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Diagnosis, management, and outcome of urethral obstruction secondary to the capsule associated with the artificial urethral sphincter device

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Abstract

Background: Urethral obstruction secondary to artificial urethral sphincter (AUS) implantation is a recognized complication in dogs. However, urethral obstruction secondary to AUS-associated capsule formation has been described rarely.

Hypothesis: Describe clinical and diagnostic findings, management, and outcome in 6 dogs with urethral obstruction secondary to AUS-associated capsule formation.

Animals: Six client-owned dogs.

Methods: Retrospective study. Medical records between January 1, 2010, and June 30, 2021, were reviewed to identify dogs with urethral obstruction associated with the AUS device.

Results: The AUS device was implanted a median of 884 days (range, 20-2457 days) before presentation for urethral obstruction. Median age at time of urethral obstruction was 4.7 years (range, 3.1-8.7 years). Clinical signs at the time of urethral obstruction were stranguria (n = 4), pollakiuria (3), weak urine stream (2), and worsened urinary incontinence (1). In all dogs, the urethra was noted to be stenotic during urethroscopy and positive contrast cystourethrography. All dogs underwent surgery, and a fibrous capsule associated with the AUS was found to be causing urethral stenosis. Resolution of urethral obstruction occurred in all dogs after transection or removal of the capsule. Positive bacterial cultures were obtained from the capsule, AUS, or both in all dogs. Recurrence of urethral obstruction had not occurred in any dog at the time of follow-up.

Conclusions and Clinical Importance: Urethral obstruction secondary to capsule formation is an uncommon but clinically important complication associated with use of the AUS. Continued investigation is needed to evaluate this complication more thoroughly, and its possible association with infection.

KEYWORDS
incontinence, urethra, urinary surgery, urinary tract obstruction

Abbreviations: AUS, artificial urethral sphincter; HCT, hematocrit; RR, reference range; UCD-VMTH, University of California, Davis William R. Pritchard Veterinary Medical Teaching Hospital; UTI, urinary tract infection.

1 | INTRODUCTION

Artificial urethral sphincter (AUS) devices generally are placed in dogs for urinary incontinence that have failed to respond to medical or surgical treatment or both. Most dogs have improvement in their urinary incontinence after AUS placement,[1-5] with functional urinary continence reported in 67% of dogs in 1 study; the rate of continence increased to 92% when owners were compliant in following up with cuff inflations.[1] Minor complications after AUS placement including lower urinary tract signs and seroma formation at the SC port of the AUS occur in 37% to 82% of dogs; major complications such as urethral obstruction are less common (7%-18% of dogs).[1,3,6] Dogs with AUS devices have developed urethral obstruction unrelated to AUS cuff inflation.[1,3,6] In these instances, urethral obstruction occurred because of an extraluminal urethral stricture requiring urethral stent placement,[1] intraluminal urethral web formation,[1] and a band of tissue surrounding the urethra at the site of the AUS cuff.[3,6] In 1 case of urethral obstruction, in which adhesions developed between the small intestine and tubing, the cause of urethral obstruction was not described.[3] In 2 of the 3 dogs in which a band of tissue was identified surrounding the urethra at the level of the AUS cuff, it was unclear whether this tissue was the cause of urethral obstruction because urinary catheters were placed without resistance and the urinary bladder could be expressed manually with ease.[3]

Development of urethral obstruction secondary to AUS-associated capsule formation has been poorly characterized in dogs. We aimed to describe the clinical presentation, diagnostic findings, management, and outcome in 6 dogs that presented to the University of California, Davis William R. Pritchard Veterinary Medical Teaching Hospital (UCD-VMTH) for urethral obstruction secondary to AUS-associated fibrous capsule formation.

2 | MATERIALS AND METHODS

Medical records from the UCD-VMTH between January 1, 2010, and June 30, 2021, were retrospectively reviewed to identify dogs that underwent removal of the AUS device because of urethral obstruction secondary to urethral stenosis associated with AUS fibrous capsule formation. Information extracted from the medical record included signalment, body weight, clinical signs and their duration at the time of urethral obstruction, and physical examination and laboratory findings at the time of urethral obstruction. Additionally, results of abdominal ultrasound examination and cystourethroscopy were recorded. The dog's history of urinary incontinence, including diagnostic evaluation, and history of positive urine culture samples also were reviewed. It was not possible to determine in all cases whether a positive urine culture was associated with clinical signs (ie, urinary tract infection [UTI]) on medical record review. Variables related to AUS placement, including timing relative to development of urethral obstruction and whether or not cystopexy was performed, also were recorded.

