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Case Report

Conservative management of iatrogenic bladder rupture and uroperitoneum in a gelding with urolithiasis

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Summary

This report details a case of iatrogenic bladder rupture in a gelding that was successfully treated with conservative management. Rupture occurred at the time of attempted cystoscopy, whilst the bladder was being insufflated with air for further investigation of stranguria and suspected urolithiasis. The gelding was treated with temporary perineal urethrostomy, peritoneal lavage and drainage of urine from the bladder and made a complete recovery.

Introduction

Bladder rupture has a reported prevalence of 0.2–2.5% (Kablack *et al*. 2000) in foals but is rare in adult horses. A prevalence of 0.01% has been reported in broodmares (Higuchi *et al*. 2002), and there have been reports in male horses secondary to urolithiasis (Gibson *et al*. 1992; Walesby *et al*. 2002) or urethral stricture caused by a squamous cell carcinoma (May *et al*. 2008). Conservative management of uroperitoneum in mares has been reported (Gibson *et al*. 1992; Peitzmeier *et al*. 2016); however, surgical repair of bladder tears is advocated (Schott and Woodie 2012; Snalune and Mair 2006) to reduce uroperitoneum which is associated with potentially fatal chemical and septic peritonitis and electrolyte imbalances (Pye *et al*. 2018). Walesby *et al*. (2002) reported a horse with a bladder tear that developed marked chemical peritonitis despite medical management with peritoneal lavage, prompting surgical repair. Surgical access to bladder tears is difficult, particularly if they are located in the caudal bladder (Ragle 2008; Higuchi *et al*. 2012), and different approaches to bladder repair have been described, including a standing laparoscopic technique (Tuohy *et al*. 2009) and repair via laparotomy under general anaesthesia (Ragle 2008; Schott and Woodie 2012). The following report documents iatrogenic bladder rupture in a gelding with urolithiasis and subsequent conservative management of the bladder rupture with catheterisation of the bladder and abdominal drainage and lavage. Successful medical management of such a large bladder tear is unusual and, to the authors' knowledge, iatrogenic rupture of the bladder during attempted cystoscopy has not been reported previously.

Case history and clinical investigations

A 14-year-old Irish Sport Horse gelding was presented to the referring veterinary surgeon with stranguria. There was no improvement following 5 days of treatment with trimethoprim sulfadiazine (30 mg/kg bwt per os b.i.d. Norodine granules)[1] and enrofloxacin (7.5 mg/kg bwt per os b.i.d. Baytril oral solution)[2], and cystoscopy was attempted. A manual pump (details unknown) was used in association with the endoscope (details unknown) to insufflate air via the biopsy channel and provide positive pressure to dilate the urethra and bladder. A spiculated urolith was visualised briefly prior to a loud noise being audible and the endoscopy image being lost. The horse dropped to the floor immediately and exhibited signs of acute abdominal pain which failed to improve following the administration of flunixin meglumine (1.1 mg/kg bwt i.v. Meflosyl)[3]. Abdominocentesis yielded a pale yellow peritoneal fluid sample which had a smell of urine. A rupture of the urinary tract was suspected, and the horse was referred for further investigation and treatment. Procaine benzylpenicillin (20 mg/kg bwt i.m. Depocillin)[4] and gentamicin (6.6 mg/kg bwt i.v. Genta equine)[5] were administered, and the horse was sedated with 5 mg detomidine (Domosedan)[6] and 5 mg butorphanol (Torbugesic)[3] for transport.

Investigation

On presentation at the referral hospital, the gelding was restless and shifted weight continuously despite being obtunded consistent with the recent administration of detomidine and butorphanol. Urine dripped from the sheath slowly but continuously. Heart and respiratory rates and rectal temperature were within normal limits. Audible borborygmi were considered normal in magnitude and frequency. On transcutaneous ultrasonographic examination of the abdominal cavity, an increased quantity of anechoic free fluid was evident (**Fig 1**). Fluid could not be visualised within the bladder. The sample obtained at abdominocentesis prior to referral had a lactate concentration of 1.4 mmol/l (reference <2 mmol/L), total protein concentration of 8 g/L (reference <20 g/L) and a total nucleated cell count of 2.1×10^9/L (reference $<5 \times 10^9$/L). Serum creatinine (533 μmol/L, reference <147 mmol/L) and potassium (7.5 mmol/L, reference 3–4.6 mmol/L) concentrations were increased consistent with the presence of uroabdomen and resorption back into the peripheral circulation.

