Uroperitoneum secondary to rupture of the urachus associated with *Clostridium* spp. infection in a foal: a case report

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ABSTRACT: An 8-hour-old Andalusian colt was referred to the Veterinary Teaching Hospital of the University of Cordoba due to weakness, lateral recumbence, diarrhoea and absent sucking reflex. At admission the foal was obnubilated, with cold limbs, pale mucous membranes, tachycardia and diarrhoea. Laboratory results revealed increased hematocrit and total protein concentration, hyperfibrinogenemia, leukopenia, azoetemia, hypertryglyceridemia and a decreased IgG level. A diagnose of failure of passive transfer of immunoglobulins and hyperlipaemia was made. On the third day of hospitalization the foal presented painful and swollen joints, abdominal distension and fever. The ultrasonographic study at this point showed abundant hypoechogenic free fluid content in the peritoneal cavity. Abdominocentesis demonstrated a high peritoneal creatinine:serum creatinine ratio. Culture of the peritoneal fluid was *Clostridium* spp. positive. Necropsy revealed a tear in the internal umbilical remnant. The final diagnosis was uroperitoneum secondary to rupture of the urachus associated with a *Clostridium* spp. infection. New emerging aetiologies responsible for uroperitoneum, either by septic urachitis or omphalophlebitis, are emerging. Nonetheless, cases of uroperitoneum induced by *Clostridium* spp. infection are rare and constitute a new and important finding for equine neonatal medicine. In addition, the hyperlipaemic status in this animal could be a plausible cause leading to septicemia and subsequent uroperitoneum.

Keywords: internal umbilical remnant; peritonitis; uroabdomen; clostridium; colt

Uroperitoneum in foals has been widely described over the last 50 years. Most published reports regarding uroabdomen describe the bladder defect as the main underlying cause (Richardson and Kohn, 1983; Butters, 2008), and only a few reports describe a primary defect in the urachus as the cause of this disease, both in horses (Kablack et al., 2000) and other animals (Muller and Gregory, 1978; Baxter et al., 1992; Braun et al., 2009).

The diagnosis is based on history and signalment, clinical presentation and pathological results. The main clinical findings include abdominal distension, anorexia, depression, tachycardia, tachypnea, colic signs and urination alterations, and the most important profile work findings are hyponatremia, hypochloremia and hyperkalemia (Behr et al., 1981; Baerveldt and Klein, 1991). Nonetheless, sometimes, due to the short duration of the uroperitoneum or previous fluid therapy treatment given by the referring veterinarian, these findings may go unnoticed. Other clinical symptoms compatible with uroperitoneum include light to moderate metabolic acidosis, respiratory acidosis due to abdominal distension, enhanced serum creatinine and ureic nitrogen values, a huge level of abdominal free fluid content with a low cellularity and density, and a ratio of peritoneal creatinine : serum creatinine concentration higher than 2 : 1 (Hardy, 1998). Usually, the transabdominal sonogram shows relatively anechogenic free fluid and sometimes it is possible to visualize the defect in the urinary tract (Kablack et al., 2000; Pokar, 2004).

The majority of equine neonates with uroabdomen require surgical reparation of the tear (Edwards

et al., 1995; Butters, 2008) but a previous preoperative medical stabilization is necessary in order to avoid the numerous complications described during the anaesthesia (i.e., those related to acid-base and electrolytes disturbances, mainly due to hyperkalemia) (Kritchevsky et al., 1984). Hyperkalemia is corrected through the use of free potassium fluids, bicarbonate, dextrose, calcium gluconate, insulin or by peritoneal dialysis. Currently, with the development of safer anesthetic drugs, such as isofluorane, it is possible to minimize the complications during surgery. In some cases the tear may be repaired by catheterizing the defect of the urinary tract with a Foley probe or something similar (Lavoie and Harnagel, 1988).

The objective of this work was to describe a clinical case of a foal with a failure of passive transfer of immunoglobulins and hyperlipaemia associated with an atypical presentation of uroperitoneum. The uroabdomen was secondary to the rupture of urachus associated with an infection of the internal umbilical remnant by *Clostridium* spp.

