A 5-year-old 5.7-kg (12.5-lb) spayed female Yorkshire terrier mix was referred to VCA Animal Specialty Group because of continued stranguria 1 day after a urethral obstruction was relieved by the referring veterinarian. The patient had initially been evaluated by the referring veterinarian because of hematemesis and bleeding from the vulva. Results of physical examination performed by the referring veterinarian indicated the dog was dehydrated (5%) with hemorrhagic vulvar discharge and a palpably large and firm urinary bladder; signs of pain were elicited during palpation of the urinary bladder. Abdominal radiography revealed the urinary bladder was severely distended with fluid and contained multifocal eccentrically located areas of mineral opacity (Figure 1). Results of a CBC indicated polycythemia (9.54 × 10^6 RBCs/µL [reference interval, 5.5 × 10^6 RBCs/µL]; PCV, 60% [reference interval, 37% to 55%]) and lymphopenia (480 cells/µL; reference interval, 1,000 to 4,800 cells/µL); these results were attributed to hemoconcentration and a stress response. Results of serum biochemical analyses indicated azotemia (BUN concentration, 78 mg/dL [reference interval, 7 to 20 mg/dL]; creatinine concentration, 1.7 mg/dL [reference interval, 0.3 to 1.4 mg/dL]); hyperphosphatemia (6.9 mg/dL; reference interval, 2.9 to 6.6 mg/dL); hypokalemia (135 mmol/L; reference interval, 138 to 160 mmol/L), and hyperkalemia (6.5 mmol/L; reference interval, 3.7 to 5.8 mmol/L); these results were attributed to postrenal azotemia. Results of urinalysis indicated proteinuria, severe hematuria, a urine pH of 6.5, and a urine specific gravity of 1.028. Results of bacteriologic culture of a urine sample, which were available several days later, indicated considerable growth (> 100,000 colony-forming units/mL) of coagulase-positive Staphylococcus pseudintermedius. A urinary catheter was placed by the referring veterinarian without difficulty, and 400 mL of severely hemorrhagic urine was drained from the bladder of the dog. The patient was hospitalized during that day and received fluids, ampicillin trihydrate (8.8 mg/kg [4.0 mg/lb], SC), and medications for management of pain. The dog was discharged, and cefadroxil (8.8 mg/kg, PO, q 12 h) was prescribed. The dog was referred to VCA Animal Specialty Group the following morning because of continued lethargy and stranguria.

Results of evaluation at the VCA Animal Specialty Group indicated the dog was dehydrated and had pollakiuria, stranguria, hematuria, and a small, thickened urinary bladder; signs of pain were elicited during palpation of the urinary bladder; signs of pain were elicited during palpation of the urinary bladder.

Successful treatment of encrusted cystitis associated with *Staphylococcus pseudintermedius* infection in the urinary bladder of a dog

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**Case Description**—A 5-year-old female spayed mixed-breed dog was examined because of signs of persistent stranguria following treatment for urethral obstruction.

**Clinical Findings**—Radiographic, ultrasonographic, cystoscopic, and histologic findings were consistent with encrusted cystitis. Results of bacteriologic culture of urine and bladder wall biopsy samples indicated growth of *Staphylococcus pseudintermedius*.

**Treatment and Outcome**—The dog was initially treated via IV administration of fluids, placement of an indwelling urinary catheter, lavage of the bladder with sterile saline (0.9% NaCl) solution, and administration of antimicrobial drugs and betahanechol (to improve voiding of urine from the bladder). Antimicrobial drugs were administered for 3 months, and a commercially available diet for dissolution of urinary calculi was fed. Clinical signs of encrusted cystitis gradually resolved during the 3 months after the initial examination. Results of urinalysis and abdominal ultrasonographic examination performed 4 months after the initial examination indicated resolution of the disease.