Diagnostic evaluations of urethral obstruction included abdominal ultrasound examination and either cystourethroscopy or retrograde positive contrast cystourethrography or both. Abdominal ultrasound examinations were performed by a board-certified radiologist or by a resident under the supervision of a board-certified radiologist. Cystourethroscopy was performed with the dog in right lateral or dorsal recumbency using an appropriate rigid cystoscope determined by dog size and urethral diameter (9.5 French 14 cm rigid 30° cystoscope, Storz BA-67030, Karl Storz SE & Co. KG, Tuttlingen, Germany or 14 Fr 18 cm rigid 30° cystoscope, Storz Hopkins II, Karl Storz SE & Co. KG). Retrograde positive contrast cystourethrography was performed by C-arm fluoroscopy using iodinated non-ionic contrast medium.

For exploratory laparotomy, the dog was placed in dorsal recumbency. If cystourethroscopy was repeated intraoperatively, postoperatively, or both, the abdomen was draped to include the vulva or prepuce. A standard caudal midline laparotomy was performed for removal of the AUS. The capsule surrounding the AUS device was dissected to expose the implant. The AUS cuff was removed by cutting the eyelet suture, and the AUS tubing and SC port were removed. A sample from the capsule surrounding the AUS device and urethra or from fluid associated with the capsule was collected for aerobic and anaerobic bacterial culture.

Descriptive statistics were performed using Microsoft Excel (2018). Data were summarized using median and range.

3 | RESULTS

3.1 | Signalment

Between January 1, 2010, and June 30, 2021, 6 dogs were diagnosed with urethral stenosis secondary to AUS-associated capsule formation at the UCD-VMTH. Five of the 6 dogs had their AUS device placed at the UCD-VMTH (with 40 dogs undergoing AUS placement in the same time period at the same hospital). Median age at presentation for urethral obstruction was 6 years (range, 3-9 years). Breeds represented were Labrador retriever (dog 1), Chihuahua cross (dog 2), boxer (dog 3), Golden retriever (dog 4), Jack Russell terrier (dog 5), and Yorkshire terrier cross (dog 6). All were spayed females except dog 6, which was a castrated male. Median weight was 13.8 kg (range, 5.3-31.0 kg).

3.2 | History before urethral obstruction

Of 6 dogs, 5 had congenital anatomical abnormalities of the urogenital system. Intramural ectopic ureters had been diagnosed and treated in 2 of 6 dogs by cystoscopic-guided laser ablation before AUS placement; dog 1 had a left intramural ectopic ureter opening in the mid-urethra and dog 2 had bilateral intramural ectopic ureters opening in the distal urethra. Both dogs had a vestibulovaginal septal remnant (which was not treated in either dog) and dog 1 had a subjectively short urethra diagnosed by retrograde cystoscopy and contrast cystourethrography performed with fluoroscopic guidance. Dog 4 had

a right intramural ectopic ureter opening in the proximal urethra that was diagnosed at the time of evaluation of AUS-associated urethral obstruction. Dog 5 had multiple congenital abnormalities of the urogenital tract: left renal agenesis, underdeveloped left uterine horn, vaginal hypoplasia, vestibulovaginal septal remnant, and poorly defined vestibule and urethra; these abnormalities were diagnosed based on a combination of abdominal ultrasound examination, retrograde cystourethroscopy, and fluoroscopic-guided retrograde contrast cystourethrography. Dog 6 had a history of a urethrorectal fistula diagnosed at 2 years of age using abdominal computed tomography (CT) scan and fluoroscopic-guided retrograde contrast cystourethrography during initial investigation of urinary incontinence and recurrent UTI. This dog also had marked dilatation of the prostatic, membranous, and proximal penile urethra identified on retrograde positive contrast urethrography. A cystopexy was performed at the time of surgical repair of the urethrorectal fistula. The dog had persistent urinary incontinence 4 months after these procedures and the AUS was placed. Dog 3 did not have any abnormalities on cystourethroscopy and retrograde positive contrast cystourethrography approximately 2 months before AUS placement and was assessed as having urinary incontinence because of urethral sphincter mechanism incompetence; urethral pressure profilometry was not performed.

