Bladder rupture in the adult horse

Early and accurate diagnosis of cystorrhexis, coupled with appropriate and prompt medical and surgical treatment, will allow the majority of adult horses to make a full recovery, write Turlough McNally MVB Diplomate ACVS Diplomate ECVS MRCVS, Anglesey Lodge Equine Hospital, The Curragh, Co Kildare, and Nikki Walsh MVB, Greenmount Equine Hospital, Limerick

Cystorrhexis (rupture of the urinary bladder) occurs most commonly in foals, with a reported incidence of 2.5%.[1] In contrast, it is considered an uncommon observation in the adult horse; in mares, cystorrhexis occurs with an incidence of 0.01%, or approximately one in 10,000 births.[2] Reported predisposing factors include parturition,[3,4,5] urethral obstruction,[6] trauma,[7,8] and following neonatal infection or bladder surgery that compromised the bladder wall.[9,10] Despite this being an uncommon condition, early and accurate diagnosis coupled with prompt intervention is crucial to a successful outcome.[11]

AETIOLOGY

Parturition is the most common cause of cystorrhexis in the adult horse. It has been proposed that the pathogenesis of cystorrhexis during parturition involves compression of the bladder between the foal and the pelvic brim,[3,12] or occlusion of the urethra causing an increased intravesicular pressure and subsequent rupture.[3,13] However, dystocia is not necessarily a predisposing factor, with seemingly normal foalings resulting in cystorrhexis.[2] Although acute tears to the bladder wall are the most commonly recognised form of urinary bladder compromise in the immediate postpartum period, urinary bladder rupture may occur some time after parturition. Trauma to the bladder wall during parturition may result in necrosis of the bladder wall, ultimately leading to rupture. A single case of bladder wall rupture was reported and proposed to be associated with rupture of the internal pudendal artery during parturition and subsequent ischaemic necrosis and rupture some time later.[12] Interestingly, a case of a bladder wall tear in association with a uterine torsion pre-partum has been described;[4] therefore, bladder rupture should always be considered as a differential diagnosis when evaluating peri-partum colic in the mare.

Urethral obstruction may also result in urinary bladder rupture. This is quite rare in females due to the relatively shorter and wider urethra; however, it is well documented in males, particularly geldings, without any specific breed predilection.[6,14] Uroliths are a common cause of urethral obstruction; typically, uroliths are formed within the bladder and may move distally along the urethra where they can become lodged as it narrows. Approximately 90% of uroliths are composed primarily of calcium carbonate ($CaCO_3$), are yellow or green, and may be quite spiculated on their surface (type 1 uroliths). I have removed small uroliths from immediately proximal to the urethral process in numerous geldings presenting with signs of colic.

Neoplasia may also result in urinary outflow obstruction and consequent bladder rupture. Squamous cell carcinomas (SCCs) are the most common external genital neoplasm in the horse and have been reported to cause urethral obstruction. Squamous cell carcinomas are commonly associated with mucocutaneous junctions, particularly those with little or no pigmentation. Typically, the tumour invades the urethra causing necrosis and narrowing of the lumen.

Dissecting penile haematomas have also been observed to obstruct the urethra in stallions following penile trauma.[14] External abdominal trauma resulting in a sudden increase in intra-abdominal pressure, such as that experienced during recovery from general anaesthesia, running through a fence, road traffic accident or following a fall, may also result in urinary bladder rupture. Severe abdominal trauma was implicated as the cause of not only bladder rupture but also infarction of the colon in a filly that crashed through a fence.[7]

Some authors have postulated that weak scar tissue from a previously ruptured bladder repair as a neonate predisposed an adult horse to cystorrhexis.[9] It has also been speculated that previous urachal infection treated conservatively as a foal could have compromised the apex of the bladder, resulting in subsequent rupture.[10] Irrespective of the cause, the adult bladder usually ruptures on the dorsal surface in close proximity to the

