### Title

Congenital urachal and urinary bladder defects leading to uroperitoneum in a neonatal quarter horse colt.




### Journal

The Canadian veterinary journal. La revue veterinaire canadienne, 65(11)

### ISSN

0008-5286



### Authors

Karam, Bruno

Arndt, Stefanie

Magdesian, K

et al.



### Publication Date

2024-11-01

Peer reviewed

# CASE REPORT
RAPPORT DE CAS

# Congenital urachal and urinary bladder defects leading to uroperitoneum in a neonatal quarter horse colt

Bruno Karam, Stefanie Arndt, K. Gary Magdesian, Tom Cullen, Julie E. Dechant

## ABSTRACT

A newborn (5 h old) quarter horse colt was presented because of lethargy and severe abdominal distention. Uroperitoneum was suspected during initial workup, based on sonographic imaging and peritoneal fluid analysis. Definitive diagnosis was confirmed during exploratory celiotomy. Surgery revealed a congenitally abnormal allantoic stalk/urachal remnant and a failure of embryological fusion of the dorsal bladder wall. Recovery was successful and the animal is now a healthy, 4-year-old western performance gelding. These specific congenital abnormalities have not been previously documented in the peer-reviewed literature.

Key clinical message:

Congenital abnormalities of the urachus and the urinary bladder should be suspected in foals with uroperitoneum at birth. Cases involving congenital abnormalities of the urachus and urinary bladder might have favorable prognoses if animals retain adequate function of the urogenital tract and do not have secondary complications before and following surgery.

## RÉSUMÉ

**Anomalies congénitales de l'ouraque et de la vessie urinaire conduisant à un uropéritoine chez un poulain quarter horse nouveau-né**

Un poulain quarter horse nouveau-né (âgé de 5 h) a été présenté en raison d'une léthargie et d'une distension abdominale sévère. Un uropéritoine a été suspecté lors du bilan initial, sur la base d'une imagerie échographique et d'une analyse du liquide péritonéal. Le diagnostic définitif a été confirmé lors d'une cœliotomie exploratoire. La chirurgie a révélé une tige allantoïdienne/restant de l'ouraque congénitalement anormal et un échec de la fusion embryologique de la paroi dorsale de la vessie. La guérison a été réussie et l'animal est maintenant un hongre western performant en bonne santé, âgé de 4 ans. Ces anomalies congénitales spécifiques n'ont pas été documentées auparavant dans la littérature évaluée par des pairs.

Message clinique clé :

Les anomalies congénitales de l'ouraque et de la vessie urinaire devraient être suspectées chez les poulains présentant un uropéritoine à la naissance. Les cas impliquant des anomalies congénitales de l'ouraque et de la vessie urinaire pourraient avoir un pronostic favorable si les animaux conservent une fonction adéquate du tractus urogénital et ne présentent pas de complications secondaires avant et après la chirurgie.

(Traduit par Dr Serge Messier)

Can Vet J 2024;65:1119–1123

Pilchuck Veterinary Hospital, Snohomish, Washington, USA (Karam); Department of Clinical Sciences, Cummings School of Veterinary Medicine, Tufts University, North Grafton, Massachusetts, USA (Arndt); Department of Surgical and Radiological Sciences (Magdesian) and Department of Medicine and Epidemiology (Dechant), School of Veterinary Medicine, University of California, Davis, California, USA; Equine Medical Center of Ocala, Ocala, Florida, USA (Cullen).

Address all correspondence to Stefanie Arndt; email: stefanie.arndt@tufts.edu

Bruno Karam and Stefanie Arndt contributed equally to this work.

Explicit informed consent was received for the client-owned animal.

## CASE DESCRIPTION

### History

A 5-hour-old quarter horse colt weighing 45.2 kg was presented to the William R. Pritchard Veterinary Medical Teaching Hospital because of abdominal distention, evident since birth, as well as lethargy, absence of a suckle reflex, and mild hemorrhagic discharge from an enlarged umbilical stump. The colt was weak, unable to stand, and had not nursed prior to presentation. The colt was the result of an embryo transfer to a maiden recipient mare that carried the pregnancy to term and foaled unassisted. No other foals at the owner's property were reported to have had any medical issues.