Case description

History and clinical findings. An 8-hour-old Andalusian colt, weighing 42 kg, was referred to the Veterinary Teaching Hospital of the University of Cordoba (Spain) due to progressive depression, anorexia, weakness and recumbence. The mare, a four year old, was at that time on a regimen of vaccinations (tetanus, rhinoneumonitis and influenza) and deworming. She was fed oats (2 kg twice a day), alfalfa hay *ad libitum*, and had free access to water. The pregnancy time was 328 days, while foaling was normal, and shorter than 15 min in duration. Placental separation occurred in the following hour after foaling. Colostrum loss was not apparent previously to the day of labour. After delivery, the colt rapidly stood up, sucked, urinated and eliminated the meconium normally. After 5 h of life, the owner decided to refer the foal to the hospital due to prolonged recumbency and the absence of sucking. At admission to the hospital physical exploration of the foal revealed obnubilation, lateral recumbency, tachycardia (132 beats/min), regularly regular cardiac rhythm and soft pulse, normal temperature and normal breathing frequency (40 breaths/min). The mucous membranes were slightly pale, with decreased capillary refill time and elasticity of the skin. The animal also presented a doughy brown colour diarrhoea, diminished sucking reflex, cool limbs and entropion in both eyes. Neither joint swelling nor abdominal distension were observed.

Laboratory findings and ancillary tests. Analysis of the blood yielded the following results: increased packed cell volume (43%), normal total protein concentration (6.5 g/dl) and leukopenia (4.9×10^3/µl) with left shift (neutrophils: 3.6×10^3/µl). The biochemistry profile showed elevated creatinine (2.5 mg/dl), lactate (8.1 mmol/l), fibrinogen (400 mg/dl) and triglyceride (1080 mg/dl) values. The IgG concentration (< 400 mg/dl) was under the normal level while glucose was within the normal range (98 mg/dl). The sepsis score was estimated to be less than 11 points. The ionogram showed a lightly ionized hypocalcemia (1.30 mmol/l) and hyponatremia (128.1 mmol/l); the remaining ions were within the normal reference range (K^+ 4.7 mmol/l and Cl^- 96 mmol/l). The main abnormalities found in the gas study were acidemia (pH 7.30) and hypercapnia (pCO_2 49.4 mm Hg). Pulmonary, transabdominal and umbilical ultrasound analyses were normal. Additionally, the size of both the internal umbilical remnant (< 2.5 cm) and the umbilical arteries and vein were normal (< 1 cm).

Taken together, the results of the physical examination, laboratory analyses and ultrasonographic findings led us to make a diagnosis of failure in passive transfer of immunoglobulins and hyperlipaemia.

Treatment. The established treatment consisted of: (a) plasma transfusion (5 ml/kg) obtained from an adult gelding; (b) fluid therapy with polyionic crystalloid solution (Ringer Lactate, 210 ml/kg, *i.v.*, q 1 h); (c) antimicrobials: amykacin (25 mg/kg, *i.v.*, q 24 h) and ceftiofur (10 mg/kg, *i.v.*, q 12 h); (d) nonsteroid anti-inflammatory drug, flunixin meglumin (0.25 mg/kg, *i.v.*, q 12 h); (e) omeprazol (4 mg/kg, *p.o.*, q 24 h); (f) maternal milk as enteral nutrition, 250 ml every hour through a nasogastric tube; (g) unilateral nasal oxygenotherapy (5 l/min), and (h) routine care and handling of hospitalized foals, i.e., cleaning of the navel with chlorhexidine 1% q 4 h, change of lateral recumbence, etc.

Outcome. After 24 h of hospitalization, the foal's health status improved considerably, swollen joints were not observed, fever had ceased, diarrhoea frequency was decreasing and the colt was able to stand up and feed by himself. Moreover, an excellent recovery of packed cell volume, total protein, plasmatic creatinine and lactate (35 %, 4.9 g/dl,

