A 17-year-old Quarter Horse-Thoroughbred gelding was evaluated because of dysuria and inappetence of 2 days’ duration and weight loss of 10 days’ duration. The current owners had bought the horse 3 years previously; a prepurchase examination had not revealed any abnormalities. Nine months prior to referral, the owners had noticed that the horse frequently held its penis in a dropped position and that thick mucus dripped from the end of the penis. No treatment had been sought at that time, and clinical signs resolved within 2 weeks. Similar signs were seen 4 months later, but no abnormalities were detected by means of urethroscopy or cystoscopy, and results of a urinalysis and cytologic evaluation of a urine sample were normal. The horse was vaccinated against eastern, western, and Venezuelan encephalomyelitis; equine influenza; tetanus; and West Nile virus infection 2 weeks prior to referral. Its diet consisted of 0.5 kg (1.1 lb) of dry cob, 0.5 kg of grain pellets, 1.7 kg (3.7 lb) of beet pulp, 0.5 kg of bran, and 4 flakes of grass hay daily. The horse was dewormed with fenbendazole (10 mg/kg [4.5 mg/lb], PO) 60 days prior to referral.

Abnormalities detected during the initial physical examination included dysuria, hematuria limited to the last portion of voided urine, inappetence, and scalding of the cranial aspects of the distal portions of the hind limbs. Heart rate was 48 beats/min. The bladder wall appeared to be thicker than normal during palpation per rectum. The penis was manually extruded without sedation, and reddish urine was seen around the external urethral opening. There was no external evidence of damage to the penile shaft or preputium, and the urethral opening was free from smegma. Differential diagnoses for hematuria that were considered at this time included renal neoplasia, cystitis, pyelonephritis, pigmenturia, drug-induced nephropathy, and urethral defects.

A CBC and serum biochemical profile were performed. Abnormalities that were detected included leukocytosis (15,400 cells/µL; reference range, 6,000 to 12,000 cells/µL) with mature neutrophilia (9,670 cells/µL; reference range, 3,000 to 6,000 cells/µL), hyperfibrinogenemia (500 mg/dL; reference range, 100 to 400 mg/dL), and hyperbilirubinemia (3.6 mg/dL; reference range, 0.8 to 2.6 mg/dL). The hyperfibrinogenemia was considered to most likely be a result of inflammation, and the hyperbilirubinemia was considered to most likely be a result of anorexia.

Results of bilateral renal ultrasonography were normal. The gelding was sedated with detomidine hydrochloride (0.01 mg/kg [0.005 mg/lb], IV) and butorphanol tartrate (0.01 mg/kg, IV), and urethroscopy and cystoscopy were performed with a 100-cm videoendoscope. No abnormalities were seen during examination of the urethra and ureteral openings into the bladder. However, severe multifocal coalescing mucosal ecchymosis with luminal hemorrhage was seen during examination of the urinary bladder (Figure 1). Scattered accumulations of crystalloid sludge were seen on the ventral wall of the bladder, and diffuse ecchymoses were seen on the dorsal wall of the bladder.

Encrusted cystitis secondary to Corynebacterium matruchotii infection in a horse

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bladder. Biopsy samples of the bladder mucosa were obtained and submitted for histologic examination. A urine sample obtained with the endoscope was cloudy red with specific gravity of 1.010 (reference range, 1.008 to 1.020) and alkaline pH (pH, 9.0; reference range, 7.0 to 9.0). Analysis of the urine sample revealed pyuria (60 WBCs/hpf; reference range, <3 WBCs/hpf), hematuria (>500 RBCs/hpf), bacteriuria (>10 rod-shaped bacteria/hpf and <5 cocci/hpf), and numerous calcium carbonate crystals. A sample of the urine was submitted for bacterial culture, and after 3 days, a pure growth of *Corynebacterium* sp susceptible to penicillin, sulfonamide, and enrofloxacin was isolated. Findings were consistent with a diagnosis of encrusted cystitis exacerbated by sabulous urolithiasis.

A 14-gauge polypropylene catheter was inserted in the left jugular vein, and the horse was treated with penicillin G potassium (22,000 U/kg [10,000 U/lb], IV, q 6 h) for 3 days, followed by trimethoprim-sulfamethoxazole (25 mg/kg [11.4 mg/lb], PO, q 12 h) for an additional 4 weeks. Daily for the first 3 days of treatment, an endoscope was inserted into the bladder and the bladder was drained and then infused over a period of 20 minutes with a warmed balanced electrolyte solution (5 L) containing dimethyl sulfoxide (0.5 g/kg [0.23 g/lb]) in an effort to remove necrotic debris and crystalline sludge from the bladder. Dimethyl sulfoxide was used because of its locally acting analgesic, antibacterial, and anti-inflammatory properties.