### Presentation

At presentation, the colt was dull and in left lateral recumbency in the horse trailer. Despite being stimulated, he was unable to stand. The foal's heart rate was 100 beats per minute, respiratory rate was 60 breaths per minute, and rectal temperature was 36.6°C. Mucous membranes were mildly hyperemic and tacky with a capillary refill time of 1 s. The colt had severe abdominal and umbilical stump distention and blood was dripping from the urachus. Gastrointestinal motility was absent on abdominal auscultation, no cardiac murmurs were detected, and auscultation of lungs revealed mild crackles diffusely throughout the left lung field. Supplemental oxygen was administered at a rate of 5 L/min.

Venous blood gas analysis revealed hyponatremia [127 mmol/L, reference range (RR): 131 to 144 mmol/L] and acidemia (pH: 7.212, RR: 7.350 to 7.450). The acidemia was associated with hyperlactatemia (5.5 mmol/L, reference: $< 3.0$ mmol/L). The plasma potassium concentration was 4.8 mmol/L (RR: 3.2 to 4.9 mmol/L). Azotemia was present, including increased creatinine (17.4 mg/dL, RR: 1.2 to 2.4 mg/dL). A complete blood (cell) count revealed a leukopenia (4230 cells/μL, RR: 5300 to 14 000 cells/μL) with a mild neutropenia (3198/μL, RR: 3400 to 11 900/μL). Mild hyperfibrinogenemia was present (400 mg/dL, RR: 100 to 300 mg/dL).

Diffuse free peritoneal fluid that was anechoic with hyperechoic particulates was detected on abdominal ultrasonographic examination. The umbilical arteries had normal size and echogenicity when traced to the collapsed bladder. The umbilical vein and the urachus were not visualized in entirety due to excessive free peritoneal fluid. A single cyst $\leq 3$ cm in diameter was present in each kidney.

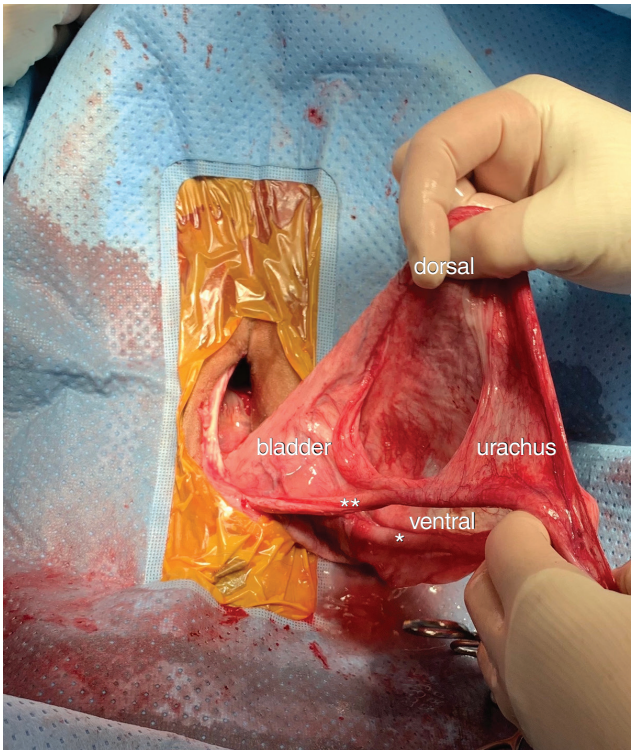


**FIGURE 1.** Intraoperative photograph taken after resection of the umbilical vein in a neonatal colt. Note the caudal defect in the enlarged and abnormally formed urachal remnant, contiguous with a caudoventral defect in the bladder. In addition, a craniodorsal bladder defect is present close to the right umbilical artery (**). Left umbilical artery (*). Cranial direction is to the top of the image.

The remainder of the ultrasound examination revealed no clinically relevant abnormalities.