Detomidine 4 mg i.v. (Equimidine)[3] was administered and continued to be infused diluted in saline to effect via an intravenous catheter in the jugular vein. Epidural anaesthesia was provided with a combination of 0.17 mg/kg bwt xylazine (Nerfasin)[7] and 0.1 mg/kg bwt morphine[8] prior to passage of

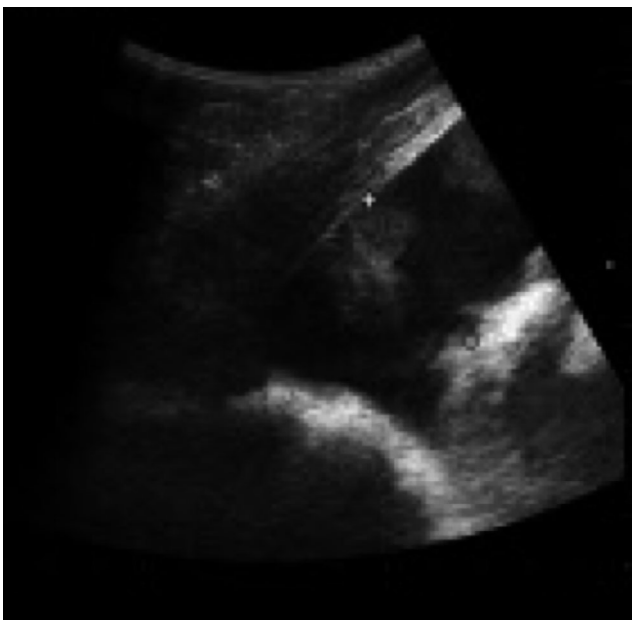

Fig 1: Ultrasonograph of the ventral abdomen of a 14-year-old Irish gelding showing an accumulation of anechoic fluid in the ventral abdomen following iatrogenic bladder rupture.

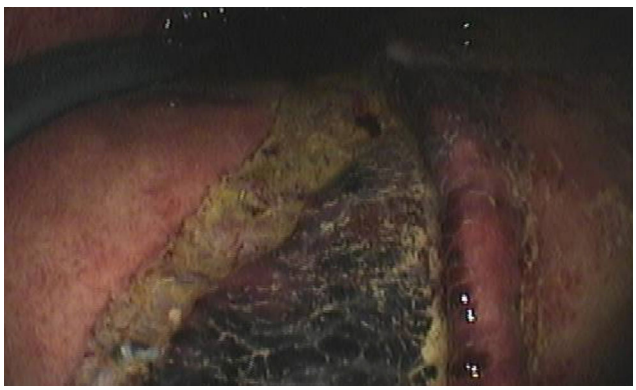

Fig 2: Cystoscopy image of the bladder of a 14-year-old Irish gelding shortly after bladder rupture is assumed to have occurred. Oriented with dorsal to the top. The ridge of tissue with petechial haemorrhages to the right is thought to represent one margin of the bladder mucosa that has retracted. The haemorrhagic area in the centre may represent deeper layers of the bladder wall possibly supported by other underlying viscera. The yellow regions are assumed to be areas with adherent sabulous material.

a 1.5 m endoscope (Olympus)[9] retrograde via the urethra. A spiculated urolith estimated to be around 10 cm diameter was visualised 55 cm from the end of the penis. A perineal urethrotomy was performed to remove the urolith as reported previously (Lillich and DeBowes 2006). A urinary catheter[10] was advanced proximally through the urethra to the level of the urolith for identification of the urethra, and an 8 cm incision was made on the perineal midline 10 cm below the anus. The subcutaneous tissues were digitally retracted to expose the urethra which was incised over the urolith to enable removal with a pair of Rochester Carmalt forceps (details unknown). With the urolith removed, the endoscope was passed via the urethrotomy into the bladder allowing visualisation of a tear in the ventral bladder to the right of the midline (**Fig 2**). The bladder was empty of urine. The margins of the tear were inflamed and irregular and petechial haemorrhages covered the surrounding mucosa. A 30 cm Foley catheter[11] was passed into the bladder via the perineal urethrotomy, inflated with 8 mL of water and sutured in situ to enable drainage of urine from the bladder. The location of the tear in the caudoventral bladder was considered difficult to access surgically so the decision was made to attempt supportive medical treatment in the hope that the tear would heal by secondary intention.

