Cutaneous seeding of transitional cell carcinoma of the urinary bladder after placement of a subcutaneous ureteral bypass device in a dog with bilateral ureteral obstruction

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CASE DESCRIPTION

A 12-year-old spayed female Jack Russell Terrier was presented with pollakiuria and stranguria.

CLINICAL FINDINGS

Transitional cell carcinoma (TCC) of the urinary bladder trigone and urethra was diagnosed via CT, cystoscopic, and histologic examinations. Azotemia developed 2 weeks following diagnosis, secondary to bilateral ureteral obstruction.

TREATMENT AND OUTCOME

Percutaneous antegrade ureteral stenting was unsuccessful; therefore, a subcutaneous ureteral bypass (SUB) device with 2 nephrostomy and 1 cystostomy catheters was surgically placed. Two months following placement of the SUB device, the dog developed a firm, multilobulated cutaneous mass at the site of the subcutaneous access port of the SUB device. Results of cytologic examination of cells aspirated from the mass were consistent with TCC. Within 1 month of confirmation of TCC of the cutaneous mass, the mass was ulcerated and infected, and the dog was euthanized because of signs of pain and perceived poor quality of life.

CLINICAL RELEVANCE

Seeding of neoplastic cells is a known complication of needle aspiration or biopsy or surgery in people and dogs with carcinomas. The occurrence of TCC at the SUB port site suggested caution with the placement of a SUB device in dogs with obstructive TCC. (*J Am Vet Med Assoc* 2021;258:877–882)

A 12-year-old spayed female Jack Russell Terrier presented to a tertiary referral veterinary hospital with a history of polyuria, polydipsia, pollakiuria, and stranguria. On initial physical examination, body weight was 13 kg (28.6 lb) and body condition score was 8/9. Numerous variably sized, soft, freely movable subcutaneous masses were palpable; cytologic examination of aspirated cells was consistent with lipomas. Irregular thickening in the proximal intrapelvic portion of the urethra was evident on rectal examination. Analysis of a midstream voided urine sample revealed a specific gravity of 1.004, pH of 7.0, nonsquamous epithelial cells, and moderate coccoid bacteria. Quantitative culture of the urine yielded no bacterial growth. Results of a CBC were unremarkable; biochemical analysis revealed increased serum creatinine concentration (1.8 mg/dL; reference interval, 0.6 to 1.5 mg/dL) and BUN concentration within reference limits (25 mg/dL; reference interval, 9 to 31 mg/dL). Ultrasonographic images[a] of the dorsal aspect of the wall at the trigone of the urinary bladder were consistent with a mass that had frond-like luminal projections. The ventral portion and portions of the apical aspects of the bladder wall were thickened. The right kidney had moderate hydronephrosis, and the left kidney had mild pyelectasis.

ABBREVIATIONS

FNA	Fine-needle aspirate
SUB	Subcutaneous ureteral bypass
TCC	Transitional cell carcinoma

No radiographic evidence of metastatic neoplasia was noted in 3 views of the thorax. Computed tomographic pre- and postcontrast[b] (770 mg of I/kg [350 mg of I/lb], IV) images were acquired with a helical 64-slice CT scanner[c] for radiotherapy planning. Images were not formally evaluated by a board-certified radiologist, but a radiologist reviewed the images alongside a radiation oncologist for radiotherapy planning.[d,e] Computed tomographic images were concordant with the ultrasonographic images, with an irregularly thickened, contrast-enhancing broad-based mass at the trigone of the bladder that extended to the proximal portion of the urethra and along the dorsolateral bladder wall **(Figure 1)**. Bilateral hydroureter (right ureter diameter, 0.4 cm; left ureter diameter, 0.6 cm) and hydronephrosis with pyelectasia (right renal pelvis width, 2.5 cm; left renal pelvis width, 1.6 cm) were present. Transurethral cystoscopy[f] confirmed the presence of the mass and its extension into the proximal portion of the urethra. A tissue sample was obtained with endoscopic forceps, and the microscopic morphological diagnosis was urothelial TCC.

After diagnosis, the owner elected to begin piroxicam[g] (0.3 mg/kg [0.14 mg/lb], PO, q 24 h), with an intent to pursue palliative radiotherapy. After 7 days of piroxicam administration, biochemical analysis revealed progressive azotemia (serum creatinine, 2.8 mg/dL; BUN, 35 mg/dL); piroxicam was discontinued. Fourteen days following the diagnosis of TCC and im-