Abdominocentesis revealed a grossly dark-yellow-to-brown-colored fluid with free-floating particulates. The abdomen was drained of ~3 L of fluid over 2 h, while intravenous (IV) fluid therapy was initiated simultaneously. Peritoneal fluid analysis revealed potassium concentration of 3.9 mmol/L, creatinine concentration $> 22$ mg/dL (above the upper limit of the analyzer), glucose concentration of 62 mg/dL (peripheral glucose: 97 mg/dL), lactate concentration of 3.2 mmol/L, and pH of 7.234. The peritoneal fluid was cytologically categorized as a pure transudate with a total protein of 1.4 g/dL and total nucleated cell count of 80 cells/μL with 84% nondegenerate neutrophils. No evidence of infectious organisms was noted on cytological examination. The origin of the fluid particulates was not evident on cytology. An initial IV bolus (20 mL/kg) of isotonic fluids with 2% dextrose was administered over 45 min. This was followed by isotonic fluids at a rate of 4 mL/kg per hour and an IV dextrose infusion of 4 mg/kg per minute. A continuous electrocardiogram completed at the time of admission revealed normal sinus rhythm.

Based on the history, physical examination, sonographic findings, and peritoneal fluid potassium and creatinine concentrations, a presumed diagnosis of uroperitoneum was made, even though peritoneal creatinine concentration was not documented to be at least twice as high as the plasma creatinine concentration since the value was above the analyzer's upper limit of quantification. Near equilibration of potassium in the serum and abdominal fluid and markedly increased serum creatinine concentration at such an early time point after birth were suggestive of a chronic, likely *in utero,* congenital urinary ascites, considering the foal's young age (5 h). Uroperitoneum in neonatal foals is usually secondary to trauma during parturition or postpartum infection or trauma (1). The consideration of *in utero* uroperitoneum was consistent with the foal's history of being born with preexisting abdominal distention.

Approximately 2.5 L of urine was drained from the abdomen. Hyperimmune plasma (1 L) was administered IV due to concerns of failure of passive transfer and delayed colostral ingestion, given that the colt had not nursed before presentation. After medical stabilization, exploratory celiotomy was conducted to investigate, treat, and further characterize the ascites. Perioperative antimicrobial therapy with ceftiofur sodium (5.5 mg/kg, IV, q12h) was initiated.

## Surgery

The colt was premedicated with hydromorphone (0.01 mg/kg, IV) and midazolam (0.1 mg/kg, IV) and induced with ketamine (2.2 mg/kg, IV). Anesthesia was maintained with sevoflurane in oxygen, which was administered to effect and adjusted to achieve surgical depth of anesthesia. The colt was placed in dorsal recumbency. An 8 French infant feeding tube (Kangaroo PVC, 8 Fr × 107 cm) used as a urinary catheter was placed aseptically in the bladder and the penis was secured between the hind limbs, away from the surgical site. After the ventral abdomen was clipped, the umbilicus was oversewn with an inverting suture pattern using 2-0 polypropylene suture (Prolene; Ethicon US, Cincinnati, Ohio, USA). Aseptic preparation of the abdomen was completed with 2% chlorhexidine gluconate and isotonic 0.9% sterile saline solutions.

A fusiform incision was made through the skin and subcutaneous tissues, extending 4 cm caudal and 4 cm cranial to the umbilicus. The incision was extended down to the umbilical ring. An incision was made slightly to the right of the *linea alba,* 1.5 cm cranial to the umbilicus, with care to not incise or transect the umbilical vein. The peritoneal cavity was opened digitally, and vacuum suction was used to evacuate the fluid contents within the abdominal cavity. The umbilical stump was dissected from the body wall. The umbilical vein was identified, isolated, and double-ligated with 3-0 polydioxanone suture (PDS; Ethicon US). The urachal remnant was enlarged and abnormally formed, with a large defect in the caudal aspect (Figure 1). Cranial tension was applied to the umbilicus to exteriorize the ventral portion of the bladder. The abnormal urachal remnant and associated defect were contiguous with the ventral aspect of the bladder, which also had a large ventral defect.

