Standing surgical repair of a ruptured bladder in a broodmare

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Introduction

Rupture of the urinary bladder is well-recognised in newborn foals but is rare in adult horses. It has been described as an uncommon complication in periparturient mares, with a reported incidence of 0.01%.

Clinical signs are often vague and may take up to five days post-rupture to appear, making diagnosis and early intervention challenging. Without diagnosis and management, a ruptured bladder will lead to haemoconcentration, azotaemia, electrolyte imbalances, and death.

While a ruptured bladder is relatively easily accessed via a conventional midline laparotomy in neonatal foals, in adult horses surgical repair is much more challenging. A number of different surgical methods to repair ruptured bladders in adult horses have been reported, including direct or laparoscopic repair both under general anaesthesia and with standing sedation.

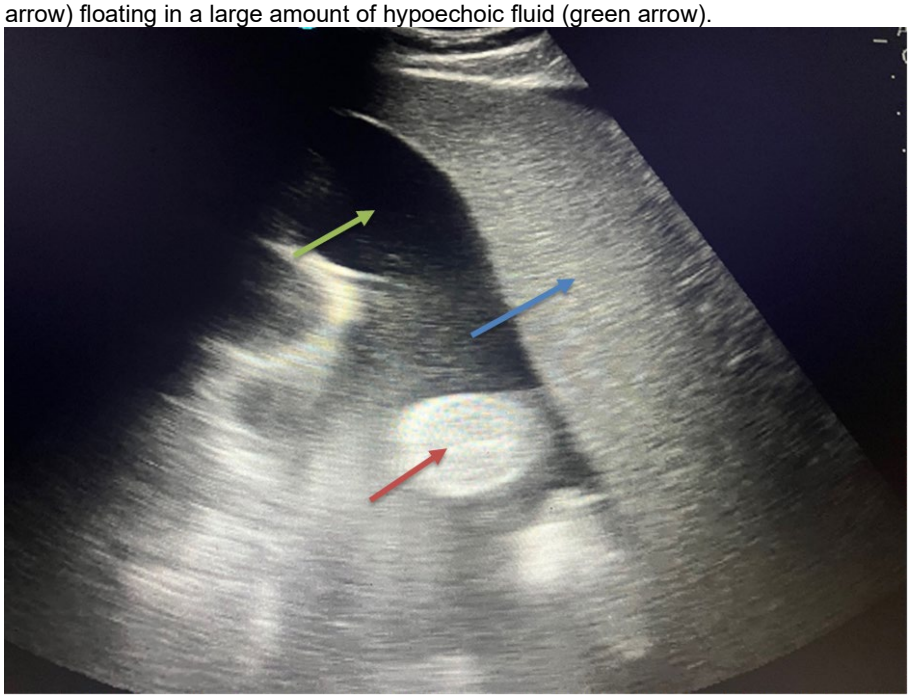
History and clinical signs

A 10-year-old multiparous Thoroughbred broodmare was initially seen in the field for signs of abdominal discomfort/colic close to foaling. At that time she presented with a dull demeanour, reduced faecal output, mild tachycardia (48 bpm), inappetence, reduced water intake and reduced urinary output. Differential diagnoses included idiopathic peri-parturient abdominal discomfort, uterine torsion, uterine artery haemorrhage, uterine tear, peritonitis, large intestinal displacement/torsion, enteritis/colitis, and bladder rupture. A complete blood count (CBC) revealed a mild neutrophilic leucocytosis ($8.0x10^9$ cells/L, reference range $2.5\text{-}6.9x10^9$ cells/L); all other parameters including blood fibrinogen and lactate were within normal range. The mare was treated with anti-inflammatory drugs (Flunixin meglumine, 1.1mg/kg IV SID) and oral fluids via naso-gastric tube as required and monitored closely. She foaled normally 48 hours following initial presentation but deteriorated post-foaling, presenting with injected mucous membranes, reduced capillary refill time, dehydration, and tachycardia with an irregularly irregular cardiac arrhythmia.

Diagnosis

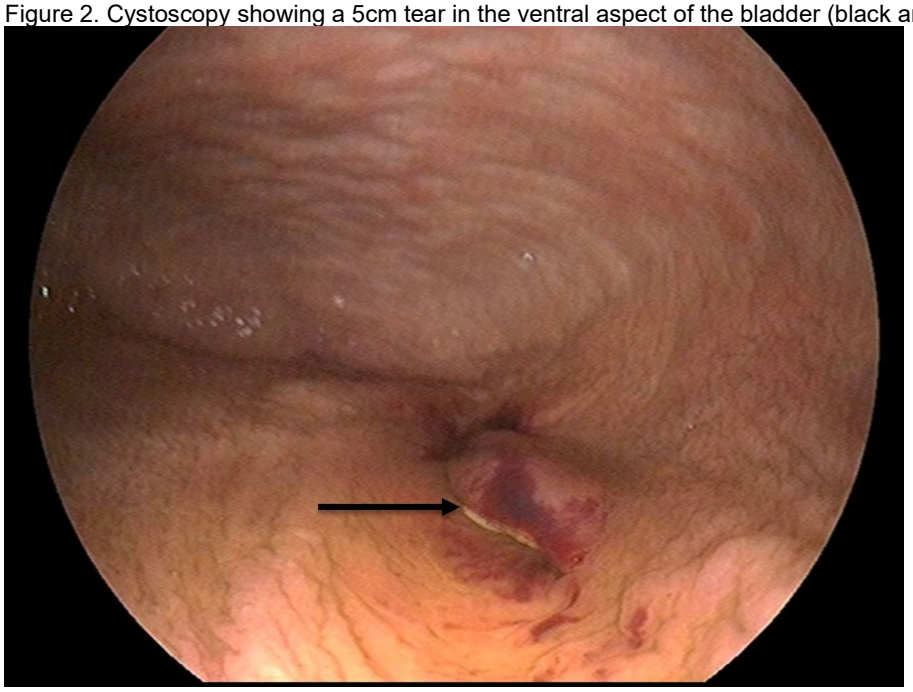
A repeat CBC revealed severe dehydration with a PCV of 66%. Blood biochemistry abnormalities included elevated blood urea (31.2mmol/L, reference range 3.9-9.6mmol/L) and serum creatinine (598umol/L, reference range 35-195umol/L), as well as severe hyperkalaemia (7.7mmol/L, reference range 1.9-4.1mmol/L). Abdominal and rectal ultrasound examination revealed a large amount of hypoechoic fluid within the abdominal cavity (Figure 1) and the presence of a small irregular bladder. Abdominocentesis yielded clear pale-yellow fluid with a peritoneal creatinine level of 3028umol/L, which was five times the blood creatinine level.