**Clinical Relevance**—Encrusted cystitis is extremely rare in small animals and has previously only been associated with *Corynebacterium* spp infection of the urinary bladder. Resolution of encrusted cystitis has previously been achieved via surgical debridement of the bladder and treatment with antimicrobial drugs. The clinical findings and successful resolution of clinical signs in the dog of the present report suggested that urease-positive bacteria other than *Corynebacterium* spp can cause encrusted cystitis and that feeding of a diet for dissolution of urinary calculi in conjunction with antimicrobial treatment may result in resolution of urinary bladder lesions and clinical signs attributable to the disease without the need for surgical debridement of encrusted plaques. (*J Am Vet Med Assoc* 2013;242:798–802)
urinary bladder. Results of biochemical analysis of a venous blood sample indicated creatinine, BUN, sodium, and potassium concentrations were within the reference intervals. Results of this venous blood analysis also indicated polycythemia (Hct, 67%; reference interval, 37% to 55%), a pH of 7.16 (reference interval, 7.35 to 7.45), high chloride concentration (113 mmol/L; reference interval, 98 to 109 mmol/L), high P<sub>CO</sub><sub>2</sub> (48.1 mm Hg; reference interval, 35 to 45 mm Hg), low bicarbonate concentration (17.1 mmol/L; reference interval, 18 to 23 mmol/L), and a base excess of −12 mmol/L (reference interval, −2 to 3 mmol/L). These results were interpreted as mixed (metabolic and respiratory) acidosis. Prothrombin time and partial thromboplastin time were within the reference intervals. Abdominal ultrasonography revealed a mildly distended urinary bladder with an eccentrically thickened bladder wall (greatest thickness [8.4 mm] detected at the apex). The mucosal surface of the urinary bladder was hyperechoic and irregular in shape with diffuse acoustic shadowing of the deep aspect consistent with mural mineralization (Figure 2). The ultrasonographic appearance of the trigone region of the bladder was unremarkable. A small amount of peritoneal fluid was detected surrounding the urinary bladder. The ultrasonographic appearance of the urethra was unremarkable.

A Foley catheter was placed, and the urinary bladder was lavaged multiple times with warm sterile saline (0.9% NaCl) solution (total volume, 600 mL). A closed drainage system was then attached to the Foley catheter. The dog received buprenorphine (0.01 mg/kg [0.0045 mg/lb], IV), lactated Ringer’s solution (4.5 mL/kg/h [2.0 mL/lb/h], IV), ampicillin (22 mg/kg [10 mg/lb], IV, q 8 h), and famotidine (0.43 mg/kg [0.20 mg/lb], IV, q 12 h). The following morning, the dog was eating well and treatments were changed to amoxicillin–clavulanate acid (11 mg/kg [5 mg/lb], PO, q 12 h) and famotidine (0.44 mg/kg [0.20 mg/lb], PO, q 12 h). That morning, the bladder was lavaged with 250 mL of warm sterile saline solution. Hemorrhagic clots and gritty material were detected in the lavage fluid. On day 3 of hospitalization, the rate of IV administration of lactated Ringer’s solution was decreased (2 mL/kg/h [0.9 mL/lb/h]) and the urinary catheter was removed. The dog began to strain during urination and was only able to pass a small amount of urine. Cystoscopy was performed later that same day.

Cystoscopy revealed a large amount of severely hemorrhagic urine and debris in the bladder. The bladder mucosa was diffusely inflamed with areas of erosion and ulceration, several polyloid masses, and numerous white, firm plaques. Multiple biopsy samples of the lesions were submitted for histologic evaluation and bacteriologic culture. Histopathologic findings included suppurative inflammation with substantial cocoid bacterial colonization and multifocal mineralization. Results of bacteriologic culture of urinary bladder biopsy samples indicated growth of <i>S</i> <i>pseudintermedius</i>; moderate growth of <i>E</i> <i>coli</i> was also detected. On the basis of results of radiography, ultrasonography, cystoscopy, and histologic examination of urinary bladder biopsy samples, a diagnosis of encrusted cystitis was made.