Treatment

Isotonic polyionic fluids (Aquapharm 11)[12] were administered at 4 ml/kg/h i.v. for 24 h to replace estimated fluid deficits. Gentamicin (9 mg/kg bwt s.i.d. i.v. Genta-ject)[5], flunixin meglumine (1.1 mg/kg bwt b.i.d. i.v. Flunixin)[1] and procaine benzylpenicillin (20 mg/kg bwt i.m. Depocillin)[4] were also administered. Following clipping of the abdomen, aseptic preparation and infiltration with mepivacaine hydrochloride (Intra-epicaine)[5], a full thickness stab incision was made through the skin and subcutaneous tissues using a No. 10 scalpel blade approximately 4 cm to the right of midline and approximately 20 cm cranial to the umbilicus at the most dependent point of the abdomen. A 32 French thoracic drain[13] was inserted through the stab incision into the abdomen and secured to the body wall with 0 polypropylene (Prolene)[14] suture placed in a Chinese finger trap suture pattern. A Heimlich flutter valve (details unknown) was attached to the end of the drain and secured in place.

Sixteen litres of fluid was removed from the abdomen. The drains were left in situ to allow urine to drain and to enable daily lavage of the abdomen with 10 L of sterile isotonic fluids[12]. Initially, large volumes of urine and sediment drained passively via the abdominal drain, but this gradually decreased over subsequent days as increasing volumes of urine were voided via the Foley catheter in the bladder. Serum creatinine and potassium concentrations returned to within normal ranges within 48 h.

By day 7 of treatment, no fluid was exiting via the abdominal drain, lavage of the abdomen was not associated with removal of sediment, and there was no evidence of accumulation of fluid within the abdomen on ultrasonographic examination. The abdominal drain was therefore removed. The internal appearance of the bladder was assessed with cystoscopy on days 2, 4 and 8. By day 8, urine was seen pooling ventrally and 1 L was removed via a urinary catheter to enable the torn region to be examined. The edges of the tear appeared rounded and what appeared to be a bed of granulation tissue was present between them. Administration of flunixin, gentamicin and penicillin was discontinued, and oral trimethoprim sulfadiazine (30 mg/kg bwt Norodine granules)[1], phenylbutazone (2.2 mg/kg bwt b.i.d. Equipalazone)[5] and misoprostol (5 µg/kg bwt s.i.d. Cytotec)[15] were initiated. With the findings of cystoscopy and ultrasonography indicating that urine was being held within the bladder and was not accumulating in the abdomen, the Foley catheter was removed. To prevent pressure accumulating within the bladder and to enable the

perineal urethrostomy to heal by secondary intention, a urinary catheter[10] was placed into the bladder via the penis and was sutured in situ. The urinary catheter was removed after a further 7 days of observation (14 days after admission), and the horse was discharged to continue oral trimethoprim sulfadiazine, phenylbutazone and misoprostol at home for a further 14 days. Six weeks after discharge, the horse was reported to be clinically well and urinating normally via the penis. The perineal urethrostomy had healed without complication. The horse made a complete recovery and returned to competition work.

Discussion

To the authors' knowledge, this is the first report of iatrogenic rupture of the bladder as a result of overinflation and is one of few reports of successful medical management of a bladder tear and associated uroperitoneum. Bladder tears typically occur in post-parturient mares as a direct complication of foaling (Jones *et al*. 1996; Higuchi *et al*. 2002; Stephen *et al*. 2009; Higuchi *et al*. 2012) or in foals after parturition (Kablack *et al*. 2000). Medical management of a stallion with a small (<5 cm) bladder rupture at the apex proved unsuccessful when septic peritonitis developed (Walesby *et al*. 2002). Successful medical management of uroperitoneum was reported in a colt, although cystoscopy was not performed and the size or location of the tear was not confirmed (Peitzmeier *et al*. 2016). Although surgical intervention was considered in the present case, there was a concern that the ventral location and length of the tear which extended caudally would have prevented effective closure. Rather than risk a surgical procedure that might not have been successful, the decision was made to assess the response to medical management and to attempt surgery if it proved unsuccessful. Despite the extensive nature of the tear such that no urine could be held within the bladder, the gelding responded well to medical treatment and the tear sealed within a week.