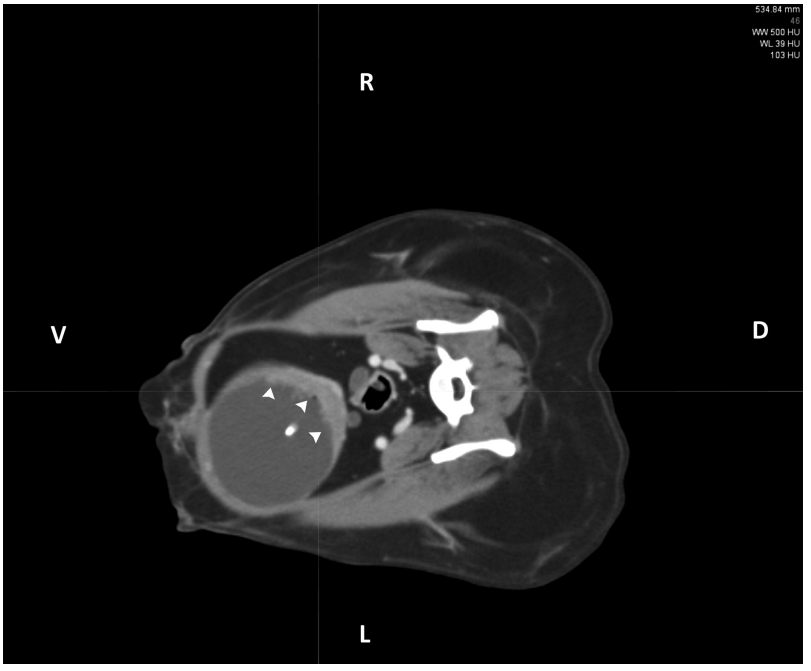

Figure 1—Transverse postcontrast CT abdominal image obtained at the level of L7 in a 12-year-old dog with TCC of the urinary bladder and subsequent bilateral ureteral obstruction. An irregularly thickened, mildly rim-enhancing, infiltrative mass affects the wall of the dorsal aspect of the urinary bladder wall (arrowheads). Window width, 500 HU; window level, 39 HU; 2-mm slice thickness. D = Dorsal. L = Left. R = Right. V = Ventral.

mediately prior to radiotherapy, the dog became anorexic and lethargic. Biochemical analysis revealed severe azotemia (serum creatinine, 7.5 mg/dL; BUN, 130 mg/dL), hyperphosphatemia (9.1 mg/dL; reference interval, 3.3 to 6.8 mg/dL), and hyperkalemia (5.9 mg/dL; reference interval, 3.6 to 5.3 mg/dL). Repeated ultrasonographic examination showed bilateral obstructive hydronephrosis, which prompted the owner to elect intervention. Percutaneous antegrade ureteral stenting was attempted as previously described.[1] Stenting of the right ureter was attempted first because the right renal pelvis was more dilated (vs the left renal pelvis); however, the guidewire, through the placed renal access sheath, or combined guidewire and catheter could not be passed across the malignant obstruction at the ureterovesicular junction.

Because of the inability to place a percutaneous stent, the owner was given 2 options: stopping intervention or converting to an open surgical approach for bilateral ureteral reimplantation or placement of a SUB device,[h] consisting of a nephrostomy and cystostomy catheter connected to a subcutaneous shunting port; the owner elected the latter, considering the relative short time to complete a SUB procedure versus to perform bilateral ureteroneocystostomies. Briefly, the abdomen was incised at the ventral midline, with the incision extending from the xiphoid to the pubis. Abdominal surgical exploration revealed bilateral, irregularly shaped kidneys, bilateral hydroureters, a firm palpable mass at the dorsal aspect of the trigone of the urinary bladder, and multifocal to coalescing regions of white discoloration and heterogeneous texture on the spleen. The caudolateral pole of the right kidney was approached first. A 6.5F locking-loop nephrostomy catheter was placed over a guidewire into the right renal pelvis following antegrade positive contrast pyelography with 1 mL of iodinated contrast solution,[b] as previously described.[2] The same procedure was repeated with the left kidney. The urinary bladder was isolated and several saline (0.9% NaCl) solution–moistened laparotomy sponges were placed around the bladder. Following the placement of a purse-string suture, a stab incision was made into the bladder with a cystostomy insertion stylet. A single 6.5F locking-loop cystostomy catheter was then placed and secured to the bladder. The catheters from the kidneys and bladder were tunneled through the right lateral body wall and secured to a shunting port with 2 nephrostomy catheter outlets and 1 cystotomy catheter outlet. Device patency and absence of catheter kinking and leakage were confirmed with a Huber infusion set,[h] digital compression, and several fluoroscopic examinations. The shunting port was then sutured to the right lateral body wall. A standard left-sided urinary cystopexy was performed to ensure consistent positioning of the bladder to aid future radiotherapy, if elected. Because of the appearance of the spleen, a splenectomy was subsequently performed with a vessel-sealing device,[i] and the spleen was submitted for histologic examination.