The dog was discharged with a urinary catheter and closed collection system in place, and amoxicillin–
clavulanic acid (11 mg/kg, PO, q 12 h), enrofloxacin (6.0 mg/kg [2.7 mg/lb], PO, q 24 h), famotidine (0.44 mg/kg, PO, q 24 h), and bethanechol (1.1 mg/kg [0.5 mg/lb], PO, q 8 h; to improve voiding of urine from the bladder) were prescribed. After 3 days at home, the urinary catheter was removed and the dog was able to fully empty its urinary bladder, although the bladder remained firm and extremely thick after voiding of urine. Feeding of a commercially available diet for dissolution of urinary calculi was started, and administration of amoxicillin–clavulanic acid, bethanechol, and famotidine was continued for an additional 5 days. The dog was frequently brought by its owner to the clinic for recheck examinations during the 4 months following the initial evaluation. The dog continued to have incontinence and hematuria for the first 3 months following the initial evaluation. Results of abdominal ultrasonography performed during each visit indicated gradual improvement of the appearance of the bladder wall with a progressively decreasing amount of mineralization. Results of urinalyses performed 3 and 4 months after the initial evaluation indicated gradual resolution of hematuria. Enrofloxacin administration was discontinued 3 months after the initial evaluation; results of urinalysis performed at that time indicated no evidence of bacteriuria or pyuria. At 4 months after the initial evaluation, the dog was completely continent, had no hematuria, had unremarkable abdominal palpation findings, and had an ultrasonographically normal urinary bladder.

Discussion

Encrusted cystitis is an uncommon cause of urinary bladder wall mineralization that is associated with infection by urease-producing bacteria, resulting in chronic, ulcerative inflammation of the urinary bladder with deposition of inorganic salts onto the damaged mucosal surface.1–11 Encrusted cystitis is extremely rare in humans and other animals; this disorder has been reported for 1 cat,9 1 horse,6 and a small number of dogs.7,8 The prevailing theory regarding the pathophysiology of encrusted cystitis is that urease-producing bacteria produce ammonia, which alkalizes the urine and increases the solubility of minerals, causing them to precipitate and become adhered to the inflamed and damaged bladder mucosal surface and resulting in dystrophic mineralization.8,10–12 Other theories for the pathophysiologic disorder include involvement of a nanoparticle that causes calcification of tissues and upregulation of expression of osteogenic proteins following tissue injury.11,12 The composition of encrusted urinary bladder plaques has been determined for only 1 dog with encrusted cystitis; those plaques consisted primarily of struvite, with a small amount of calcium phosphate (apatite).1 Struvite and apatite are also the primary components of encrusted urinary bladder plaques in humans.5,9,5,5,5,5

In all cases8,6 of encrusted cystitis reported in the veterinary medical literature, the disorder has developed secondary to Corynebacterium spp infection. Although Corynebacterium spp also cause most cases of encrusted cystitis in humans, >40 species of urease-positive bacteria have been implicated in development of the disorder since the condition was first described in the early 20th century.4 Rarely, Proteus mirabilis and Pseudomonas aeruginosa have been implicated in the disease in humans.13,14 For the dog of the present report, results of bacteriologic culture of urine and urinary bladder biopsy samples indicated growth of coagulase-positive S pseudintermedius. Staphylococcus pseudintermedius are urease-positive bacteria commonly implicated in pyoderma and urinary tract infections in dogs.15–20 An estimated 37.2% of healthy dogs and 87.5% of atopic dogs are carriers of such bacteria; in dogs, S pseudintermedius is most commonly isolated from the perineum.21 To the authors’ knowledge, S pseudintermedius has not previously been implicated in encrusted cystitis. However, Staphylococcus spp have been implicated in encrusted cystitis in humans and urolithiasis in dogs.22,23 In addition, multiple urease-positive Staphylococcus spp are capable of causing mineral deposition and encrustation of biofilms on indwelling urinary catheters.24 Moderate growth of E coli was also detected in bacterial cultures of bladder wall biopsy samples obtained from the dog of the present report. Because E coli was not originally cultured from the urine sample obtained 4 days prior to the biopsy samples, iatrogenic introduction of these bacteria via performance of multiple urinary catheterizations was suspected.