The AUS device (Norfolk Vet Products, Inc, Skokie, Illinois) was placed a median of 1215 days (range, 20-2457 days) before presentation for urethral obstruction. It was placed because of urinary incontinence refractory to medical management with estriol, phenylpropanolamine, or both. In all dogs that had cystourethroscopy, the AUS device was not placed during the same anesthetic event to avoid urethral trauma that might result in urethral stricture formation.[1] Recorded AUS cuff sizes were 16 × 14 mm (n = 1), 12 × 14 mm (2), 10 × 14 mm (1), and 8 × 14 mm (2). No complications were recorded during AUS placement in any dog. Cystopexy had been performed 1 month (dog 4), 2 months (dog 3), and 4 months (dog 6) before AUS placement in 3 of 6 dogs; the cystopexy site evaluated at AUS placement was appropriate in all 3 dogs. Cystopexy was performed at the same time as AUS placement in 1 dog (dog 1). Dog 5 was receiving amoxicillin clavulanate at the time of AUS placement for treatment of a UTI (*Escherichia coli*) and this medication was continued post-operatively; 4 dogs were not treated with any antibiotics after AUS placement. Antibiotic use post-operatively was unknown in 1 dog (dog 4) because medical records were not available for review.

Positive urine cultures were documented before AUS placement in 4 of 6 dogs and after AUS placement in 4 of 6 dogs, with 3 of these dogs having positive cultures both before and after AUS placement. Three dogs had >1 positive urine culture after AUS placement. One dog (dog 3) did not have any positive urine cultures before or after AUS placement. No dog had a positive urine culture at the time of AUS placement but only 4 of 6 dogs had urine culture performed in the 14 days before AUS placement. The remaining 2 dogs did not have any clinical signs of UTI at the time of AUS placement.

Swelling of the AUS port site developed 952 days after AUS placement in dog 6 (and 707 days before the AUS was removed because of urethral obstruction). Cytology evaluation of a fine needle aspirate from the site showed bacterial cocci, and *Staphylococcus* spp. was cultured from this sample. The dog was treated with amoxicillin-clavulanate (20.8 mg/kg PO q12h) for 7 days based on sensitivity testing and the infection resolved.

One dog (dog 1) had been diagnosed with International Renal Interest Society (IRIS) stage 2 chronic kidney disease on the basis of persistently increased serum creatinine concentration after AUS placement.[7] This dog had a history of recurrent positive urine cultures after AUS placement.

3.3 | Clinical features at time of urethral obstruction

Clinical signs at the time urethral obstruction secondary to AUS was diagnosed were stranguria (n = 5), pollakiuria (3), weak urine stream (2) and progressive urinary incontinence with a large bladder (suspected overflow incontinence) (2). The median duration of clinical signs before presentation was 22 days (range, 2-294 days). Physical examination findings included a moderate- to large-sized urinary bladder (4), recessed vulva (2), urinary incontinence (2) and abdominal discomfort (1). Rectal examination was performed in 5 of 6 dogs and was normal in 4 of these dogs. In 1 dog, the AUS cuff could be palpated and no pain was associated with palpation. One dog's urinary bladder could be partially emptied by manual expression, but this dog was unable to void voluntarily. Two other dogs were observed attempting to void and had stranguria with either no urine passage or with only drops of urine passed. One dog (dog 3) had a urinary catheter placed at presentation for urethral obstruction; a urinary catheter was not placed in the remaining dogs.

The AUS cuff contained no saline in 3 of 6 dogs at the time of presentation for urethral obstruction. In 1 dog, the AUS cuff had never been filled with saline (dog 3). In 3 other dogs, saline had been removed in an attempt to empty the AUS (when stranguria first developed) 3 months (dog 6), 9 months (dog 4) and 15 months (dog 1) before presentation for urethral obstruction. In these 3 dogs, urination did not normalize after removal of the fluid from the AUS port using a Huber needle. In the remaining 2 dogs, saline from the AUS cuff was removed at the time of presentation for urethral obstruction using the AUS port, but in 1 dog, only 0.46 mL could be retrieved of previously reported instilled volume of 1.05 mL despite no kinking of the AUS tubing. Both dogs had no change in their clinical signs after removal of saline from the AUS cuff.

3.4 | Diagnostic testing

The median hematocrit (HCT) was 43.8% (range, 25.7-51.5%; reference range [RR], 40-55%), median leukocyte count 9750/μL (range, 5560-13 080/μL; RR, 6000-13 000/μL) and median neutrophil count 6365/μL (range, 3780-10 595/μL; RR, 3000-10 500/μL). Dog 1 had normocytic hypochromic regenerative anemia (HCT, 25.7%; MCV, 69.5 fl; RR, 65-75 fl; MCHC, 32.7 g/dL; RR, 33-36 g/dL; reticulocytes, 167 300/μL; RR, 7000-65 000/μL). Anemia was suspected to be a consequence of gastrointestinal hemorrhage; no evidence of urinary tract hemorrhage was found based on urine sediment examination

