Traumatic rupture of the urinary bladder in a horse

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An 18-months-old Thoroughbred filly weighing 400 kg presented at the Rural Veterinary Centre 12 h after running through a fence. Two hours before presentation the filly showed signs of depression and reluctance to move. Physical examination revealed an increased heart rate (80 beats/min) and respiratory rate (40 breaths/min) and reduced gastrointestinal motility on auscultation of the abdomen. Moderate soft tissue swelling of the musculature of the left flank was associated with superficial skin abrasions. The ascending colon was moderately distended on transrectal palpation and there was 5 L of spontaneous reflux after passage of a nasogastric tube.

Apart from an increased haematocrit (0.52 L/L), other haematological values were within normal limits. Serum biochemical analysis indicated a hyperkalaemia (6.81 mmol/L) and increased creatinine (188 μmol/L) and blood urea nitrogen (10.35 mmol/L) concentrations and creatinine kinase activity (1216 U/L). Abdominocentesis yielded a large volume of slightly turbid, yellow fluid with increased creatinine concentration (1134 μmol/L). The protein concentration (1.0 g/L) and total nucleated cell count (420 cells/μL) were within normal limits. A transabdominal ultrasound examination confirmed the presence of a large volume of fluid in the abdominal cavity.

On the basis of the clinical findings, uroperitoneum secondary to rupture of the urinary tract was suspected. The filly was sedated and the vulva and the caudal vaginal vault aseptically prepared. A 1 m videoendoscope that had been chemically sterilised with dialdehyde* and rinsed with sterile sodium chloride was passed through the external urethral orifice into the bladder. A full thickness tear approximately 3 cm long could be seen at the apex of the bladder. The endoscope was passed through the tear into the abdominal cavity and the parietal peritoneum underlying the skin abrasions on the left flank was inspected. There was a laceration in the parietal peritoneum with separation from the deep abdominal musculature at the ventral aspect of this laceration.

Surgical repair of the bladder defect through a ventral midline coeliotomy was recommended. Because of financial constraints the owner declined surgery in preference to medical management. In fact, internal intravenous fluid therapy consisted of 50 L of 0.45% sodium chloride and 5% dextrose administered over the first 24 h of hospitalisation. The ventral midline of the anterior abdomen was aseptically prepared and a sump drain placed transabdominally under local anaesthesia and sutured in place. Approximately 10 L of urine was drained from the abdominal cavity. A 14 gauge Foley balloon tipped catheter with a one-way valve was passed through the urethra and secured in the bladder by inflation of the balloon. Antimicrobial therapy was commenced with procaine penicillin (15 000 IU/kg, intramuscularly, every 12 h), and gentamicin sulphate (3.3 mg/kg, intravenously, every 12 h). Additionally the filly was given flunixin meglumine (0.25 mg/kg, intravenously every 8 h) as prophylaxis for endotoxaemia. These agents were administered throughout hospitalisation.

Serial blood samples collected during the following 24 h indicated that the serum potassium, creatinine and blood urea nitrogen concentrations and the abdominal fluid creatinine concentration (174 mmol/L) had returned to within normal limits. There was an increase in the protein concentration (24 g/L) and total nucleated cell count (8449 cells/μL) in a sample of the abdominal fluid collected from the drain 24 h after admission. There were no bacteria seen on a Gram-stained preparation of the abdominal fluid. The intravenous fluids were changed to Ringer’s lactate and the serum electrolyte concentrations were monitored and remained within normal limits. The rate of administration was reduced to 30 L over the following 24 h. Thirty-six hours after placement of the abdominal sump drain the volume of fluid draining from the abdominal cavity had reduced and there was no evidence of abdominal fluid on transabdominal ultrasound examination, so the drain was removed. Urine continued to flow from the urethral catheter.

