetOne® PenOne Pro™ Injectable Suspension USP, Vedco Inc.) was given intramuscularly and gentamicin ([6.6 mg/kg every 24 hours]; VetOne® Gentamicin Sulfate Solution, MWI Animal Health) was given intravenously. Mare was also given intravenously 5 IU of oxytocin IM (VetOne® Oxytocin Injection, Bimeda-MTC Animal Health Inc.) every 2 hours to promote fetal membrane expulsion. Overnight, the mare remained bright and alert, demonstrating good appetite and interest in her foal.

Daily abdominal lavage was performed through the indwelling abdominal drain. Uterine lavage (~ 30 - 40 liters [5 - 10 liters per instillation]) of nonsterile 0.9% saline was performed, releasing a portion of the chorioallantois from the caudal uterus, and was repeated twice daily until resolution of the retained fetal membranes. Intramuscular oxytocin treatment was increased to 10 IU (0.014 IU/kg every 2 hours). A small weight was affixed to the chorioallantois to provide passive traction. Intravenous fluid therapy was decreased to 1.5 ml/kg/hour and CRI of lidocaine was discontinued.

At 30 hours postoperatively, mare displayed tachycardia (60 beats/minute) with diffuse muscle fasciculations. A nasogastric tube was passed with no net reflux and a CRI of lidocaine (0.04 mg/kg/minute) was reinitiated; after treatment mare appeared more comfortable and passed adequate feces. On day 4 (48 hours postoperation), fetal membranes were still retained, and prophylactic ice boot therapy was initiated (Cordura Ice Boots, Jacks' Manufacturing, Washington C.H., OH). By day 5, the mare appeared comfortable with normal vital parameters. Intravenous fluid therapy and the CRI of lidocaine were again discontinued.

On day 6, fetal membranes were completely removed. Approximately a 20 cm in diameter, roughly circular, soft, fluctuant mass acutely developed cranioventral and craniolateral to the left flank with an ultrasonographic appearance was consistent with acute subcutaneous hematoma and no evidence of abdominal herniation or body wall muscle rupture. Abdominal drain was removed, and a hernia belt (CM Equine Heal Belt, CM Equine Products, Norco, CA) was placed over the caudal abdomen encompassing the mass. Eighteen hours after initial observation, it was noted that the mass had increased in size cranially. Intravenous fluid therapy with Ringer's lactate solution (3 ml/kg/hour after 10 liters bolus) and a CRI of lidocaine (0.04 mg/kg/minute) were reinitiated. All other treatments continued as described.

On day 8, the hematoma in the left flank had increased in size to encompass left stifle, and mare was markedly painful and reluctant to ambulate. That evening, mare became acutely painful, tachycardic, and displayed colic signs. Butorphanol tartrate ([0.02 mg/kg]; Torbugesic®, Zoetis Inc.) was given intravenously with no resolution of clinical signs. Abdominocentesis yielded opaque, yellow peritoneal fluid with an elevated lactate (5.6 mmol/l). Transcutaneous abdominal ultrasonography and transrectal palpation findings were unremarkable and nasogastric intubation yielded 4 liters of spontaneous reflux. Colic signs continued and morphine (0.13 mg/kg) was given intramuscularly, to provide comfort throughout the night.

Foal

Filly was initially fed via indwelling nasogastric tube due to signs of mild neonatal maladjustment syndrome and was pro-

phylactically treated intravenously with ceftiofur sodium ([5 mg/kg every 12 hours] Naxcel® Ceftiofur Sodium Sterile Powder, Zoetis Inc.). Foal was given 3 liters of equine plasma (ImmunoGlo™ 1700 Normal Equine Plasma, MG Biologics Inc., Ames, IA). Due to mare's declining health, filly was separated and successfully grafted to a nurse mare.

Outcome

Mare

On the morning of day 9 (7 days after surgery), mare became acutely painful, collapsed, and died following suspected cardiovascular collapse. Necropsy revealed ~ 35 cm of small intestinal loops protruding from ventral midline incision with dark serous to hemorrhagic fluid drainage. Serosal surfaces of all abdominal viscera were hyperemic and coated in fibrinous mats. Subcutaneous tissue in the region of the left flank was markedly expanded by clear, glistening fluid. Approximately 40 cm in diameter in the region of left lateral body wall contained muscle fibers that were extensively frayed and separated by hemorrhage and edema. Caesarean section closure was intact, with minimal fetid fluid and fibrinous material within uterine lumen. Cystorrhaphy site was intact. On histopathology, the left thigh subcutis contained severe hemorrhage and vasculitis, with occasional fibrin thrombi and degenerate neutrophilic/lymphocytic infiltrates with fewer macrophages in the surrounding tissue.