and no hemolysis was detected based on normal red blood cell morphology, normal serum bilirubin concentration and negative direct Coomb's test. The dog had low normal vitamin B12 concentration (331 ng/L; RR, 271-875 ng/L) and on abdominal ultrasound examination, mildly enlarged mesenteric and medial iliac lymph nodes, but no changes in the gastrointestinal tract were noted. The anemia in this dog responded after change to a hydrolyzed protein diet, treatment with an acid suppressant (omeprazole) and vitamin B12 administration. Median serum creatinine and BUN concentrations were 1.3 mg/dL (range, 0.6-2.1 mg/dL; RR, 0.8-1.5 mg/dL) and 25 mg/dL (range, 10-47 mg/dL; RR, 11-33 mg/dL), respectively. Median urine specific gravity for 4 dogs was 1.020 (range, 1.010-1.040). No bacteriuria, pyuria or hematuria were observed on urine sediment examination in any dog at the time of urethral obstruction. Bacterial urine culture was performed in 5 of 6 dogs at the time of urethral obstruction and was negative for bacterial growth in 5 of 5 dogs in which it was performed, but 1 dog (dog 1) was receiving nitrofurantoin for treatment of *E. coli* UTI at the time that negative urine culture was obtained.

Abdominal ultrasound examination was performed in all dogs. In dog 1, an abdominal ultrasound examination disclosed moderate thickening of the urinary bladder. The left ureter was diffusely thickened with mild dilatation distally. Mild thickening and dilatation of the right ureter were observed. The left kidney was decreased in size to 3.2 cm in length (normal kidney length for 30-34 kg dogs, 7.2 cm)[8] with decreased corticomedullary distinction. The right kidney had mild pyelectasia. The medial iliac lymph nodes were enlarged and hypoechoic. The AUS device (including tubing) was identified; no abnormalities were detected. In dog 2, marked bilateral pyelectasia (left renal pelvis, 1.56 × 1.15 × 1.26 cm and right renal pelvis 1.60 × 1.10 × 1.10 cm) was observed with blunting of the renal papillae. Both ureters were diffusely dilated to 0.8 cm. The urinary bladder was large. The AUS cuff was identified and no abnormalities were observed. In dog 3, on abdominal ultrasound examination, hyperechoic, lobular tissue was identified ventral to the urinary bladder neck and surrounding the AUS tubing. The urinary bladder wall appeared thickened because of its small size with a urinary catheter in place. In dog 4, the urinary bladder was large. Diffuse right ureteral dilatation to 12 mm and renal pelvic dilatation (10 mm) were observed. The left ureter was normal and the left renal pelvis minimally dilated. The right medial iliac lymph node was mildly enlarged (10 mm). The AUS cuff was identified in the region of the trigone, with no abnormalities. In dog 5, abdominal ultrasound examination showed absence of the left kidney. The AUS tubing was kinked. In dog 6, dilatation of the urethra and mild thickening were observed. The AUS cuff was in place.

Abdominal radiographs were performed in 3 of 6 dogs. In dog 5, abdominal radiographs confirmed kinking of the AUS tubing that was recognized on abdominal ultrasound examination. The AUS device was appropriately in place in the remaining 2 dogs (dog 3 and dog 6), with no other abnormalities detected.

Cystourethroscopy was performed in 4 of 6 dogs. In these 4 dogs, marked narrowing of the urethra was observed at the proximal to mid-urethra (Figure 1). In dog 1, the cystoscope could not be passed through the area of stenosis. In dog 2, mild erythema of the urethral mucosa was observed at the level of the urethral narrowing, and both ureteral orifices in the urinary bladder trigone were dilated. In dog 4, the right ureteral orifice was ectopic, approximately 15 mm distal to the trigone and approximately 3 mm cranial to the urethral obstruction; the left ureteral orifice was located within the trigone. In this dog, balloon dilatation of the area of urethral narrowing to 10 mm was attempted (balloon size and type not available); this diameter was selected based on the apparent diameter at cystoscopy. Despite multiple attempts at balloon dilatation using appropriate size and filling of the balloon, the stenotic region was not noted to change. Retrograde positive contrast cystourethrography was performed in 6 of 6 dogs and showed marked narrowing of the contrast column at the level of the AUS cuff in all dogs (Figure 2). Vesicoureteral reflux and diffuse dilatation of both ureters were observed in dog 2. Dog 6 also had marked dilatation of the distal prostatic urethra, which had been recognized before AUS placement.