Cystoscopic examination was being attempted in order to investigate dysuria which developed as a result of urolithiasis. Urolithiasis is a relatively common condition in horses with clinical signs being reported more commonly in male horses due to the anatomy of the male urinary tract (Jaeger *et al*. 2000). Urolithiasis has been proposed as the cause of bladder rupture in a stallion (Tuohy *et al*. 2009), although the cause was not confirmed. In the case reported here, the rupture of the bladder was assumed to have been the result of excessive positive pressure caused by manual insufflation of air via the endoscope. It is likely that the urolith and the endoscope prevented or restricted the egress of air that would normally occur via the urethra as pressure within the bladder accumulates. It is unclear whether the endoscope actually entered the bladder at the initial assessment, or whether this was prevented by the presence of the urolith. Urolithiasis is associated with inflammation of the bladder and may cause the bladder walls to become more friable or even necrotic and therefore more prone to rupture (Snalune and Mair 2006). Inflammation will be more marked ventrally consistent with the site of the bladder rupture in the current report. The mucosa of the bladder and urethra is friable, particularly when it is inflamed, and it is easily damaged during endoscopy (Jaeger *et al*. 2000). Pre-existing pathology or trauma caused by the endoscope (if it entered the bladder) might also have contributed to the bladder rupturing.

Cystoscopy is an invaluable technique when investigating any case demonstrating haematuria, stranguria or dysuria. However, this case report demonstrates that it is not without risk, particularly in a patient with urethral or bladder pathology. The need for care, lubrication and asepsis has been emphasised previously (Menzies-Gow 2007). Insufflation of air is necessary to distend the urethra and bladder in order to allow them to be examined. Typically, this is performed using the positive pressure pumps which are integral to endoscopy equipment; however, in this case, an exogenous device was used, and insufflation was performed continuously as the endoscope was passed, which it is assumed resulted in over-distention of the bladder and rupture. Catheterisation is generally performed to empty the urinary bladder prior to cystoscopy and also serves to confirm the presence of a patent urethra. It is unclear whether prior catheterisation was performed in this case. This case highlights the need for judicious insufflation with air particularly if an obstruction is suspected or has been identified on catheterisation.

Uroperitoneum is an inevitable consequence of a full thickness bladder tear that communicates with the peritoneal cavity. Left untreated, uroperitoneum may lead to azotaemia, electrolyte imbalances (most notably hyperkalaemia), haemoconcentration, hypovolaemia, septic peritonitis and potentially death (Genetzky and Hagemoser 1985; Higuchi *et al*. 2002; Jenei 2012). There is therefore an understandable tendency towards closing the tear in the bladder as soon as it is identified in order to prevent further contamination of the abdomen with urine (Snalune and Mair 2006; Schott and Woodie 2012; Pye *et al*. 2018). However, surgical access to the bladder via laparotomy is limited, and hydro-distention which is reported to improve access is not possible where the bladder is torn (Russell and Pollock 2012). Surgery and general anaesthesia are also associated with risks to the patient, particularly one that is metabolically compromised (Peitzmeier *et al*. 2016).

Laparoscopic repair has been reported, both in anaesthetised horses in dorsal recumbency (Walesby *et al*. 2002; Rijkenhuizen *et al*. 2008) and in a standing horse (Tuohy *et al*. 2009) with a cranioventral tear that was considered a high risk for anaesthesia. This may offer a surgical option without the requirement for an anaesthetic; however, the tear must be in an accessible location and the procedure is not without risk. Access to the bladder via the vagina (Higuchi *et al*. 2012) or the urethra (Pye *et al*. 2018) has been reported but is only possible in mares. In this gelding in the current report, the bladder tear was located in the ventral body of the bladder, a location that is extremely difficult to access surgically (Ragle 2008; Higuchi *et al*. 2012; Pye *et al*. 2018) via any approach.

