A clinical case: bladder rupture in newborn foals

Um caso clínico: rotura da bexiga em poltros

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Summary: A clinical case with a 5 days old, deceased, stallion is discussed. In 0.2 – 1% of the newborn foals a rupture of the urine bladder occurs. This pathological situation leads to the accumulation of urine in the abdominal cavity. This is the start of a cascade of pathological alterations and clinical signs. The differential diagnoses are discussed. If a practitioner is confronted with a patient suffering this discomfort, the first thing to be done is to stabilise the electrolyte imbalances, in particular the hyperkalemia because it can lead to life-threatening arrhythmias. In fact, some foals die before surgery, probably from cardiac arrest partially due to elevated potassium levels. Surgery consists in the closure of the urine bladder and the washing of the abdominal cavity. When diagnosed quick, in general, the prognosis is favourable: about 80-90% patients survive.

Resumo: Descreve-se um caso clínico referente a um poldro de 5 dias de idade que morreu por rotura da bexiga. Isto ocorreu em 0,2 a 1% dos poltros nascidos. Esta situação leva à acumulação de urina na cavidade abdominal responsável por várias alterações patológicas e sinais clínicos. Os diagnósticos diferenciais são referidos. No caso de um clínico deparar com um paciente com este problema deve em primeiro lugar corrigir os desequilíbrios electrolíticos, em especial a hipercalemia visto poder originar arritmias. De facto, alguns poltros morrem antes da cirurgia por paragem cardíaca devido aos elevados níveis sanguíneos de potássio. A cirurgia consiste na sutura da bexiga e na lavagem da cavidade abdominal. Quando esta situação clínica é diagnosticada precocemente, o prognóstico é bastante favorável: cerca de 80 a 90% dos pacientes sobrevivem.

Case description

In April 2001, our clinic received a request to do a post-mortem examination on a 5 days old foal that was found dead in the pasture. Because this was the second dead foal within 1 week, the veterinarian at the farm was worried about possible infectious diseases among the herd. The foal was born normally and didn’t look ill during the first days of life. The only abnormal finding observed was that it looked as if he was urinating frequently without producing urine. After day 3 the foal was drinking less frequently, recognisable by the udder of the mare leaking milk. Very mild colic signs were seen in the evening of day 4.

The necropsy revealed a foal with a normal build. The only remarkable finding was a dilated abdomen. After opening the abdominal cavity, we first had to remove 10 litres of a yellowish, bad smelling fluid, obviously urine, before we could diagnose the course of the death of this foal: the urine bladder ruptured. Is this something that could be prevented? How do you recognise it?

Incidence and aetiology

Ruptured urinary bladder is the most common bladder disorder in neonatal foals. The incidence is 0.2 to 1% (Baerveldt and Klein, 1991). It occurs most often in colts less than 1 week of age, but can also occur in fillies and older foals. There is no known breed predilection.

Bladder rupture is a tear or leak in the urinary bladder that results in uroperitoneum. Uroperitoneum, the accumulation of urine in the peritoneal (abdominal) cavity, can also result from disruption of other parts of the urinary tract. It is difficult to predict when bladder ruptures might occur; thus, they are difficult to prevent. Although they can occur in adult horses, usually from urinary tract obstruction, the vast majority of bladder ruptures occurs in foals. Here, the rupture usually occurs in the bladder itself, but can also occur in the urachus. The urachus, a structure in the foetus, which lies between the tip of the bladder and the umbilical cord, allows the excretion of urine. This structure is normally closed at birth, and over several weeks contracts to a thick band of tissue.

Most bladder ruptures in foals occur during parturition (Baerveldt and Klein, 1991; Hardy, 1998). It is thought that the increased abdominal pressure the foal experiences during birth can lead to rupture of a full bladder if urine cannot be easily voided. The anatomy of the male pelvis and the longer length of the male urethra tend to predispose the male foal to this problem more often than the female. However, the condition can also be caused by any type of trauma such as kicks or rough handling. Congenital defects of the bladder have also been reported (Richardson and Kohn, 1983). Finally, bladder rupture can occur secondary to septic omphalitis. The infection may extend to the bladder resulting in necrosis and weakening of the wall, predisposing it to rupture.
Physical examination and diagnosis

Early clinical signs are subtle and the foal may appear normal at birth. Typical early signs are non-specific and include depression, inappetence, tachycardia, and tachypnea. The foal’s appetite is most easily determined by observation of how full the mare’s udder is. Most male foals urinate within 6 hours of birth, and if urination has not been observed and the above non-specific signs are evident, ruptured bladder should be considered. Clinical signs progress to mild abdominal pain, increased abdominal distension, scrotal accumulation of urine, and respiratory distress due to abdominal pressure. Most foals with a ruptured bladder show frequent straining to urinate, although some appear to urinate normally. It is important to differentiate straining to urinate from straining to defecate due to a meconium impaction. Severe electrolyte imbalances due to bladder rupture may result in neurological signs, severe arrhythmias, and death (Baerveldt and Klein, 1991).