All dogs underwent standard caudal midline laparotomy. In dog 3, minimal purulent discharge was noted at the ventral abdominal midline incision site. Multiple adhesions were identified between the urinary bladder and body wall in this dog; it had undergone cystopexy

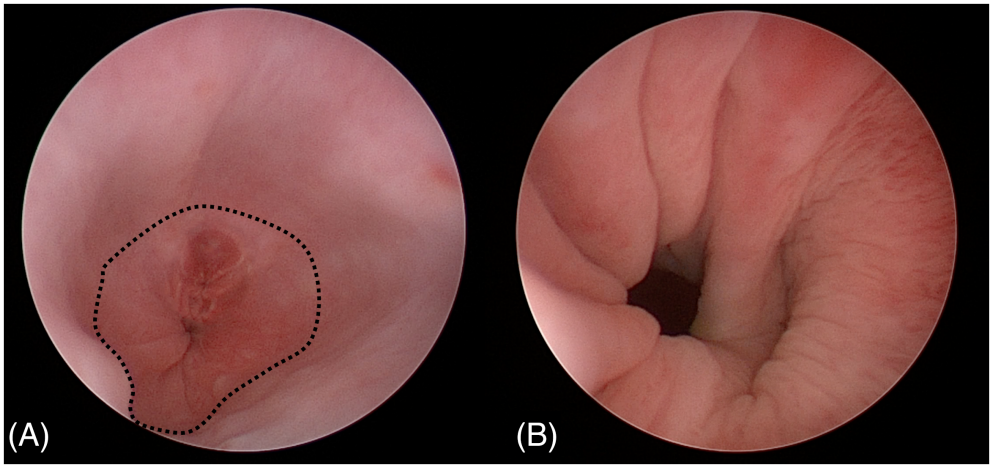

FIGURE 1 Urethroscopic images of a 5-year-old male castrated terrier mixed breed dog before (A) and after (B) removal of an AUS device. (A) The opening to the bladder is not able to be visualized, and a region of obvious urethral constriction is seen (dashed line). (B) After removal, the cystourethral junction is clearly visible and urine is able to pass freely from the bladder into the urethra

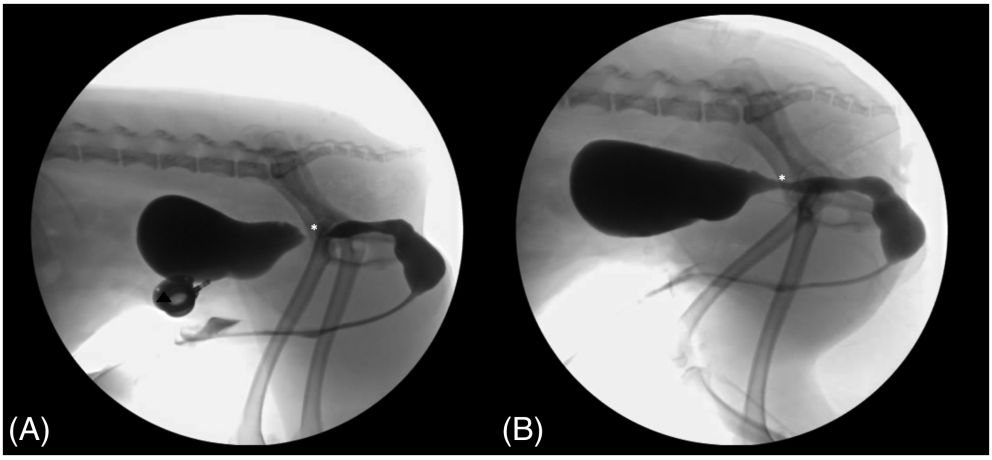

FIGURE 2 Lateral fluoroscopic images of the caudal abdomen of a 5-year-old male castrated terrier mixed breed dog during contrast cystourethrogram. (A) A focal region of loss of the contrast medium column because of constriction can be noted in the region of the artificial urethral sphincter (AUS) (asterisk). The injection port (black triangle) can still be seen sitting SC before removal. (B) After removal of the AUS, a contrast cystourethrogram was repeated, and opening of the urethra in the region of where the AUS had been (asterisk) is now noted

20 days before the time of AUS placement. In all dogs, the AUS device was encapsulated and did not contain any fluid. After dissection of the AUS device from the surrounding capsule and removal, a firm capsule remained, encircling and constricting the urethra (Figure 3). The capsule prevented manual expression of the urinary bladder in all dogs at the time of surgery. In 5 of 6 dogs, the capsule was transected and removed after being dissected away from the urethra. The capsule surrounding the urethra was transected to release constriction of the urethra but not removed in 1 dog (dog 2) because of its integration with the urethral tissues and therefore concern for excessive disruption of the urethra and surrounding tissues. In 2 dogs, a small amount of red-brown fluid was found between the AUS cuff and capsule and, in 1 of these dogs, similar fluid was found within the AUS tubing. In these dogs, samples of the fluid were collected for aerobic and anaerobic bacterial culture using sterile swabs in addition to sampling the capsule for culture. In the remaining 4 dogs, samples for culture were collected from the capsule and AUS device.