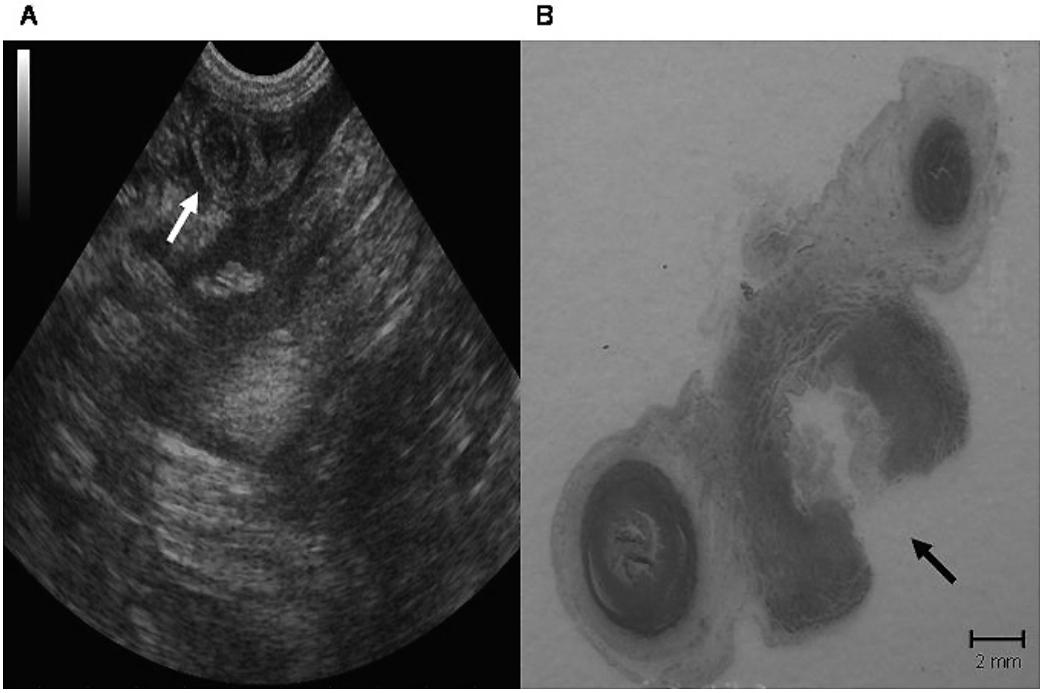

Figure 1. Sonogram of the ventral midline of the abdominal cavity (A). This sonogram was obtained 80 h post admission with a 6.6 MHz curved (convex) linear transducer operating at a displayed depth of 6 cm. Note the enhanced size of the left umbilical artery (> 1.5 cm; arrow) and large hypoechogenic free fluid content in the peritoneal cavity. Hematoxilin-eosin stain on internal umbilical remnant embedded in paraffin (B). The left umbilical artery is increased in size compared to the contralateral one. Notice the rupture of the urachus in the dorsal aspect (black arrow). An important decrease in the arterial lumen can be also observed; submacro image

1.5 mg/dl and 1.6 mmol/l, respectively) was detected. However, a second plasma transfusion was performed because IgG values remained low after the first transfusion (< 400 mg/dl). The rest of the treatments were also continued. At 80 h old (roughly three days of age) several swollen joints (both carpi and hocks), abdominal distension, tachypnea (80 breaths/min), tachycardia (140 beats/min), fever (102.5°F) and oedema in the escrotum and umbilicus were noted.

At this point, the abdominal ultrasonography was repeated and showed abundant hypoechogenic free fluid content and an enlargement of the left umbilical artery (> 1.5 cm) suggestive of arteritis (Figure 1A). Abdominal paracentesis was performed revealing a yellow and cloudy fluid, with a total protein concentration of 0.8 g/dl and elevated leukocytes and creatinine content (18.2 × 10^3/µl and 3.9 mg/dl, respectively). Urinary catheterization was carried out to confirm the diagnosis of uroabdomen. For that purpose, a sterile fluorescein solution was infused through the urinary probe and a second abdominocentesis was performed. Free fluorescein was detected in the peritoneal fluid by cobalt blue light, confirming a final diagnosis of uroabdomen. Clinical signs, the abdominal ultrasonography, the peritoneal fluid characteristics and the peritoneal creatinine:serum creatinine ratio (3.9 : 1.2 mg/dl) were all indicative of uroperitoneum and peritonitis. Cytology on the peritoneal fluid revealed numerous Gram positive rods and a sample was sent for microbial culture. In view of this finding, antimicrobial therapy was changed to amykacin (25 mg/kg, *i.v.*, q 24 h), potassium penicillin (22 000 UI/kg, *i.v.*, q 6 h) and metronidazol (15 mg/kg, *p.o.*, q 8 h).

As a consequence of the bad prognosis and the economic costs associated with the continued use of joint lavages and the reparative surgery of the defect in the urinary tract, the owner decided to sacrifice the animal.