Uroperitoneum can also develop in neonates with debilitating problems or systemic infection. Leaking of urine into the abdomen might occur through tears in the bladder or the urachus. These foals do not rupture the bladder at birth. Infection might not primarily involve the urachal area, but can adversely affect it. Consequently, the tissue in the area becomes swollen and inflamed, loses integrity, then starts separating and leaking. This probably accounts for about 50% to 40% (Hardy, 1998; Richardson and Kohn, 1983) of registered uroperitoneum cases. The clinical signs seen in these foals are similar to those mentioned earlier, except these foals have some other disease process present. Most of these clinical signs will occur somewhere between four and 12 days of age.

Confirmation of a suspected case requires laboratory data, diagnostic imaging techniques, and ancillary tests.

The results of the laboratory data, in special the clinical pathology, will show a patient with an azotemia (elevated blood urea nitrogen and creatinine). There might be a hyponatremia, a hypochloremia and a hyperkalemia. Most affected foals have a metabolic acidosis (Baerveldt and Klein, 1991).

Urine is high in potassium and low in sodium and chloride. As it passes into the abdomen it equilibrates with the plasma, causing the above metabolic disturbances.

Analyses of the abdominocentesis will show an abdominal /serum creatinine > 2:1. Calcium carbonate crystals are sometimes observed. Secondary to a septic omphalitis there might be a peritonitis diagnosed, but urabdomen alone does not normally cause peritonitis. An elevated urea nitrogen level may also be observed, but this equilibrates quickly with the plasma and is therefore not a reliable test. Creatinine is a larger molecule and equilibrates more slowly, making it a more reliable parameter.

Using diagnostic imaging, there is the choose between plain abdominal radiograph, contrast radiography and ultrasonography. A flaccid, folded bladder is observed in radiographs, and the defect may be visible with contrast imaging. Ultrasonography is rapid, non-invasive, and allows defect location. A large amount of abdominal fluid may be observed by ultrasound, and the collapsed, flaccid bladder is visible.

Ancillary tests that can be performed are: the methylene blue test (inject sterile dye into urinary catheter and observe its flow into the abdomen) or heat the abdominal fluid to detect ammonia.

The list with differential diagnosis contains: colic (especially meconium impaction), ureteral defects, urachal rupture and intestinal atresia.

Ureteral defects and urachal rupture can cause uroperitoneum with similar signs to bladder rupture.

Emergency medical therapy

The immediate concern is to correct the electrolyte imbalances, particularly the hyperkalemia, which can lead to life-threatening arrhythmias. In fact, some foals die before surgery can be performed, probably from cardiac arrest partially due to elevated potassium levels. To alleviate that risk, the abdomen should be drained (at least partially) and intravenous fluids (without potassium) should also be administered. Intravenous NaCl is often enough to correct imbalances, with or without dextrose. Sodium bicarbonate can be administered in order to correct acidosis. Insulin (and glucose) is also helpful in resolving severe hyperkalemia by driving K+ into cells (Baerveldt and Klein, 1991; Hardy, 1998; Richardson and Kohn, 1983).

Elective surgical treatment

The foal must be metabolically stable before surgery to avoid anaesthetic complications. The placement of a urinary catheter is controversial among practitioners and may or may not be performed.

The surgical steps are the following: after a caudal midline incision around umbilicus the bladder is exteriorised. The surface of the rupture location is debrided. The abdominal cavity is emptied by suction and after that cleaned by lavage with huge amounts of fluid. The bladder is closed with a double inverting suture. At the end the urachus is resected and the incision is closed.

Laparoscopic surgery is an alternative to conventional surgical treatment. It is less invasive and allows better visualisation, but also requires specialised equipment and expertise (Baerveldt and Klein, 1991; Hardy, 1998; Johnson, 1970; Kablack et al., 2000; Richardson and Kohn, 1983).

Complications and prognosis

During the pre-operative period there is a chance of urachal trauma with haemorrhage of umbilical artery. In worse cases, the foal may die due to severe metabolic disturbances.
After surgery some problems can occur: recurrence of the uroabdomen due to leakage from the suture line in the bladder; a failure to close all of defect; complications from the incision or adhesions.

Post-operative treatment consists of antibiotics and anti-inflammatory for at least a few days. The administration of other compounds depends on the condition of the foal.

In general, the prognosis is favourable; about 80-90% of primary bladder rupture patients survive. Foals that are sick for other reasons and develop uroperitoneum have a poor prognosis, i.e., about 50% survive. Some of those foals are in marginal condition, and they will not survive (Baerveldt and Klein, 1991; Hardy, 1998; Johnson, 1970).

Bibliography