In all dogs, after transection of the capsule, with or without removal of the bulk of the capsule constricting the urethra, the urinary bladder could be expressed easily and a normal urine stream was observed. In 1 dog (dog 4) after transection of the capsule, cystotomy was performed to pass a 14 French red rubber catheter normograde into the distal urethra; no resistance to its passage was detected. Digital palpation also confirmed patency of the proximal urethra in this dog.

Retrograde cystourethroscopy (n = 1), cystourethroscopy combined with retrograde positive contrast cystourethrography (3), or retrograde positive contrast cystourethrogram alone (1) was repeated after capsule removal. These studies showed resolution of the urethral narrowing, with normal urethral contrast column on cystourethrography (Figures 1 and 2). A urinary catheter was not placed after surgical intervention in any dog.

Aerobic and anaerobic bacterial culture yielded aerobic bacterial growth in all dogs; no anaerobes were cultured (Table 1). Cultures included moderate growth (*Staphylococcus pseudintermedius*; n = 1),

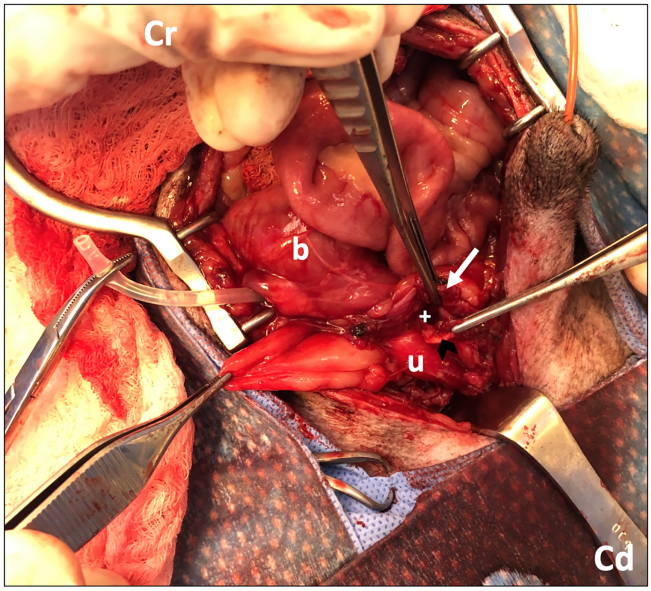

FIGURE 3 Intraoperative images of a 5-year-old male castrated terrier mixed breed dog during removal of an AUS device (Cr = cranial, Cd = caudal). The bladder (b) and urethra (u) can be seen in the caudal abdomen. There are 2 layers of fibrous tissue: an inner fibrous tissue band (black arrowhead) located inside the AUS (immediately adjacent to the urethra) and an outer fibrous tissue band (white arrow) located externally to and encasing the AUS. The AUS has been removed, but the location of the AUS is marked by the white plus sign. Before removal, both the inner and outer fibrous tissue bands completely surrounded the entire urethral circumference

mild growth (*Staphylococcus pseudintermedius* and *E. coli*; n = 3), and growth in enrichment broth (coagulase-negative *Staphylococcus* spp.; n = 2). Although culture from dog 1 grew only very small numbers of *E. coli*, cytology of the fluid showed small numbers of Gram-positive rods and small numbers of neutrophils. Cytology from the bacterial culture sample in dogs 3 and 6 identified Gram-positive cocci. No

TABLE 1 Bacteria cultured from AUS device and associated capsule, fluid associated with the AUS device and capsule or both

Bacteria	Number of dogs
Staphylococcus pseudintermedius	3
Coagulase-negative *Staphylococcus* spp.	2
Escherichia coli	1

organisms were observed on direct smear from the samples collected in other dogs.

Comparing bacterial cultures from the capsule, AUS device, and any associated fluid collected at the time of AUS removal with bacteria detected in urine cultures performed any time after AUS implantation, the same bacteria type was identified on at least 1 occasion in 3 of 6 dogs. When evaluating bacteria cultured from the urine of dogs before AUS placement, the same bacteria were not cultured from intraoperative samples.