The filly continued to demonstrate signs of depression with an increased heart rate. However, no signs of abdominal pain were evident. Although intestinal motility was reduced, faecal output was considered normal. Forty-eight hours after presentation the filly demonstrated signs of violent abdominal pain that proved unresponsive to flunixin meglumine (1.1 mg/kg intravenously) followed by xylazine (0.5 mg/kg intravenously). There was an increase in the heart rate (120 beats/min) and haematocrit (0.61 L/L) and a decrease in the plasma protein concentration (40 g/L). In view of the severe abdominal pain, and because surgical intervention was not an option, the filly was euthanased.

Necropsy revealed diffuse, acute, severe peritonitis associated with ingesta leaking from a 3 cm full-thickness defect in the centre of a circular area of infarction in the right ventral colon. There were two other 10 to 20 cm areas of infarction of the right ventral colon. There was a 2 cm tear in the apex of an empty urinary bladder and the edges of the tear were necrotic, with no evidence of new granulation tissue. There was no significant volume of fluid in the abdominal cavity. A post-mortem diagnosis of rupture of the bladder and focal colonic infarction with subsequent mural perforation, both secondary to severe external abdominal trauma, was made.

Rupture of the urinary bladder has been reported in most domestic species and humans (Tulleners et al 1980; Behr et al 1981; Bertone and Smith 1984; Hackett 1984; Nyrop et al 1984; Roussel and Ward 1985; Lavoie and Harnagel 1988; Martinez and Schulman 1988; Renvall et al 1989; Sweeney et al 1991; Gibson et al 1992; Vacek et al 1992; Carr et al 1993; Andrews et al 1995). Obstructive urolithiasis is the most common cause of rupture of the urinary tract in domestic ruminants (Soklett and Knight 1984). Rupture of the urinary bladder in foals is thought to occur commonly secondary to vesicular compression during parturition, but it may also occur secondary to congenital defects in the bladder or urachal infections (Hackett 1984). Adams and Koterba (1988) have also suggested that spontaneous rupture may occur subsequent to septicaemia. Rupture of the urinary bladder in adult horses is less common and has been associated with obstructive urolithiasis (Gibson et al 1992; Vacek et al 1992), parturient trauma (Nyrop et al 1984) and neoplasia (Firth 1976; Sweeney et al 1991). It has also been reported to have occurred spontaneously in an adult horse that underwent surgery to correct a ruptured bladder as a foal (Pankowski and Fubini 1987). In the case reported here rupture of the urinary bladder presumably occurred at the time of trauma to the left flank.

The presence of hyperkalaemia, combined with an elevated ratio of peritoneal fluid creatinine to serum creatinine (6:1), was suggestive of uroperitonitis. Peritoneal fluid to serum creatinine ratios greater than 2:1 are considered strongly suggestive of this condition (Behr et al 1981; Richardson and Kohn 1983; Hackett 1984; Genetzky and Hagemoser 1985; Adams and Koterba 1988; Slone 1990; DeBowes 1992). Other serum biochemical abnormalities commonly associated with uroperitonitis, including hypernatraemia, hyperchloraeemia, hyperkalaemia and azotaemia do not always occur (Behr et al 1981; Richardson and Kohn 1983; Hackett 1984; Genetzky and Hagemoser 1985). While this filly had hyperkalaemia and mild

* Cidex®, Johnson and Johnson, North Ryde, NSW
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azotaemia, serum sodium and chloride concentrations were within normal limits.

Cystoscopy of the bladder provided a definitive diagnosis of rupture of the urinary bladder and is considered to be a safe and useful diagnostic aid in cases where patient size and demeanor permit urethral passage of an endoscope and cystoscopic examination (Nyrop et al 1984; Sullins and Traub-Dargatz 1984; DeBowes 1992; Gibson et al 1992). Passing of the endoscope through the tear in the bladder permitted limited exploration of the abdominal cavity and observation of the damaged parietal peritoneum. Unfortunately the infarcted areas of the right ventral colon were not identified. The normal laparoscopic anatomy of the abdominal cavity in the horse has been described (Galluppo et al 1995) and laparoscopic diagnosis of a uterine tear in a post-partum mare has been reported (Hassel and Ragle 1994). In this case, laparoscopic examination of the abdominal cavity would have been unlikely to have been useful in identifying the infarcted areas of the colon.