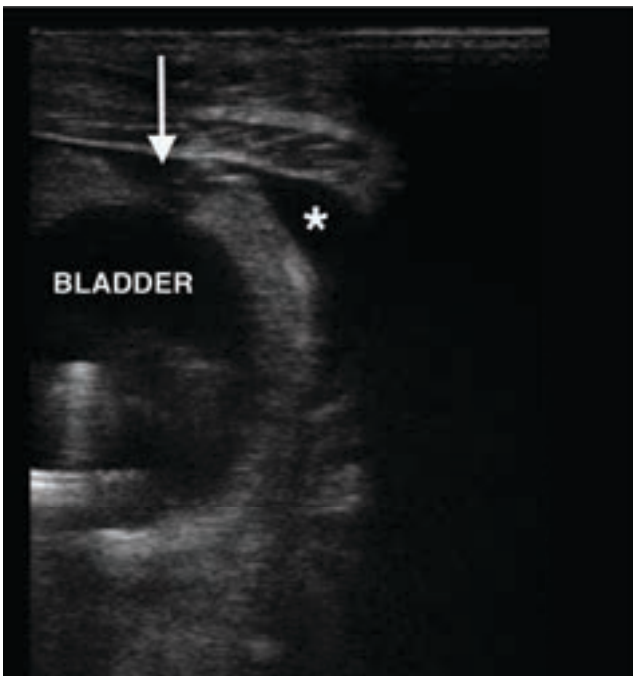

Figure 1: Ultrasonographic image obtained using a 7.5MHz linear transducer. The arrow denotes a rent in the bladder wall and the asterix denotes peritoneal free fluid which was confirmed to be urine. Ventral is to the top of the image. Courtesy: KT Corley.

apex as this is a natural weak point where the layers of the bladder wall cross each other; however, ventral tears may also occur.

CLINICAL SIGNS

The presenting clinical signs associated with urinary bladder rupture are a result of uroperitoneum and the ensuing electrolyte derangements. The signalment, combined with a full and thorough clinical examination, will often reveal findings which may rouse the clinician's suspicion for urinary tract compromise. Some cases present acutely;[7,8] however, often the clinical signs only manifest three to five days post insult.[2,5,10] Patients become depressed, anorectic and develop progressive abdominal distension.[3] Unless the primary cause is urethral obstruction, pollakiuria and stranguria with the production of small quantities of urine is a common characteristic, with anuria seen uncommonly.[9] Individuals with obstructive disease of the urethra may present with signs of colic. Unlike in neonates, uroperitoneum in adult horses results in peritionitis either due to chemical irritation of the peritoneum causing inflammation and/or from a bacterial cystitis seeding the abdominal cavity.[3,6,7] Peritonitis may be associated with signs of ileus, decreased faecal output and mild colic. These signs, combined with depression, are usually the first abnormalities noted by owners.[15] Horses with obstructive urethral disease may also present with mild-to-moderate signs of colic.

Increases in the extracellular fluid volume and the resultant hypovolaemia precipitate tachycardia, congested mucus membranes and a prolonged capillary refill time.[3,15] Increased intra-abdominal pressure, due to excess peritoneal free fluid, compromises cardiopulmonary function resulting in tachypnoea.[3] Abdominal distention from uroperitoneum may be difficult to appreciate in the adult horse, particularly in the post-partum mare; however, an attempt should be made to ballott the abdomen to detect the presence of peritoneal free fluid. Metabolic derangements such as hyperkalaemia can present as neurological deficits, muscle fasciculations and cardiac arrhythmias in severe cases.[16] Observed cardiac arrhythmias include bradycardia, third-degree atrioventricular node blockade and ventricular fibrillation.

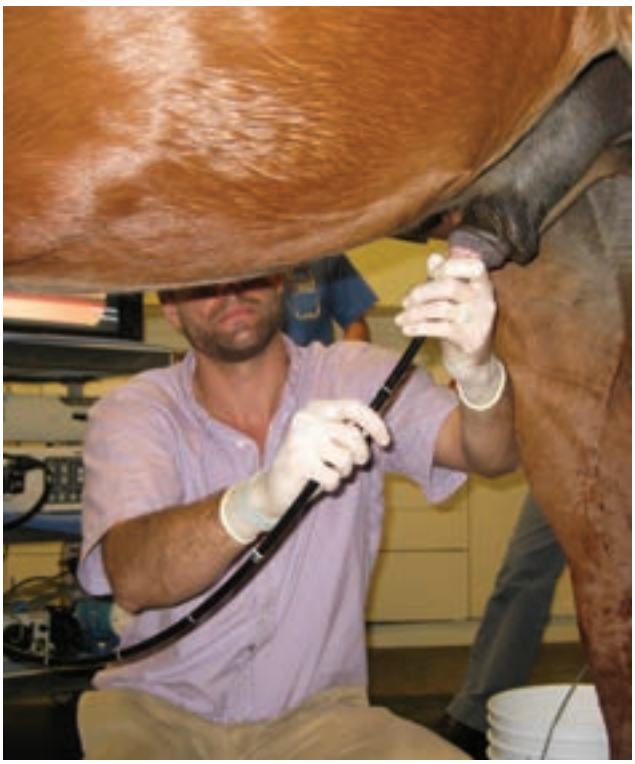

Figure 2: Cystoscopic examination of a gelding with suspected cystorrhexis.