Necropsy and bacteriological findings. The main findings in the post-mortem study were limited to the urinary tract. Gross evaluation showed a tear in the dorsal aspect of the internal umbilical remnant with a diameter of 1 × 1.5 inches approximately, lined by hyperaemic tissue with numerous haemorrhages, discarding a post-mortem artefact (Figure 2). In the internal umbilical remnant, only

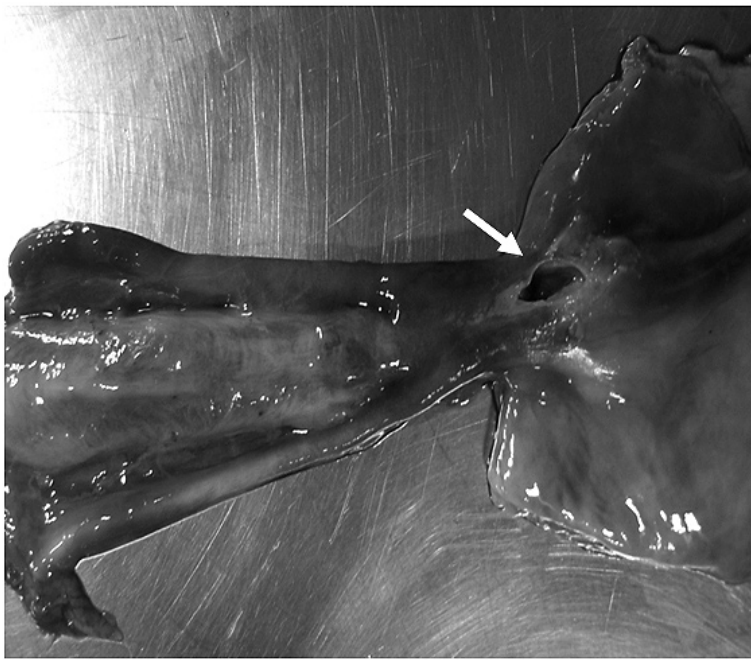

Figure 2. Gross aspect of the internal umbilical remnant. In this picture the bladder, both umbilical arteries, the urachus and the umbilical vein may be seen. Note the tear in the dorsal aspect of the urachus (arrow), the increased size of the left umbilical artery compared to the contralateral one and the intense congestion around the defect

the left umbilical artery was altered, with an increased size and lack of elasticity (Figure 2). The urinary bladder and the rest of urinary structures showed no remarkable defects. Microscopic analysis revealed severe thromboarteritis of the left umbilical artery, with a large intraluminal clot occupying ca. 90% of the arterial lumen (Figure 1B). A severe perivascular lymphoplasmocytic infiltrate was found and abundant degenerative neutrophils were seen in the serosa. The defect in the urachus resulted in important tissue remodelling with a large amount of activated fibroblasts and unorganized collagen fibres, suggesting an active scaring process, in addition to the inflammatory cellular infiltrate. Gram staining revealed Gram positive rods, some of them showing terminal spores, in the ruptured umbilical area and multifocally in the clotted umbilical artery (Figure 3). Microbiological culture of the peritoneal fluid identified these bacteria as *Clostridium* spp.

DISCUSSION AND CONCLUSIONS

An 8-hour-old Andalusian colt was referred to the Veterinary Teaching Hospital of the University of Cordoba due to obnubilation and absence of the sucking reflex. A final diagnosis of uroperitoneum

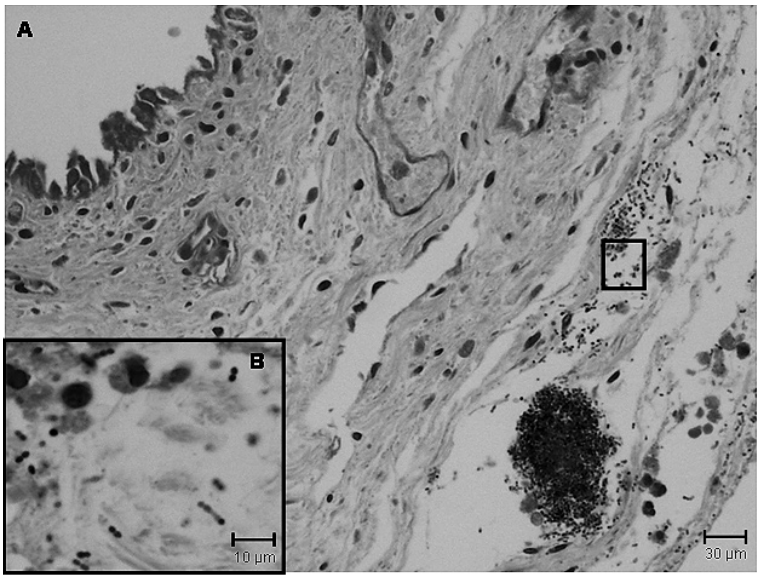

Figure 3. Gram stain on a paraffin-embedded urachus sample (A). Note the disruption between the urachus layers, and a large septic clot; 20×. Same stain as in A image with 40× augmentation (B). Notice the rod-shape bacteria with spore-shaped morphology lining the urachus defect

secondary to rupture of the urachus associated with a *Clostridium* spp. infection was made.