The preferred method of treatment for tears in the bladder is primary surgical closure through a ventral midline coeliotomy (Slone 1990; DeBowes 1992). The successful medical management of uroperitoneum, secondary to a devitalised bladder without a discrete tear in a gelding (Gibson 1992) and a foal with rupture of the urinary bladder (Lavoie and Harnagel 1988) have been described. Surgical correction in this case was not an option for financial reasons and given these previous reports conservative management was considered as a therapeutic option. The owner consented to conservative treatment despite the uncertainty of the prognosis.

Continuous drainage of urine from the peritoneal cavity, with concurrent replacement of pre-existing fluid and electrolyte losses, and the maintenance of ongoing fluid losses, will usually correct many of the metabolic derangements occurring in a horse (Slone 1990; DeBowes 1992). Intravenous solution containing 5% dextrose is reported to be beneficial in the treatment of hyperkalaemia associated with uroperitoneum (Hodgson 1987). In the case reported here, when serum concentrations of sodium and chloride were within normal limits dextrose was added to a commercial preparation of 0.45% sodium chloride. When the serum potassium was within normal limits a balanced isotonic maintenance fluid was administered and the serum electrolyte concentrations monitored.

Indwelling urethral catheters are an important component of conservative therapy for rupture of the urinary bladder in people. Urethral catheters prevent the pooling of urine in the bladder and aid healing of the defect by second intention within 2 to 3 weeks (Mulkey and Witherington 1974). In ruminants bladder defects can be sealed by omentum and fibrin deposition in 3 to 5 days without the need for a urethral catheter (Walker and Hull 1984; Powe 1986). The efficacy of indwelling urethral catheters in horses remains to be determined. An indwelling catheter attached to continuous suction drainage led to the sealing of a bladder defect in 5 days in a foal (Lavoie and Harnagel 1988). Indwelling urethral catheters in horses remain to be determined. An indwelling catheter attached to continuous suction drainage led to the sealing of a bladder defect in 5 days in a foal (Lavoie and Harnagel 1988). Indwelling urethral catheters were not used in this filly because, in the authors experience, passive drains with a one-way valve have previously been found to provide adequate drainage of the bladder. Despite urine draining from the catheter and the absence of urine in the abdomen post mortem, gross examination of the tear revealed no evidence of healing.

Although septic peritonitis is not a common complication of rupture of the urinary bladder in otherwise healthy foals, it may be more common in adult horses (Firth 1976; Trotter et al 1981). Gibson et al (1992) propose that most cases of septic peritonitis in adult horses, following bladder rupture, are related to the presence of a bacterial cystitis associated with urinary calculi. However, urine causes a chemical peritonitis, which may predispose the abdominal cavity to secondary bacterial infection (Sackett and Knight 1984; Powe 1986). There was no evidence of a septic or chemical peritonitis in the first sample of peritoneal fluid collected from this filly. There was a modest elevation of the total protein concentration and total nucleated cell count in the peritoneal fluid sample collected 24 h after admission, but no bacteria were seen. These changes may have been associated with a developing chemical peritonitis, the presence of the drain, or the infarcted areas in the wall of the right ventral colon. Based on these previous reports (Firth 1976; Trotter et al 1981; Gibson et al 1992) and current recommendations (Slone 1990; DeBowes 1992) the horse in this report received broad spectrum antimicrobial agents.

Infarction of discrete areas of the large colon or rupture of the bladder secondary to external trauma has not been described in the horse. Rupture of the bladder, caecal rupture, and discrete mesenteric tears of the small colon and jejunum have been described as sequelae to internal trauma associated with parturition (Nyrop et al 1984; Dart and Pascoe 1994). In the filly described here it is proposed that the external abdominal trauma lead to rupture of the urinary bladder and infarction of areas of the large colon with subsequent necrosis and perforation.

References


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