The umbilical arteries were identified and isolated, and 3-0 polydioxanone suture (PDS; Ethicon US) was used to double-ligate both arteries. Two stay sutures were placed into the ventral aspect of the bladder, abaxial to the arteries, using 0 polydioxanone suture (PDS; Ethicon US). The abnormal urachal remnant on the ventral aspect of the bladder was resected using Mayo scissors. The defect in the ventral bladder was then sutured in a simple continuous pattern using 3-0 poliglecaprone 25 suture (Monocryl; Ethicon US). The primary closure layer was then over sewn in a Cushing pattern with 3-0 poliglecaprone 25 suture (Monocryl; Ethicon US).

Sterile isotonic 0.9% saline was infused into the bladder *via* the pre-placed urinary catheter. The bladder distended with no evidence of leakage from the sutured defect. The abdominal cavity was thoroughly lavaged with sterile isotonic 0.9% saline. The external rectus sheath of the rectus abdominus muscle was closed in a simple continuous pattern using 2 polyglactin 910 suture (Vicryl; Ethicon US). The subcutaneous tissue was closed in a continuous pattern using 2-0 poliglecaprone 25 suture (Monocryl; Ethicon US). An intradermal pattern using 3-0 poliglecaprone 25 suture (Monocryl; Ethicon US) was used to close the skin. The incision was covered with sterile, 4 × 4 gauze and an iodine-impregnated drape for recovery. Recovery from general anesthesia was uneventful.

## Postoperative management

Banked colostrum acquired from the mare was administered *via* a nasogastric tube, as the colt was still ≤ 24 h of age. The plasma IgG concentration at 18 h of age was > 800 mg/dL using a semiquantitative immunoassay (SNAP foal IgG test; IDEXX Laboratories, Westbrook, Maine, USA). The colt was maintained on IV fluids, along with ceftiofur, as previously described. Sucralfate (22 mg/kg, PO, q6h), vitamin E (10 IU/kg, PO, q24h), and lactase enzyme (60 FCCU/kg, PO, with every milk feeding) were added. Selenium (2.5 mg, IM, once) was also administered, considering the prevalence

of selenium deficiency in the geographic region. A urinary catheter was maintained for 5 d following surgery.

Administration of milk from the dam was achieved using a nasogastric tube, initially at 5% of body weight per day. The volume was increased every 12 h, by 2.5% of body weight per day, until the colt was able to nurse from the mare independently. Intravenous fluids were continued (constant-rate infusion, 5 mL/kg per hour) following surgery, with dextrose supplementation (4.0 mg/kg per minute). Rates were adjusted based on serial monitoring of serum creatinine, glucose, and electrolyte concentrations, and an increasing nutritional plane during subsequent days of hospitalization. Serum creatinine concentration decreased to 8.7 mg/dL 12 h postoperatively, and to 2.1 mg/dL 36 h postoperatively. At discharge, the serum creatinine concentration was 1.0 mg/dL.

The colt remained separated from the mare for ~60 h postoperatively, to ensure that production from the urinary catheter could be monitored. Due to the unusual nature of the case and concerns over bladder function due to congenital defects, the urinary catheter was left in place for 5 d. The colt was observed to urinate normally after removal of the urinary catheter.

Once introduced to the mare, the colt was able to nurse unassisted after ~24 h. Intravenous fluids were discontinued at 3 d postoperatively. The nasogastric feeding tube remained in place for 3 d, until the foal was able to sustain normal electrolyte and glucose concentrations through nursing alone.

On Day 6 of hospitalization, antibiotic therapy was adjusted to sulfadiazine-trimethoprim suspension (24 mg/kg, PO, q12h). At that time, urine specific gravity was 1.002 and there was no evidence of erythrocytes or heme proteinuria. The colt was discharged from the hospital on Day 7 and received an additional 3 d of antimicrobial therapy. Recommendations included confining the mare and foal to a stall for an additional 7 to 10 d, then housing in a stall with a small paddock for an additional 2 wk, to facilitate incision healing. The colt was monitored for the following 2 wk by the referring veterinarian. The patient is now a 4-year-old gelding with no known complications associated with the urogenital tract or surgery.