Histopathology of the capsule surrounding the AUS device and constricting the urethra was performed in 2 dogs. In dog 5, cell-poor, collagenous fibrous connective tissue was observed, characterized by segmentally underlying loose connective tissue or mature adipose tissue, consistent with mature fibrous tissue. No active inflammation was present. In dog 6, severe fibrosis with moderate multifocal histiocytic, neutrophilic, and lymphoplasmacytic inflammation was observed.

3.5 | Outcomes

Dogs were hospitalized for a median of 2 days postoperatively (range, 1-2 days). All dogs could void completely with normal urine stream and no evidence of urethral obstruction immediately postoperatively. Of 6 dogs, 3 had mild stranguria before hospital discharge. In dog 2, repeat serum biochemistry the day after surgery disclosed improved serum creatinine (0.9 mg/dL, previously 2 mg/dL at admission) and BUN (15 mg/dL, previously 47 mg/dL at admission) concentrations; initial azotemia was suspected to be post-renal and associated with urethral obstruction. Abdominal ultrasound examination in this dog 2 days postoperatively also showed improved bilateral pyelectasia (left renal pelvis, 1.0 × 0.5 × 0.7 cm and right renal pelvis 0.9 × 0.5 × 0.6 cm). The ureters were dilated proximally to 0.5 cm but tapered appropriately; the ureteral walls were mildly thickened. The urinary bladder was small. Small volumes of free peritoneal fluid and gas were present, and considered secondary to recent surgery.

Of 6 dogs, 2 were treated for 14 days with antimicrobials based on culture susceptibility results of the AUS device and associated capsule or fluid obtained at surgery. Because of the extent of inflammation noted at surgery, prednisone 0.5 mg/kg PO q24h was administered to dog 3 for 2 days in addition to antimicrobials.

Median follow-up after hospital discharge was 184 days (range, 3-2313 days); 1 dog was lost to follow-up after 3 days. No evidence of recurrent urethral obstruction was detected in any dog. Cystoscopic-guided laser ablation of the right intramural ectopic ureter identified on evaluation for urethral obstruction in dog 4 was performed without complications approximately 10 weeks after AUS removal. The urethra appeared normal during cystourethroscopic evaluation at that time. In 4 dogs with >30 days of follow-up after AUS removal, urinary incontinence worsened in 3, and 2 of these dogs had urinary incontinence adequately controlled by phenylpropanolamine, estriol, or both drugs concurrently. One dog had a partial response to estriol. One dog had static urinary incontinence. No dog had the AUS device replaced within the follow-up period.

4 | DISCUSSION

Our retrospective study describes dogs that developed urethral obstruction secondary to AUS-associated extraluminal fibrous capsule formation. This AUS-associated complication has not been described previously in detail, but development of urethral obstruction from a band of tissue surrounding the urethra has been noted in previous studies of dogs with AUS devices.[3,6] This complication appears to be rare, with only 6 dogs identified with this complication at the UCD-VMTH over a 10-year period. Although in 1 dog, it occurred 20 days after AUS implantation; in the remaining dogs, urethral obstruction was recognized between 429 and 2457 days after the AUS device was placed.

In all dogs, the capsule associated with the AUS device was causing stenosis of the urethra, which was relieved by transection or substantial removal of the capsule. When a device such as an AUS is implanted, a foreign body response occurs. This response is characterized by deposition of blood proteins, platelets, fibrinogen, and other blood products on the surface, followed by recruitment of macrophages and granulation tissue formation.[9] A fibrous capsule forms because of ongoing inflammatory signaling that activates fibroblasts; the fibrous capsule then persists while the implant is in place.[9] It is hypothesized that the capsule in the dogs of our study, which was consistent with mature fibrous tissue in the 2 dogs in which it was submitted for histopathology, underwent contracture causing urethral obstruction. It is likely that some degree of foreign body reaction and capsule formation around the urethra and AUS device develop in all dogs with the AUS device, similar to what is observed in humans. Some dogs may experience a more marked reaction, either because of individual variability or specific factors that induce a more marked immune reaction, such as bacterial infection of the associated tissues or AUS device size. This complication has been recognized in humans with AUS devices. In humans with AUS devices, a fibrous capsule consistently forms not only around the AUS device, but also around the urethra that is within the region of the AUS cuff, even in patients without any clinical signs.[10] It was initially thought that urethral atrophy secondary to cuff placement around the urethra was responsible for humans with AUS developing stranguria, but it has now been determined that affected patients had compression of the urethra from the capsule associated with the AUS device.[10,11] In these studies, after capsulotomy or capsulectomy, the urethra returned to its

normal shape from the "hour glass" appearance caused by capsular contracture.[10,11]