Figure 1. Abdominal ultrasound image showing the spleen (blue arrow) and a loop of small intestine (red arrow) floating in a large amount of hypoechoic fluid (green arrow).



Endoscopic examination of the bladder was performed via the urethra, revealing a small bladder with minimal urine content and a 5cm long, full-thickness tear along the ventral bladder wall (Figure 2).

Figure 2. Cystoscopy showing a 5cm tear in the ventral aspect of the bladder (black arrow).



A diagnosis of urinary bladder rupture was made, and the mare was referred to the hospital for medical treatment and surgical repair.

Treatment

Pre-surgical management

An intravenous catheter was placed and the mare was started on intravenous fluid therapy with glucose and a continuous-rate infusion of insulin to reduce blood potassium levels prior to surgery. An abdominal drain was placed just lateral to the ventral midline and approximately 30L of urine was drained slowly from the abdominal cavity over approximately one hour. The drain was left in place with a one-way valve to prevent air intake and allow passive drainage of urine from the abdomen as the mare moved around. The aim was to stabilise the mare's systemic parameters prior to general anaesthesia to repair the bladder rupture. However, although the mare appeared brighter over the next 24 hours her blood potassium level did not improve significantly, and the decision was made to attempt a standing surgical repair on the bladder through a urethrostomy and urethral sphincterotomy.

Surgical technique

The mare was sedated (detomidine 0.01mg/kg and Butorphanol 0.01mg/kg IV) and placed in stocks. Caudal epidural anaesthesia was administered (75mg xylazine and 4ml of 2% mepivacaine made up to 10ml volume with sterile saline), and a longitudinal incision was made through the external urethral sphincter and urethra. A hand was inserted into the bladder lumen through the incision and the bladder was manually everted into the vagina using gentle traction. The rent in the ventral aspect of the bladder was identified and closed using two layers of absorbable suture in a simple continuous appositional pattern. The bladder was repositioned, and the urethral incision was closed the same way. A urinary catheter was placed post-surgery to prevent bladder distension.

Post-surgical management

After surgery the mare was maintained on intravenous fluids, broad-spectrum antimicrobial drugs (Gentamicin 8mg/kg IV SID, Ceftiofur 2.2mg/kg IV BID and Metronidazole 10mg/kg PO BID) and anti-inflammatory therapy (Flunixin Meglumine 1.1mg/kg IV SID). By 24-hours post-surgery, the mare's blood electrolytes, urea and creatinine had returned to the normal range, and she was urinating freely through the urinary catheter. CBC revealed a significant left shift with a low total white cell count ($3.8x10^9$ cells/L, reference range $4.9\text{-}11.1x10^9$ cells/L) and the presence of immature neutrophils (band cells), and an elevated serum amyloid A (SAA) consistent with peritonitis.

Over the following days, the mare became much brighter, and her appetite improved significantly. Repeat abdominal ultrasound examination performed 48 hours post-surgery revealed no free fluid in the abdomen and the ventral abdominal drain was removed. The urinary catheter was removed 72 hours post-surgery. The mare was discharged from the hospital six days post-surgery and antimicrobial treatment (Trimethoprim-sulphonamide 30mg/kg BID PO and Metronidazole 10mg/kg BID PO) was continued for a further five days until WCC and SAA normalised. There were no further complications and the mare was successfully re-bred later in the season.

Discussion

The history in this case suggests that the bladder rupture occurred 48 hours before the mare foaled when she was showing signs of abdominal discomfort. Given the rarity of this condition and the non-specific early clinical signs the diagnosis of a ruptured bladder was delayed until changes in blood parameters were marked. This highlights the need for careful consideration of a ruptured bladder as a differential diagnosis in peri-partum mares with low-grade colic.

Having failed to normalise the blood potassium level pre-surgery it was deemed appropriate to perform the surgery without general anaesthesia to avoid the risk of associated atrio-ventricular conduction disturbances. There are several case reports of standing surgical repair of bladder ruptures in adult mares. By performing a urethrotomy and urethral sphincterotomy and everting the bladder into the vagina the tear in bladder was able to be visualised and repaired using minimal specialist equipment. However, performing a sphincterotomy or urethrotomy carries the inherent risk of post-surgical urovagina or incontinence. Other surgical methods such as standing laparoscopic repair or transurethral endoscopic-guided intraluminal closure of bladder tears provide more minimally invasive techniques without the need for sphincterotomy or urethrotomy but these require specialist equipment and proficiency in laparoscopic suturing.

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