Concomitant uroperitoneum and septicemia is a well described condition, but few investigations have shown that septicemia can be the direct causative effect of uroperitoneum in equine neonates, although it appears to be an important predisposing factor for the development of uroabdomen (Dunkel et al., 2005). In septic foals, *E. coli* and *Actinobacillus equuli* are the most commons organisms isolated from culture of the blood; while *E. coli* and *Enterococcus* spp. are the most frequently bacteria found in the peritoneal fluid of foals diagnosed with uroperitoneum (Marsh and Palmer, 2001). In this case, the microbiological isolation of *Clostridium* spp. in the peritoneal fluid and in the tissue samples taken from the internal umbilical remnant together with the histological localization of Gram positive spored bacilli in the umbilical tear support the hypothesis that the underlying cause of urachitis and uroperitoneum in this foal was a clostridial infection. Although blood culture was recommended prior to antibiotic therapy, it was not performed due to financial constraints. Moreover, in a previous report, only one out of 38 foals with confirmed clostridial enterocolitis were positive for *Clostridium* spp. from blood culture (East et al., 1998). Something to be considered in the event positive culture results is possible post-mortem contamination; however, in our case the bacteria were identified inside the tissue and not around it, ruling out that possibility. To the knowledge of the authors, only two reports have described a rupture of the urachus by *Clostridium* spp. infection, one of them induced by *Clostridium perfringens* (Hyman et al., 2002) and the other one by *Clostridium sordellii* (Ortega et al., 2007).

C. sordellii is a soil organism and is able to penetrate the wounds and mucous membranes. It has been demonstrated that *C. sordellii* induces haemorrhagic enteritis in equine neonates and oedema, infection of wounds and hyperammonemia in adult horses (Desrochers et al., 2003; Ortega et al., 2007). *C. perfringens* is a life-threatening bacterium in 1–10 day old foals, inducing necrotizing enterocolitis (East et al., 1998). It is normal resident in the healthy horse intestine, spread by the faeces until two months of age. In the case reported here, ancillary studies supported the causative role of *Clostridium* spp. We hypothesised that these bacteria could have induced the rupture of the internal umbilical remnant by contamination through either direct dissemination from the intestine and subsequent bacteraemia, or by direct contamination of the external umbilical remnant with the environment and faeces. Since no haemorrhagic enteritis was found in the post-mortem examination, contamination of the external umbilical remnant was considered the more likely cause of the problem despite the fact that navel disinfection with chlorhexidine 1% was performed. Unfortunately, in the case detailed here, culture of faeces was performed neither from the mare nor from the colt in order to detect the organism or their toxins.

Clostridium botulinum has also been isolated from foal navel, but it has not yet been identified as an underlying cause of uroperitoneum (Swerczek, 1980). Neither neurological signs nor umbilical abscesses were noticed in this colt, discarding this pathogen from the reckoning.

Hyperlipaemia is a rare condition in the newborn foal, and is more common in mature donkeys, ponies and miniature horses with a negative energetic balance (Hughes et al., 2002). The equine neonate is unlikely to mobilise and deposit fat as easily as do adults, due to its poor reserves. Neonatal hyperlipaemia may be associated with congenital disease (Gilbert, 1986), azotaemia, with cortisol and thyroid hormones abnormalities or with enhanced glucocorticoides secretion induced by stress (Tan et al., 2005). Nutritional support is the main goal in hyperlipaemia treatment. In the case described here the enteral nutrition consisted of administration of maternal milk. Parenteral nutrition was not used due to the prohibitive cost. Neither insulin nor heparin was administered to this foal due to the lack of published data on the effectiveness of these treatments in equine neonates. It has been demonstrated that the use of formulas containing lipids for parenteral nutrition increases the likelihood of inducing hyperlipaemia in septic foals and this is related to poor prognosis (Myers et al., 2008). Whether the hyperlipaemia in the foal presented here could be associated with the development of septicemia remains obscure. More studies are necessary to elucidate the link between hyperlipaemia and septicemia in equine neonates.

In conclusion, the work reported here describes a clinical case of a foal with a failure in the passive transfer of immunoglobulins and hyperlipaemia associated with an atypical presentation of uroperitoneum. The uroabdomen was secondary to the rupture of the urachus associated with an infection of the internal umbilical remnant by *Clostridium* spp.

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