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Case Report

Multiple congenital urogenital abnormalities in a Tennessee Walking Horse colt

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Summary

A 9-month-old, Tennessee Walking Horse colt was examined for urinary incontinence. Cystoscopy revealed a single identifiable ureter that appeared abnormal, and sabulous urolithiasis. Only the left kidney, which appeared lobulated and hydronephrotic, could be located using ultrasound. Results of serum chemistries were consistent with renal failure. Necropsy revealed unilateral agenesis of the right kidney and ureter with severe left ureterolithiasis, bilateral cryptorchidism and segmental aplasia of the *ductus deferens*. Histopathological examination revealed cystitis and diffuse interstitial fibrosis of the left kidney. Congenital malformations should be included as differential diagnoses for urinary incontinence and urogenital disease. Ultrasonography and cystoscopy can be useful to diagnose suspected agenesis of the urinary system in adult horses.

Introduction

There are few reports of unilateral renal agenesis in horses (Johnson *et al.* 1976; Huston *et al.* 1977). Renal agenesis and ectopic ureter result from abnormal development and fusion of the embryonic mesonephric or metanephric tissue and metanephric ducts (Noden and de Lahunta 1985a; Blikslager and Green 1992; Graves 2003). Unilateral renal and ureteral agenesis has been reported in a 4-year-old American Quarter Horse gelding examined for dysuria, polyuria, polydipsia, inappetence and weight loss (Johnson *et al.* 1976). The horse also had absence of the internal inguinal ring and *ductus deferens* with contralateral hydronephrosis due to ureterolith obstruction. Serum chemistry and urinalysis abnormalities and were consistent with renal azotaemia.

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Case details

Case history and physical examination

A 9-month-old, Tennessee Walking Horse colt was examined for urinary incontinence. The colt was reported to have dribbled urine for the past 3 months, although urine scalding of the hindlimbs and associated dermatitis had been present for only 2 weeks. The exact history of urinary incontinence was unknown, as the colt had been turned out at pasture until recently when it had begun training. The colt was able to posture and urinate normally. Physical examination was normal except that the colt was bilaterally cryptorchid and there was apparent urine scalding of the distal hindlimbs. Body condition score was good (BCS 6/9). No abnormalities were found during neurological examination.

Clinicopathological findings

Results of a complete blood count at admission indicated anaemia (packed cell volume 26.7%; reference range [rr] 32–48%). Abnormalities of the serum chemistry panel included elevated concentrations of blood urea nitrogen (BUN) (15.2 mmol/l; rr 3.2–8.6 mmol/l), creatinine (221 µmol/l; rr 0–177 µmol/l) and bicarbonate (32.4 mmol/l; rr 21–30 mmol/l). Other serum electrolyte concentrations were within expected ranges for a horse of this age. Creatinine (230 µmol/l) and BUN (15.9–17.6 mmol/l) remained elevated on Days 2 and 4 of hospitalisation.

Endoscopic examination

Cystoscopy was performed after sedation with detomidine (0.01 mg/kg bwt, i.v.) to examine the colt for

an ectopic ureter. Only the left ureter could be identified endoscopically. The ureteral opening appeared to be larger and more caudally located than normal, near the neck of the bladder and pelvic urethra (**Fig 1**). Normal appearing urine could be seen emptying from the single ureter. Urine appeared to steadily dribble from the ureter and the normal occasional pulsatile discharge of urine was not observed. A sample of urine was obtained from the bladder lumen and urinalysis was within normal limits. There was a mild amount of sabulous urolithiasis in the ventral bladder (**Fig 2**) and the *colliculus seminalis* could not be readily identified in the pelvic urethra (**Fig 3**).

Ultrasound examination

A transabdominal ultrasound examination was then performed using an HDI 5000 ultrasound machine with a C5-2 transducer[1] and only the left kidney could be identified. The left kidney appeared lobulated and hydronephrotic with a dilated renal pelvis (**Figs 4** and **5**).

Outcome

The owner elected euthanasia because of the guarded prognosis for resolution of urinary incontinence and clinicopathological evidence of renal failure with only one identifiable and ultrasonographically abnormal kidney.

Post mortem *findings*

Endoscopic and ultrasonographic findings were confirmed at necropsy. The right kidney and ureter were absent and residual renal tissue could not be identified. The left kidney was moderately enlarged (renal hypertrophy), the major and minor calyces were dilated and filled with clear serous fluid (hydronephrosis). Multiple triangular to polygonal, pale tan foci extended from the renal cortex to the medulla. The left ureter was markedly dilated (luminal diameter 7–8 mm) and the ureteral *tunica muscularis* was hypertrophied. Several ureteroliths were located approximately 20 cm distal to the kidney. The left ureter entered the bladder trigone at an oblique angle and the ureteral orifice was enlarged (approximately 1 cm in diameter). A right ureteral orifice could not be identified. The trigone area and neck of the bladder were thickened (approximately 1.5 cm) due to diffuse submucosal oedema and fibrosis and hypertrophy of the *tunica muscularis* (8 mm thick). The *colliculus seminalis* and associated ejaculatory orifices could not be identified on the mucosal surface of the proximal urethra, and the ampullae ended in small (1–2 mm) blind-ended cords dorsal to the trigone of the bladder (segmental aplasia of the *vas deferens*). Both testes were small and retained within their respective inguinal canals. Each testis