DIAGNOSIS

A full haematological and serum biochemistry analysis is an essential first diagnostic step when evaluating horses with urinary tract disorders. The peritoneal membrane allows relatively free movement of small molecules, including electrolytes. If left untreated, uroperitoneum results in haemoconcentration which may be identified on a haemogram as an increase in packed cell volume (reference range [rr]: 37.6-51.9%) and increased serum total protein values (rr: 58-68.5g/L). Leukopaenia (rr: 5-8.6 x 10^9/L) may also be evident if a significant peritonitis has established; however, concurrent disease processes may significantly alter the leukogram, in which case the signalment should also be taken into consideration, for example a post-partum mare versus a gelding. Serum amyloid A (rr: 0-5mg/L) will be elevated early in the disease process with fibrinogen (rr: 0.82-2.3g/L) becoming elevated in the days following cystorrhexis.

Metabolic abnormalities occur commonly due to the equilibration of electrolytes between the plasma and urine across the peritoneal membrane.[3] The more consistently

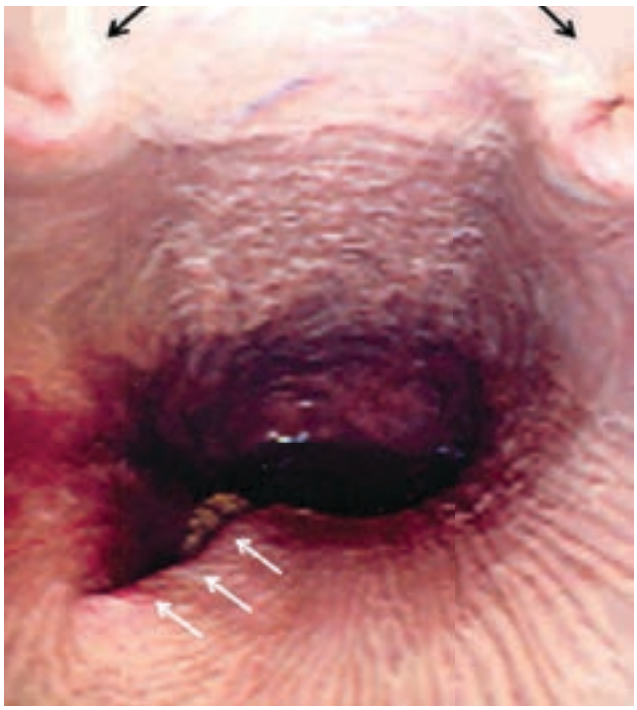

Figure 3: Cystoscopic image of the same horse in Figure 2. The black arrows denote the ureteral openings into the bladder and the white arrows outline a full thickness tear in the ventral bladder wall.

reported biochemical abnormalities include hyponatraemia, hypochloraemia, hyperkalaemia and azotaemia.[2,3,11,15] Severe hyponatraemia may lead to hyperaesthesia and seizures; however, abnormalities in serum concentrations of potassium can pose a more immediate threat to life. Discrepancies in findings of potassium have been recorded, with Genetzky and Hagemoser reporting hyperkalaemia and Highuchi et al, 2012 reporting no abnormalities in serum potassium concentrations in four mares with uroperitoneum. These differences may be accounted for by advances in diagnostic techniques, such as transabdominal ultrasonography, that have afforded a more expedient diagnosis of cystorrhexis prior to the development of significant metabolic abnormalities. Potassium is primarily an intracellular ion that acts to raise cell resting membrane potential and bring it closer to the threshold potential. Moderate increases in serum potassium levels (rr: 2.9-4.0mmol/L) lead to increased cell excitability that may be recognised clinically as muscle fasciculations, muscle weakness or cardiac arrhythmias, such as third-degree atrioventricular block and premature ventricular beats. Severe hyperkalaemia (>6.5mmol/L) further exacerbates cardiac arrhythmias and may result in ventricular fibrillation and ultimately cardiac arrest and asystole. An electrocardiogram is an excellent adjunctive diagnostic aid in the presence of any cardiac rhythm or rate disturbance that may be associated with hyperkalaemia. Recognised abnormalities include reduced P wave amplitude, increased P wave duration, a prolonged P-R interval, increased QRS complexes, shortened QT interval and increased T wave amplitude. Azotaemia is recognised as an elevation in serum urea nitrogen and creatinine concentration (rr: 3.86-7.82mmol/L and 98-182µmol/L, respectively).