## DISCUSSION

Uroperitoneum is one of the most common urogenital disorders in neonates (1). It is widely accepted that colts may be at higher risk for bladder rupture during parturition, whereas fillies may be at higher risk for developing birth-related uroperitoneum (1–3). No sex predisposition seems to be noted in cases of uroperitoneum in foals secondary to postpartum trauma, necrotizing cystitis, or septic urachitis/omphaloarteritis (2,4,5). The urinary bladder is commonly the origin of urinary leakage (73.1%), followed by the urachus (21.6%) and the ureter (5.2%) (6). Most commonly, foals with uroperitoneum have a normal abdominal contour at birth and develop abdominal distention during the hours or days after rupture. The case presented in this report did not fall under the previously mentioned categories.

Neonates presented with uroperitoneum commonly exhibit hyponatremia, hypochloremia, hyperkalemia, and azotemia. A creatinine concentration in the peritoneal fluid that is twice or more that in the serum is consistent with a diagnosis of uroperitoneum. Some of these parameters were absent in this case, and the ratio of serum:peritoneal creatinine could not be assessed because the peritoneal creatinine concentration exceeded the analyzer's upper limit of quantification (> 22 mg/dL). The high creatinine concentration reflects the uroperitoneum, with distribution of urine creatinine across the peritoneum over time. The magnitude leads the authors to hypothesize that the uroperitoneum was likely present *in utero;* alternatively, the chronicity and development of high serum creatinine can also occur with spurious hypercreatininemia, reflecting fetal stress or placental dysfunction. However, considering the foal's congenital defect, the authors believe the long-term uroperitoneum was responsible for the very high serum creatinine concentration measured shortly after birth.

This case illustrated an uncommon cause of uroperitoneum in an equine neonate that should be considered as a differential diagnosis for foals presented with uroperitoneum or ascites soon after birth. The foal in our case was presented with a congenital abnormality in the fusion of the ventral bladder and development of the urachus, characterized by a failure of the allantois (allantoic cavity) to form a complete urachal remnant connecting the bladder to the external umbilicus. Renal cysts did not appear to be clinically significant in this case, based on the rapid resolution of the post-renal azotemia, repeated examinations, and successful outcome, but may have been related congenital defects. The urachus connects the fetal bladder to the allantoic cavity and represents the most proximal part of the allantois, marking the site of origin of the allantois from the early hindgut (7).

Inspection of the placenta to further characterize the anomaly would have been ideal; however, it was not available to the attending clinicians as the colt was not born in

hospital. Congenital abnormalities involving embryological development of the bladder in foals are considered rare, with an unknown prevalence. Such defects may include bladder agenesis, hypoplasia, or failure of the bladder to fuse to the external umbilicus (8). Dorsal bladder wall defects with smooth edges have been suspected to represent developmental, rather than traumatic, lesions. Two full siblings developed uroperitoneum with dorsal bladder wall defects that had smooth margins and a lack of appreciable inflammation, implying a developmental origin (8,9). A report from 1954 described a congenital ventral bladder and urachal lesion similar to that in this report, with the ventral portion of the bladder absent between the lateral ligaments (and the umbilical arteries) from the umbilicus to the urethra. This was also suspected to be a developmental anomaly causing uroperitoneum (10). However, that foal died during examination, and it is unknown whether the bladder would have been surgically salvageable.

Another reported congenital anomaly of the bladder or urachus included anomalous fusion of the bladder to the inner umbilical ring with absence of the urachus (11), which led to a markedly enlarged bladder, termed megavesica, without bladder wall defects or uroperitoneum. In a different report, an enlarged bladder in a foal was attributed to an adhesion of the bladder to the umbilical remnant; that foal survived with resection of 50% of the enlarged bladder (12). Two other foals were reported to have megavesica or grossly distended bladders thought to have occurred due to chronic bladder distention *in utero*. There was a loss of smooth muscle in the dorsal region of the bladder, with replacement by collagen; this was thought to result in bladder rupture during foaling, with 1 foal recovering with surgery (13) and the other dying during surgery (14). After effective perioperative medical stabilization, animals in 63.6% of the cases survived following surgery (6). The most common complication was recurrence of the uroperitoneum (16 to 20%) due to disruption of the suture line or an incomplete closure of the defect (15,16).