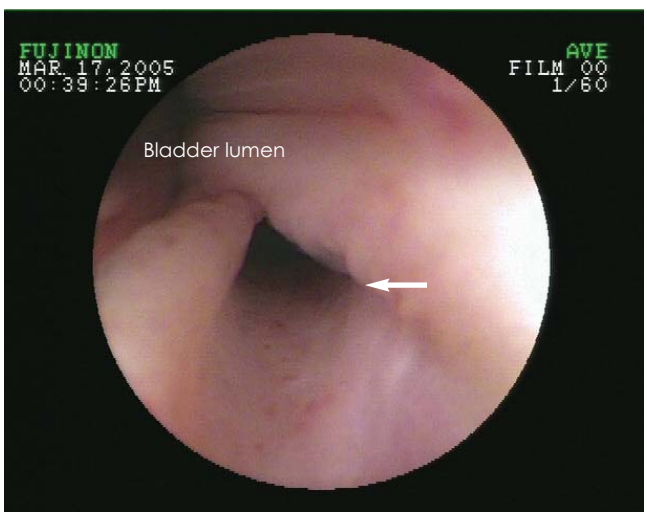

Fig 1: A single ureter (arrow) could be observed near the bladder trigone endoscopically. The ureter is enlarged and appeared more caudally positioned than normal. The bladder lumen is to the upper left of the figure.

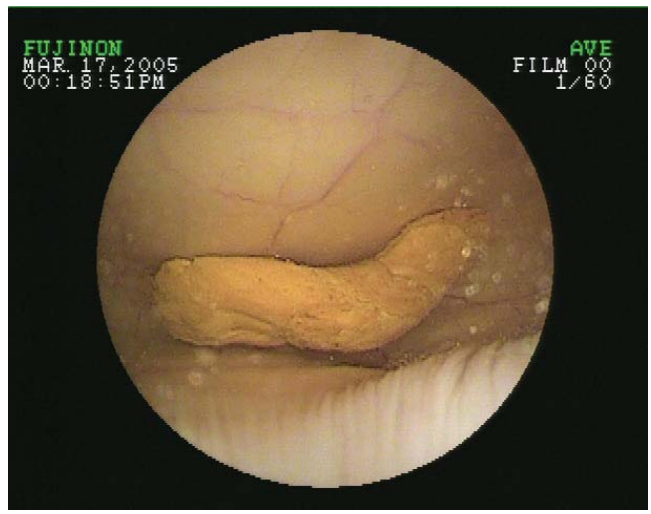

Fig 2: Cystoscopy revealed a mild amount of sabulous urolithiasis in the bladder.

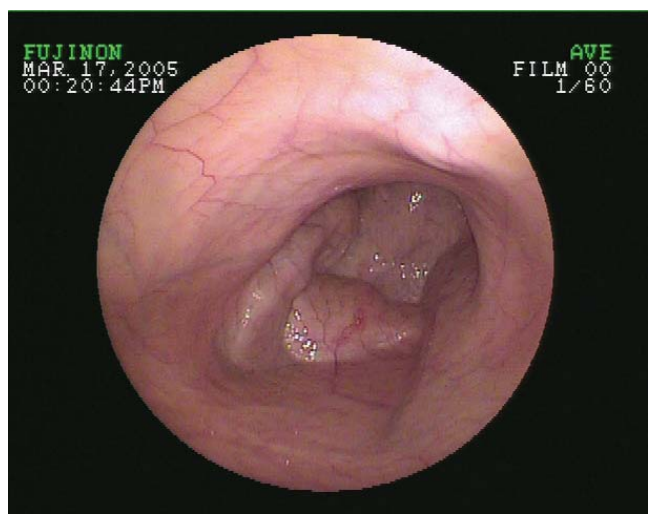

Fig 3: Agenesis of the* colliculus seminalis *observed at the pelvic urethra.

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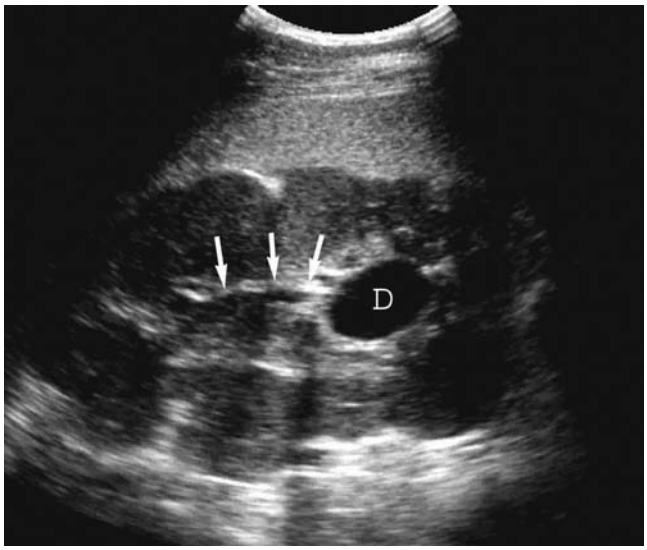

Fig 4: Transabdominal longitudinal sonographic image of the left kidney showing the renal pelvis (arrows) and a dilated portion of the renal pelvis (D).