Medical management of bladder rupture and uroperitoneum is focused on correcting metabolic derangements, removing urine from the abdomen and bladder and alleviating tension in the bladder such that it is more likely to heal (Jones *et al*. 1996; Walesby *et al*. 2002). The metabolic derangement which presents the greatest risk to the patient is hyperkalaemia. Hyperkalemia can lead to bradycardia due to reduced cardiac excitability which can progress to fatal ventricular fibrillation or cardiac arrest (Reef and Marr 2010). Hyperkalemia is generally managed with intravenous fluid

therapy, utilising fluids that have a low potassium concentration, whilst the cause is removed. If bradydysrhythmia develops, then treatment with intravenous sodium bicarbonate, dextrose and insulin may also be indicated (Marr and Reef 2010). Abdominal drainage and lavage help to reduce metabolic derangements and the risk of chemical and septic peritonitis. A degree of chemical peritonitis is inevitable when urine enters the peritoneal cavity and bacteria may also enter when there is a breach in the urinary tract, particularly if there is established bacterial cystitis (Genetzky and Hagemoser 1985; Snalune and Mair 2006). Placement of a urinary catheter into the bladder not only limits the amount of urine which drains into the abdomen but also reduces tension in the bladder facilitating healing by allowing apposition of the torn mucosa (Osborne *et al*. 1996; Walesby *et al*. 2002). Supportive care in this case consisted of intravenous fluid therapy to maintain hydration status and correct electrolyte imbalances, nonsteroidal anti-inflammatory drugs, antimicrobials that are concentrated in urine and misoprostol. Misoprostol is a synthetic PGE2 analogue that may aid healing of the bladder mucosa (Gray *et al*. 1986; Kelly *et al*. 1998). A medical approach to management of bladder tears is not without risk; adhesions may develop from the bladder defect to other abdominal viscera (Peitzmeier *et al*. 2016) or the defect may fail to heal (Tuohy *et al*. 2009) protracting treatment and recovery. Adhesions may have occurred between the bladder tear and adjacent viscera in the case we report and this may have contributed to the short time it took the bladder to heal. Adhesions were not identified on examination *per rectum* but this does not eliminate the possibility that they were present.

In a previous report of the medical management of bladder tears in three mares and one colt, healing occurred in 15–21 days (Peitzmeier *et al*. 2016). In the present case, abdominal drainage of urine ceased within 7 days and urine was evident pooling in the bladder at 8 days suggesting that the bladder had healed. Delayed healing and expense associated with prolonged hospitalisation are cited as disadvantages of medical therapy (Jenei 2012; Peitzmeier *et al*. 2016; Pye *et al*. 2018); however, the present report suggests that healing may not always be protracted with medical therapy and the costs of medical management may be lower than the costs associated with surgical repair.

The present report highlights the need for care when performing cystoscopy particularly where there is established pathology and compromise of the bladder mucosa. The use of positive pressure over and above insufflation of air via an endoscope is probably inappropriate. Medical management in this case was successful despite the large size and ventral location of the tear and it may be a safer and less expensive means of managing bladder tears than suggested by the literature currently.

Authors' declaration of interests

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Authorship

All authors contributed to case management and preparation of the manuscript.

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[3]Zoetis UK Ltd, Tadworth, Surrey, UK.
[4]MSD Animal Health Ltd, Milton Keynes, Buckinghamshire, UK.
[5]Dechra Veterinary Products, Shrewsbury, Shropshire, UK.
[6]Vetoquinol UK Ltd, Great Slade, Buckingham, UK.
[7]Le Vet B.V., Oudewater, Netherlands.
[8]Martindale Pharmaceuticals, Romford, Essex, UK.
[9]Olympus, Southend-on-Sea, Essex, UK.
[10]KRUUSE UK Ltd, Sherburn in Elmet, North Yorkshire, UK.
[11]Smiths Medical ASD Inc, St Paul, Minnesota, USA.
[12]Animalcare Ltd, York, UK.
[13]Deknatel Inc, Fall River, Massachusetts, USA.
[14]Ethicon Inc, Johnson & Johnson Medical Limited, Edinburgh, UK.
[15]Pfizer UK, Walton Oaks, Surrey, UK.

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