Clinical characteristics of humans with encrusted cystitis include dysuria, macroscopically detectable hematuria, pain, and, occasionally, elimination of small calculi via the urethra.10,25 Obstruction of the urinary tract caused by mineralized debris or small calculi has also been detected in humans with that disorder4,9; such factors may have contributed to urinary tract obstruction in the dog of the present report. Because E coli was not originally cultured from the urine sample obtained 4 days prior to the biopsy samples, iatrogenic introduction of these bacteria via performance of multiple urinary catheterizations was suspected.

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and the cat was euthanized when it became moribund. A diagnosis of encrusted cystitis is confirmed via direct observation of inflamed, ulcerated bladder wall tissue with white plaques; histologic findings of bladder inflammation, necrosis, and mineralization; and detection of bladder infection with urease-producing bacteria.

Treatment of animals with encrusted cystitis requires a multimodal approach. Humans with this disease are frequently treated via administration of antimicrobial drugs, excision of encrusted plaques, and acidification of urine via oral administration of acidifying agents (e.g., acetoxycholic acid [an urease inhibitor]) or irrigation of the bladder with acidic solutions (e.g., citric acid, Suby, or Thomas solutions).

Direct removal of plaques, which harbor the bacteria that cause the disorder, is typically necessary for resolution and may be performed endoscopically or via a traditional surgical approach. Several approaches have been used for removal of urinary bladder plaques in animals to achieve resolution of clinical signs. Resolution of clinical signs was achieved via surgical debridement and administration of antimicrobial drugs for 2 of 3 dogs of another report that had encrusted cystitis. Another dog treated via acidification of urine (via oral administration of methionine) and administration of antimicrobial drugs had resolution of clinical signs, but the bladder remained rigid with decreased capacity. Follow-up imaging was not performed for that dog; therefore, resolution of lesions could not be determined. A horse that was treated with antimicrobial drugs and daily urinary bladder infusions of a warm dimethyl sulfoxide solution had clinical and cystoscopic resolution of cystitis. Clinical signs of encrusted cystitis transiently improved in a cat that was treated with fluids, medication for management of pain, and antimicrobial drugs; however, clinical signs rapidly recurred, and the cat was euthanized when it became moribund following discharge from the hospital.

The dog of the present report was initially treated via urinary tract catheterization, lavage of the bladder with warm sterile saline solution, and administration of fluids and antimicrobial drugs. Following determination of a diagnosis of encrusted cystitis, the dog was treated with antimicrobial drugs for 3 months and a diet for dissolution of urinary calculi was fed. Although oral administration of methionine for acidification of urine has been described for 1 dog, the dog of the present report was the first dog for which a urinary calculus dissolution diet was used for treatment of encrusted cystitis, to the authors’ knowledge. Dietary treatment was used for this dog because such diets can cause dissolution of struvite calculi, which are typically the primary component of encrusted urinary bladder plaques. The clinical outcome for this dog suggested that a urinary calculus dissolution diet can aid dissolution of encrusted bladder plaques, because follow-up ultrasonography revealed resolution of bladder mucosal mineralization without performance of surgical debridement.

Encrusted cystitis is a rare cause of mineralization of the urinary bladder mucosa in dogs that is most frequently caused by Corynebacterium spp infection and is typically treated via bacteriologic culture–guided antimicrobial treatment, surgical debridement of plaques, and, occasionally, acidification of urine. The clinical outcome for the dog of the present report suggested that S. pseudintermedius, a common cause of cystitis in dogs, can also cause encrusted cystitis. The success of feeding of a diet for dissolution of urinary calculi in this dog suggested that such a diet can aid dissolution of mineralized mucosal plaques in the urinary bladder without performance of invasive surgical debridement, which has typically been used for treatment of this disease in other animals.

References