Abdominal ultrasonography is a quick and relatively easy diagnostic technique to identify abnormalities within the peritoneal cavity.[11] For optimal image quality, the area to be imaged should have the hair clipped using a number 40 surgical blade and be thoroughly cleaned. I typically use a 2-5MHz convex transducer which can achieve a depth of 30cm. In the adult horse, the urinary bladder is located at the level of the pelvic brim and when empty it is usually obscured from view by gas in the large and small colons. As the bladder fills with swirling echogenic urine, it extends cranially and ventrally and can typically be imaged trans-abdominally through the ventral and caudal abdomen. If the ultrasonographer experiences difficulty in imaging the bladder, a transrectal approach may be more successful.[3] In the case of cystorrhexis the uncatheterised bladder may be difficult to image using both techniques; when identified, the bladder wall can be recognised as a collapsed and infolded structure, with the lumen either devoid of or containing minimal urine. Some clinicians have utilised agitated saline introduced through a urinary catheter to identify the actual site of rupture (Figure 1). The presence of voluminous quantities of hypoechoic peritoneal free fluid combined with a collapsed bladder is highly suspicious of cystorrhexis. If secondary peritonitis has developed, strands of fibrin may be visualised floating in the peritoneal fluid and lining the peritoneal structures. It is important to note that ultrasound is not highly specific in differentiating the different forms of peritoneal free fluid (urine, blood or transudate) and therefore diagnostic samples should be obtained to confirm its composition. Abdominocentesis/abdominal paracentesis is a method of trocarising the abdominal wall in order to obtain a sample of peritoneal free fluid for diagnostic purposes.[11,15] Historically, this can be performed blindly through the cranial and most dependent portion of the abdomen, just to the right of midline. I prefer an ultrasound-guided technique as this increases the chances of a successful yield. Once a suitable location is identified under ultrasound guidance in the standing horse, a 10cm^2 area is aseptically prepared before the region is desensitised using 2ml of 2% mepivacaine hydrochloride. A stab incision is made through the skin and external rectus sheath using a number 15 blade and a sterile 7.5cm long teat cannula (passed through a sterile swab to prevent blood contamination of the sample) is introduced into the peritoneal cavity. Samples should be obtained for cytology, biochemical analysis and bacterial culture and sensitivity. The retrieved fluid may have a urine-like appearance[2,7,13] and ammoniacal smell.[16] Direct microscopic examination may reveal the presence of $CaCO_3$ crystals, nucleated cells, intra and extracellular bacteria and fibrin strands. The peritoneal membrane does not facilitate diffusion of large creatinine molecules and, therefore, creatinine accumulates within the peritoneal cavity within 24 hours of cystorrhexis.[15] Classically a peritoneal: serum creatinine ratio of greater than two is regarded as diagnostic for uroperitoneum. The fluid should also be analysed for nucleated cell count (normal <10,000µL), total protein

Figure 4: Semi-closed passive abdominal drain to allow continuous drainage of urine and peritoneal lavage.

(normal <2.5g/dL), glucose (normal <30mg/dL), fibrinogen (normal <2.3g/L) and a blood to peritoneal fluid lactate difference (normal <2mmol/L) to determine if any significant peritonitis co-exists.

In adult horses, cystoscopic examination of the bladder can be performed using a 1.9m x 12mm flexible fibreoptic endoscope in adult horses (Figure 2). In cases of suspected bladder rupture, it is essential to sterilise the endoscope prior to use to reduce the introduction of bacteria into the bladder and consequentially into the abdominal cavity.[11] The bladder should be drained of urine using the biopsy channel and insufflated with air. However, with bladder wall defects, distension with air to achieve a thorough examination can be difficult[4] which is the main limitation of the procedure. Tears have been visualised in several cases throughout the literature[7,8,10] and areas of bruising or necrosis can also be identified and monitored (Figure 3).[10,12] Cystoscopy allows for follow-up examination and monitoring of healing, particularly when conservative treatment is elected.[5]