The horse's appetite improved substantially on day 2, and no further episodes of dysuria or hematuria were noticed. Follow-up testing indicated that the leukocytosis, hyperfibrinogenemia, and hyperbilirubinemia had resolved by day 2. Histologic examination of the bladder mucosa biopsy specimens revealed severe diffuse neutrophilic cystitis with marked neovascularization and mineralized concretions (Figure 2). No fungal elements or neoplastic cells were seen.

On day 6, cystoscopy revealed resolution of the cystitis. Results of analysis of a urine sample obtained at this time were unremarkable, and bacterial culture of the urine sample did not yield any growth. Therefore, the horse was discharged from the hospital. Two weeks later, the owner reported that the horse had gained 100 kg (220 lb) and was urinating normally. Six weeks later, the owner reported that the horse had continued to gain weight. Results of analysis of a midstream urine sample collected at this time were unremarkable; no bacteria were seen during examination of the urine sediment.

To further characterize the bacterial isolate recovered from bladder mucosa biopsy specimens and the initial urine sample, a combination of biochemical tests, fatty acid analysis, and 16S rDNA sequencing was used. Results of fatty acid and biochemical tests were variable and could not be used to identify the isolate to the species level, probably because of limitations of the database. Molecular phylogenetic analysis based on the 16S rDNA gene sequence was used to identify and determine the phylogenetic position of the isolate. The 16S rDNA gene was amplified by means of a polymerase chain reaction assay, and analysis of assay results indicated that the isolate belonged to the genus *Corynebacterium*. Phylogenetic analysis of 16S rDNA sequence data indicated that the isolate was closely related to *Corynebacterium matruchotii*.

Urolithiasis, bladder neoplasia, bladder paralysis, and anatomic bladder defects may predispose horses to development of bacterial cystitis. Typical clinical signs include pollakiuria, hematuria, pyuria, and scalding of the hind limbs. Diagnostic evaluation includes physical examination, transrectal ultrasonography to rule out cystic calculi, urinalysis, and bacterial culture of a urine sample. Results of rectal palpation may be normal; however, in horses with chronic cystitis, the bladder wall may be palpably thicker than normal as was the case in the horse described in the present report. Bilateral renal ultrasonography will provide useful information on renal size and structure, and cystoscopy will allow examination of the bladder mucosa.

Submission of bladder mucosa biopsy specimens for histologic examination and bacterial culture may provide additional information, particularly in horses refractory to initial treatment. Organisms recovered from horses with cystitis include *Escherichia coli*, *Proteus* spp, *Klebsiella* spp, *Enterobacter* spp, *Corynebacterium* spp, *Streptococcus* spp, *Staphylococcus* spp, and *Pseudomonas* spp. Successful management of bacterial cystitis includes elimination of predisposing factors and systemic administration of appropriate antimicrobials.

Numerous *Corynebacterium* spp have been isolated from human patients. *Corynebacterium diphtheriae* causes respiratory tract failure, cutaneous ulceration, and neuronal and myocardial toxicoses. *Corynebacterium jeikeium* results in nosocomial infections in immunocompromised patients, and *Corynebacterium urealyticum* results in alkaline-encrusted...
Cystitis. Endocarditis and pneumonia can result from Corynebacterium pseudodiphtheriticum infection, pruri-
tus can result from Corynebacterium minutissimum infection, and exudative pharyngitis can result from Corynebacterium ulcerans infection.

Corynebacterium matruchotii has been used by a number of human researchers as a microbiologic model for intracellular calcification. It has been reported to be a cause of bacterial keratitis and is a microbial inhabitant of the oral cavity. In the oral cavity, C. matruchotii causes dental calculus formation by precipitation of calcium and inorganic phosphorous, which is necessary for hydroxyapatite formation.

In the horse described in the present report, the encrusted cystitis and sabulous urolithiasis were most likely a result of infection with C. matruchotii. Similar findings have been reported in human patients. Complications reported in human patients with encrusted cystitis include septic shock, urethral stenosis, and encrusted pyelitis. In some instances, surgical excision of the encrusted bladder mucosa is required.

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