Foal

Filly remained systemically healthy throughout hospitalization, with no clinical evidence of pulmonary prematurity, and was discharged at 14 days of age. At the most recent communication with the filly's owner, the filly was ~ 8 months old and reported healthy.

Discussion

This report described a case of uroperitoneum secondary to bladder rupture in a late-term pregnant mare not associated with parturition. Urinary bladder rupture in adult horses has been reported following trauma,[10] obstructive urolithiasis,[8,9] and parturition in mares.[2,4-7] It is generally believed that the mechanism by which the urinary bladder in foaling mares ruptures during parturition is either acutely by impingement of a distended bladder between the rim of mare's pelvis and foal during forceful abdominal contractions or several days after parturition by necrosis of the urinary bladder wall following an ischemic, traumatic injury sustained during foaling.[4] To the authors' knowledge, this is a first case report that described a ruptured urinary bladder in a late-term pregnant mare not associated with parturition and without history or evidence of trauma or lower urinary tract obstruction.

Uroperitoneum secondary to a bladder wall rupture was suspected after hematologic and peritoneal fluid analysis with confirmation at surgery. Without history of external trauma, observation of external trauma clinical signs, or ante or postmortem evidence to support a urinary obstruction that may have led to chronic overdistension of the bladder in this patient, an explanation for the bladder rupture could be that impingement of the bladder wall between bony structures of the mare and foal combined with possible fetal movement caused it to tear acutely. Location of the defect in this mare may further support this theory, as it was on the ventral aspect of the bladder that is commonly the location of bladder wall tears suspected to occur during foaling.[2,5,6] It is possible that while the mare was displaying colic signs prior to presentation, some combination of abdominal straining and rolling produced an intraabdominal pressure comparable to that generated during parturition in foaling mares and that the bladder became compromised in a similar way. Impingement leading to an acute tear of the bladder wall seems more likely than one resulting from ischemic necrosis in this case, as there was no evidence of devitalized bladder tissue noted during surgery or on necropsy 7 days later.

This case also demonstrated the use of a single dose of dexamethasone at ~ 340 days of pregnancy to promote fetal maturity and survivability of a near-term foal. Duration of pregnancy in Thoroughbred mares ranged from 306 to 390 days with a mean length of 347 days.[14] Approximately 5 days prior to parturition in horses, maturation of the fetal hypothalamic-pituitary-adrenal axis and a marked rise in fetal cortisol concentrations are responsible for a final wave of maturation of multiple organ systems that prepare the foal for extrauterine life.[15,16] Maternal dexamethasone treatment has been used to mimic this rise in cortisol and hasten fetal maturity prior to induced parturition or Caesarean section, to improve foal survivability in the postnatal period; however, reduced postnatal growth rate or adrenal suppression have sometimes been reported after corticosteroid use after 320 days of pregnancy.[17,18] A typical protocol is to give 100 mg of dexamethasone intramuscularly to the mare once daily for 3 - 4 days after 320 days of pregnancy; however this is not always possible to achieve in cases where the mare's or fetus's clinical condition acutely worsens or warrants immediate delivery.[17,18] Because 340 days is within the normal range of pregnancy in Thoroughbred mares, and without prior foaling history, it cannot be proven that the dexamethasone received by the mare in this case report contributed substantially to her filly's physiologic maturity or postdelivery survival; however, no adverse effects on the foal's short-term health were observed. Apparently, maladaptive neonatal maladjustment syndrome diagnosed in this filly was not associated with maternal dexamethasone treatment, but rather due to the immaturity of the hypothalamic-pituitary-adrenal axis and hypoxemic stress reported in premature foals and those delivered via Caesarean section, respectively.[19-21] Thus, a single intramuscular injection of dexamethasone at a dose of 100 mg between 335 to 340 days of pregnancy may hasten fetal maturity and improve postnatal survivability; however, further study is needed to validate this protocol.

In summary, this case demonstrated the importance of including bladder rupture as a differential diagnosis for late-term, preparturient pregnant mares presenting with a history of colic signs or abdominal straining and that a single intramuscular injection of dexamethasone may be considered to safely hasten fetal maturity prior to Caesarean section.

Learning points

- Bladder ruptures occur in late-term pregnant mares, even prior to parturition.
- Behavioral colic in late-term pregnant mares may predispose to acute bladder injury via increased abdominal pressure and bony impingement of the bladder wall.
- Dexamethasone (80 mg) given intramuscularly12 hours prior to Caesarean section between 335 - 340 days of pregnancy had no adverse effects on the foal.

Conflict of interest

Authors have no conflicts of interest to declare.

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