In humans, smooth implant surfaces and subclinical bacterial colonization of the implant are risk factors for development of excessive fibrosis that then leads to capsular contracture, with this outcome occurring months to years after implantation.[9,12,13] Bacterial colonization may enhance the immune response, leading to inflammation, fibrosis, and contracture of the capsule.[12,13] This reaction is influenced by the type and extent of bacterial colonization and bacterial biofilm formation. Certain bacteria, particularly *S. epidermidis*, have been significantly associated with contracture of the implant-associated capsule.[12,13] In studies evaluating urethral compression secondary to the AUS-associated capsule, bacterial culture of the capsule was not consistently performed and therefore it cannot be determined whether infection is a risk factor in humans with this specific implant type.[10,11] In our study, all dogs with clinically relevant contracture of the AUS-associated capsule had positive bacterial growth from samples collected from the capsule and AUS device at the time of surgery. Bacterial colonization might have contributed to capsular contracture in these dogs, similar to humans, but this outcome cannot be confirmed given the low numbers of bacteria cultured in some instances. The frequency of positive bacterial culture of the AUS device and capsule is also unknown in clinically normal dogs with AUS devices. Bacterial colonization in the dogs in our study could have originated during surgical placement of the AUS device or could have arisen secondary to translocation from the urinary tract or from bacteremia. Translocation from the urinary tract might be less likely because bacteria identified from positive urine cultures did not consistently correspond to those cultured from the AUS device, capsule, or associated fluid. In humans, prophylactic or postoperative antimicrobial administration and use of local antibiotics or antiseptics appear to decrease the incidence of capsular contracture with implants.[14]

Retrograde positive contrast cystourethrography and cystourethroscopy were important in the evaluation of urethral obstruction in the dogs in our study. Both provided complementary information on lower urinary tract anatomy and enabled diagnosis of mechanical urethral obstruction at the level of the AUS cuff. They also allowed confirmation of resolution of urethral obstruction intraoperatively, immediately after capsulotomy or capsulectomy.

In all dogs in our study, the capsule either was transected or substantially removed, resulting in resolution of urethral obstruction. Capsulotomy or capsulectomy both have been described in humans with constriction of the urethra secondary to an AUS-associated capsule.[10,11] The AUS device was not replaced in any dog in our study, and it is therefore unknown whether dogs could be predisposed to recurrent urethral obstruction because of capsular contracture, but replacement of the cuff has been performed in humans.[10,11] In the dogs of our study, urinary incontinence after AUS removal and treatment of the capsule was managed by cystoscopic-guided laser ablation of an intramural ectopic ureter and estriol in 1 dog with an ectopic ureter identified on evaluation of urethral obstruction and in the remaining dogs with medications including estriol and phenylpropanolamine. Urinary continence was adequately controlled in 2 of 3 dogs that had longer-term follow-up. In these 2 dogs, medical management previously had failed to adequately control urinary incontinence. This improved response to medical management could reflect changes in the structure of the urethra such as persistent fibrosis or urethral tissue bulking that developed secondary to presence of the AUS device.

Limitations of our study include its retrospective design, variable follow-up time, and small number of dogs considering that this complication appears to be uncommon. One dog had only 3 days of follow-up after hospital discharge from surgical removal of the AUS, but 4 dogs were followed for 247 and 2313 days (the remaining dog was followed-up for 23 days). This variable follow-up duration might have affected detection of recurrent urethral obstruction and urinary incontinence in our study. Histopathology of the capsule associated with the AUS was only available in 2 dogs; the lack of histopathology in the remaining dogs makes it more challenging to determine the clinical relevance of positive bacterial cultures in the 6 dogs in our study. Although it would be interesting to culture the AUS device (and, if present, the associated capsule) of dogs without stranguria or other AUS-related complications, doing so would require unnecessary surgery or sampling in these patients and therefore was not feasible. Obtaining both histopathology of the capsule and aerobic bacterial culture of the AUS device and capsule is recommended in dogs with AUS-related complications to assist in better defining any potential role of bacterial colonization of these in dogs with complications.

Urethral obstruction secondary to capsule formation appears to be an uncommon but clinically important complication associated with the use of AUS in dogs. Additional investigations are needed to evaluate this complication further, particularly the association with bacterial colonization of the capsule and AUS device and possible risk factors associated with development of these complications.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflicts of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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