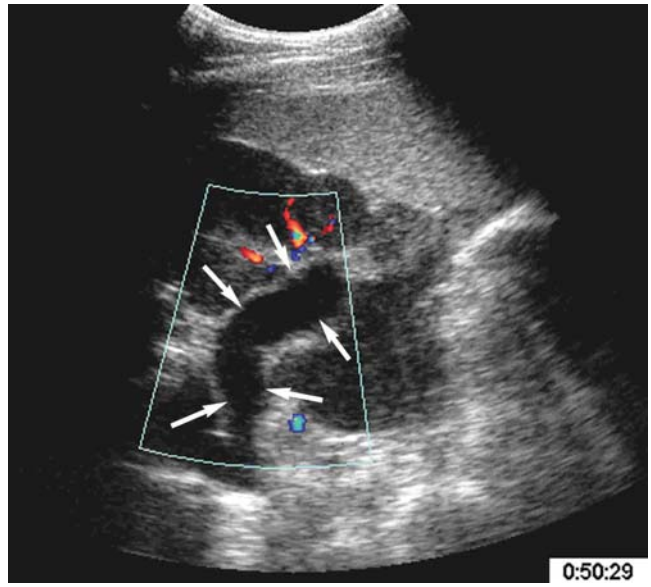

Fig 5: Transabdominal transverse sonographic image of the left kidney showing dilation of the renal pelvis (arrows).

was found with its corresponding testicular vessels and *ductus deferens*.

Microscopically, pale tan foci observed grossly in the left kidney were identified as diffuse interstitial fibrosis of the renal cortex and medulla, consistent with healed lesions of pyelonephritis. Associated tubules and glomeruli were atrophic and small aggregates of lymphocytes and plasma cells were scattered in the fibrotic interstitium. Dense aggregates of lymphocytes, plasma cells, and few neutrophils partially surrounded small arcuate arteries and veins at the corticomedullary junction. Extensive oedema and fibrosis expanded the submucosa of the left ureter and the neck region of the urinary bladder and extended into the *tunica muscularis*, separating muscle bundles. Small aggregates of lymphocytes and plasma cells were scattered in the superficial submucosa. In the urinary bladder and trigone, many myofibres were small, elongated, and contained multiple nuclei. Other myofibres were swollen and hyalinised.

Discussion

In this case, complete absence of the right kidney and ureter indicate failure of the right ureteric bud to develop from the mesonephric duct and subsequent failure to induce differentiation of the metanephric blastema on the right side. Segmental aplasia of the *ductus deferens* probably also resulted from improper development and maturation of the mesonephric ducts (Noden and de Lahunta 1985b). Ipsilateral reproductive system abnormalities are commonly associated with renal agenesis (Johnson *et al.* 1976; Huston *et al.* 1977; Graves 2003). There was no history of maternal illness or treatments during gestation that may have led to the congenital defects present in this colt.

Urinary incontinence may have been the result of an abnormally positioned ureter, although histological evidence of bladder and ureteral myositis, oedema and fibrosis probably contributed to poor bladder and urethral sphincter tone. Endoscopically, the left ureter was large and appeared to be more caudally located than normally expected. At necropsy, the single ureter was enlarged and entered the bladder trigone at an oblique angle. Abnormal ureteral anatomy and positioning probably contributed to incontinence, but this is speculative and the histopathological changes of urinary tract myositis and fibrosis probably had a greater role in producing incontinence. During endoscopy, it was difficult to assess bladder and urethral sphincter tone. However, the left ureter lacked a definite sphincter and was enlarged with poor muscle tone. Urinary incontinence had been present for some time, but the colt had only recently been noticed to have urine scalding of the hindlimbs. Urinary incontinence may not be as prominent a clinical sign in male horses because of retrograde urine flow into the bladder and location of the ectopic ureter near the external urethral sphincter (Blikslager and Green 1992; Graves 2003). In foals, contrast studies such as i.v. pyelography can be useful to diagnose ureteral ectopy, but the adult size of this horse precluded abdominal radiography.

Ureterolithiasis in this case may have resulted from urinary stasis or sloughage of renal tubular cells due to chronic pyelonephritis that became niduses for urolith formation. Some clinicians speculate that urolithiasis may occur due to the lack of some inherent renally derived inhibitor of urinary crystal agglutination. It is possible that the horse in this report may have also had other congenital urinary system abnormalities that led to ureterolith formation.

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Manufacturer's address

[1]Phillips Medical Systems North America, Bothell, Washington, USA.

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