TREATMENT

The mainstay for treatment of uroperitoneum and cystorrhexis has been drainage of the peritoneum and surgical repair of the bladder wall, respectively. Prior to any surgical intervention it is essential that any metabolic derangements and concominant pathologies are addressed prior to general anaesthesia. The use of broad-spectrum antibiotics and non-steroidal anti-inflammatory drugs are usually appropriate; choices vary depending on the attending clinician's personal preference.[3,5] Hyperkalaemia is the most important metabolic abnormality that requires treatment prior to general anaesthesia with the goal of decreasing serum potassium levels to <5.5mmol/L, thus reducing the risk of fatal cardiac arrhythmia. For a serum potassium <6.5mmol/L, fluid therapy using potassium-free intravenous fluids, such as 0.9% sodium chloride, will promote kaliuresis. This fluid may be supplemented with 5-10% dextrose solution or, alternatively, a 50% dextrose solution (2ml/kg/IV) administered over five minutes if necessary. Dextrose will result in the endogenous release of insulin which stimulates sodium/potassium-ATPase pumps to drive potassium intracellularly. For hyperkalaemia >6.5mmol/L or if a cardiac arrhythmia is detected, intravenous calcium supplementation (40% calcium gluconate [0.5ml/kg/IV] over 10 minutes) may be indicated. Calcium supplementation raises cellular membrane threshold potential and thus antagonises the negative myocardial effects of hyperkalaemia. While calcium is cardioprotective, it will in itself not act to decrease the serum potassium. In severe or non-responsive cases, the use of exogenous insulin or sodium bicarbonate may be warranted however this is rarely utilised in the adult horse and is usually reserved for critical neonates.[3,16]

Along with intravenous therapy for correction of serum potassium, urine should be removed from the abdomen as soon as possible to help correct systemic electrolyte derangements[17] and decrease the chemical irritation to the peritoneum.[15] Urinary diversion and peritoneal drainage and lavage can be successfully performed via placement of a transurethral urinary catheter and an abdominal drain inserted under standing sedation. To provide urinary diversion, a 32 French Foley catheter is placed trans-urethrally into the urinary bladder using sterile technique and the cuff is inflated with 30ml of sterile water. Subsequently, a 32 French chest tube is placed into the peritoneal cavity at the ventral most aspect of the abdomen, 5cm to the right of midline, as described by Reuss et al, 2006 (Figure 4). The abdomen should be completely drained of urine and subsequently lavaged using 10L of sterile lactated ringers solution up to twice daily. If necessary, peritoneal dialysis using a solution of 1.5% dextrose in a balanced electrolyte fluid creates a hypertonic dialysate that has been shown to result in ultrafiltration.[18] This allows for diffusion of urea nitrogen, creatinine and other elevated electrolytes from the bloodstream into the peritoneal dialysate which is then drained. Removal of large volumes of abdominal fluid can result in hypovolaemic shock.[2] Packed cell volume and serum total protein should be monitored frequently to evaluate hydration and electrolyte status should be assessed to permit adjustment of the rate and type of intravenous fluids to restore serum electrolytes to normal levels, particularly when peritoneal dialysis is also performed.

To date, most reported treatments of traumatic cystorrhexis in adult horses have involved some form of surgical repair of the bladder wall defect.[2,4,13,16,19,20,21] Exteriorisation and surgical repair of the bladder in adult horses through a ventral coeliotomy can be difficult due to its intra-pelvic location and the depth of the adult equine abdomen.[6,13,17] Adequate exteriorisation may sometimes even be impossible, particularly if the location of the tear is near the neck of the bladder.[4] Additionally, general anaesthesia carries inherent risks, particularly in the periparturient broodmare. These difficulties have led to the development of standing surgical approaches for repair of ruptured bladders which attempt to afford

the surgeon greater access to the area of interest.[4,13,20] Standing surgical repair techniques have also been associated with specific challenges and post-operative complications. These include urine pooling and dribbling after urethral sphincterotomy,[19] and complications with wound healing after incising and suturing damaged vaginal mucosa in post-parturient mares.[13] Documented accounts of successful conservative management in horses have been sparse, with one gelding managed with the aid of a perineal urethrotomy,[6] one colt foal managed with an indwelling urinary catheter[22] and one mare in which the tear was experimentally created.[15] Spontaneous healing of dorsally located bladder tears following placement of a transurethral urinary catheter has been reported in mature cows.[23] Another study reported successful medical management of cats following the creation of iatrogenic rents in the bladder wall[24] and in another, three of 14 dogs with experimentally induced cystorrhexis had successful repair recorded within 45 hours with medical management only.[25] More recently, we have reported on successful management of cystorrhexis in four adult horses using trans-urethral and trans-abdominal catheterisation.[5] Catheters in these patients continually diverted urine, decreasing tension on the bladder wall, reducing the amount of urine accumulating within the peritoneal space and allowing the bladder walls to heal by second intention. As of yet, we have been unable to determine the time period required for second intention healing to occur in the horse or whether the location of the tear in the bladder wall determines its ability to heal with conservative treatment. Conservative treatment may be of interest where owner financial constraints exist; however, given the paucity of scientific evidence, surgical repair of cystorrhexis in the adult horse remains the current gold standard.

In conclusion, early and accurate diagnosis of cystorrhexis, coupled with appropriate and prompt medical and surgical treatment, will allow the majority of adult horses to make a full recovery.

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