In the case described here, no complications were observed. The colt had an indwelling urinary catheter for 5 d, which likely reduced the risks of recurrence of uroperitoneum and disruption of the suture line. The bilateral renal cysts were an interesting finding. It is possible that they, along with the urachal anomaly, represented a complex congenital defect of the urogenital tract.

Contrast cystography may be useful for detecting uroperitoneum in foals (9). This procedure might have led to a definitive diagnosis in the present case but was not done. Sonographic findings of hypoechoic peritoneal fluid, the degree of azotemia, and concurrent small, collapsed bladder were highly supportive of uroperitoneum. An alternative cause of free abdominal fluid may have been intra-abdominal hemorrhage or chylous effusion. This was ruled out by the abdominocentesis and lack of supporting evidence on the hemogram. Definitive diagnosis of uroperitoneum was obtained through exploratory celiotomy.

Prognoses of similar cases may be presumed favorable if animals retain adequate function of the urogenital tract and do not have secondary complications before and following surgery. CVJ

## REFERENCES


1. Mackenzie HC. Disorders of foals. In: Reed SM, Bayly WM, Sellon DC, eds. Equine Internal Medicine. 4th ed. St. Louis, Missouri: Elsevier Mosby, 2018:1406–1410.
2. Lester GD. Manifestation and management of disease in foals. In: Smith BP, Van Metre DC, Pusterla N, eds. Large Animal Internal Medicine. 5th ed. St. Louis, Missouri: Elsevier Mosby, 2015:275–276.
3. Sprayberry KA. The urinary system. In: McAuliffe SB, Slovis NM, eds. Color Atlas of Diseases and Disorders of the Foal. 1st ed. Edinburgh, Scotland: Elsevier Saunders, 2010:167–188.
4. Robertson JT, Embertson RM. Surgical management of congenital and perinatal abnormalities of the urogenital tract. Vet Clin North Am Equine Pract 1988;4:359–379.
5. Dunkel B, Palmer JE, Olson KN, Boston RC, Wilkins PA. Uroperitoneum in 32 foals: Influence of intravenous fluid therapy, infection, and sepsis. J Vet Intern Med 2005;19:889–893.
6. Bernick A, Nieth J, Wehrend A. Uroperitoneum beim Fohlen: Eine Literaturübersicht. Tierärztl Prax Ausg G Grosstiere Nutztiere 2021;49:41–50.
7. Latshaw WK. Embryonic membranes and placentation. In: Latshaw WK, ed. Veterinary Developmental Anatomy: A Clinically Oriented Approach. Toronto, Ontario: BC Decker, 1987:49–74.
8. Graves EA. Congenital disorders of the urinary tract. In: Sprayberry KA, Robinson NE, eds. Robinson's Current Therapy in Equine Medicine. St. Louis, Missouri: Elsevier Saunders, 2015:445–447.
9. Wellington JKM. Bladder defects in newborn foals. Aust Vet J 1972;48:426.
10. Bain AM. Diseases of foals. Aust Vet J 1954;30:9.
11. Dubs VB. Megavesica zufolge Urachusmangel bei einem neugeborenen Fohlen. Schweiz Arch Tierheilkd 1976;118:395.
12. Adams RA, Koterba AM, Cudd TC, *et al.* Exploratory celiotomy for suspected urinary tract disruption in neonatal foals: A review of 18 cases. Equine Vet J 1988;20:13.
13. Rossdale PD, Greet TRC. Megavesica in a newborn foal. Int Soc Vet Perinat Newslett 1989;2:10.
14. Oikawa M, Yoshihara T, Katayama Y, *et al.* Ruptured bladder associated with smooth muscle atrophy of the bladder in a neonatal foal. Equine Pract 1993;15:38.
15. Ford MG, Nelson BB, Ford TS, *et al.* Complications and comorbidities in foals undergoing surgical repair for uroperitoneum. J Equine Vet Sci 2022;110:103852.
16. Kablack KA, Embertson RM, Bernard WV, *et al.* Uroperitoneum in the hospitalised equine neonate: Retrospective study of 31 cases, 1988–1997. Equine Vet J 2000;32:505–508.