The dog received IV fluid[j] therapy, which was quantitatively adjusted on the basis of weight changes and subjective postobstructive diuresis. A nasogastric tube was placed, and a continuous rate infusion of a hypoallergenic diet[k] (blenderized) and water was initiated 6 hours after surgery at one-third of the dog's resting energy requirement per 24-hour period. Codeine[l] (1 mg/kg [0.45 mg/lb], PO, q 8 h, as needed), trazodone[m] (4 mg/kg [1.8 mg/lb], PO, q 12 h, as needed), maropitant[n] (2 mg/kg [0.9 mg/lb], PO, q 24 h, as needed), and amoxicillin–clavunalate potassium[o] (13.75 mg/kg [6.25 mg/lb], PO, q 12 h) were initiated. The dog was discharged from the hospital 3 days after surgery with a mild persistent azotemia (serum creatinine, 2.7 mg/dL; BUN, 45 mg/dL). Standard flushing of the SUB device through the shunting port was performed prior to hospital discharge and 7 and 30 days after discharge. Histologic examination of the spleen revealed findings compatible with capsular fibrovascular tissue tags and fibrosis, lymphocytic hyperplasia, and extramedullary hematopoiesis. Piroxicam was not resumed postoperatively because of persistent azotemia and the dog's poor appetite.

Fifty-eight days following placement of the SUB device, physical examination revealed a large (maximum diameter, 7 cm), multilobulated, firm, fixed subcutaneous mass that originated at the site of the SUB shunting port and extended caudally to the inguinal region **(Figure 2)**. Extensions of the mass could be palpated both cranial and caudal to the port, closely associated with the insertion points of the nephrostomy and cystostomy catheters. Biochemical analysis indicated persistent azotemia (serum creatinine, 1.6 mg/dL; BUN, 57 mg/dL). Computed tomography of the thorax, abdomen, and pelvis indicated that both locking-loop nephrostomy catheters and the cystostomy catheter were appropriately positioned within the pelvis of each kidney and the lumen of the urinary bladder, respectively. Compared with the findings from the initial CT examination, bilateral hydroureter and hydronephrosis had not worsened. A 12 X 8 X 5-cm lobular, heterogeneous contrast-enhancing mass of the ventral body wall was centered at and surrounded the shunting port. This infiltrative mass extended cranially and caudally from the port and invaded into the abdominal body wall **(Figure 3)**. The irregular, soft tissue mass associated with the trigone of the urinary bladder had enlarged. Medial iliac, hypogastric, sacral, and bilateral inguinal lymph nodes were mildly to moderately enlarged. Cytologic examination of fine-needle aspirated cells from the body wall mass was consistent with TCC, characterized by polygonal cells and eosinophilic inclusions with a moderate degree of atypia **(Figure 4)**. Following diagnosis, maropitant[m] (2 mg/kg, PO, q 24 h, as needed), amoxicillin–clavulanate potassium[o] (13.75 mg/kg, PO, q 12 h), and codeine[j] (1 mg/kg, PO, q 8 h, as needed) were prescribed as palliative care. Because of rapid progressive growth, ulceration, and abscessation of the body wall mass as well as progressive lethargy, anorexia, and dehydration, the dog was euthanized (102 days after placement of the SUB device and 44 days after the diagnosis of port site metastasis). No necropsy was performed.

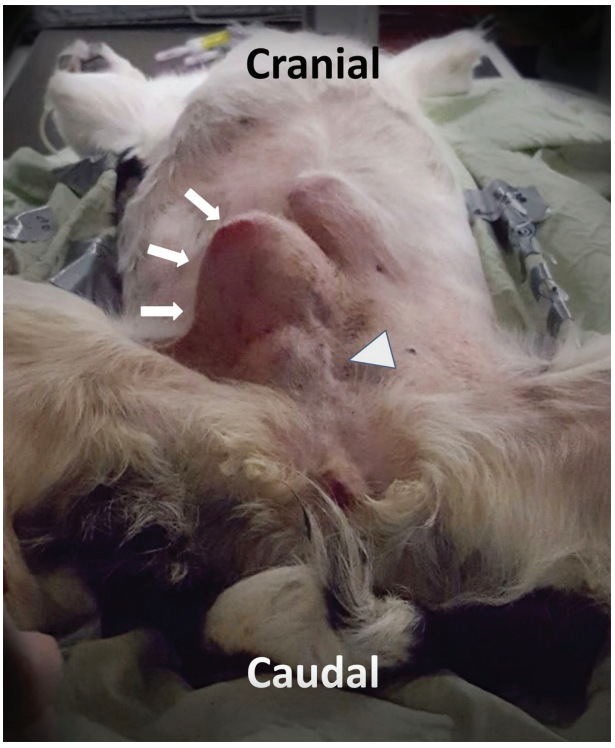

Figure 2—Photograph of a 12-year-old dog that had a large, multilobulated, firm mass (arrows) that developed to the immediate right of the ventral midline at the site of the shunting port of a SUB device that had been placed 58 days earlier because of bilateral ureteral obstruction secondary to TCC of the urinary bladder. Small extensions of the mass are seen caudally (arrowhead) near the insertion site of the cystostomy catheter. The dog is in dorsal recumbency.

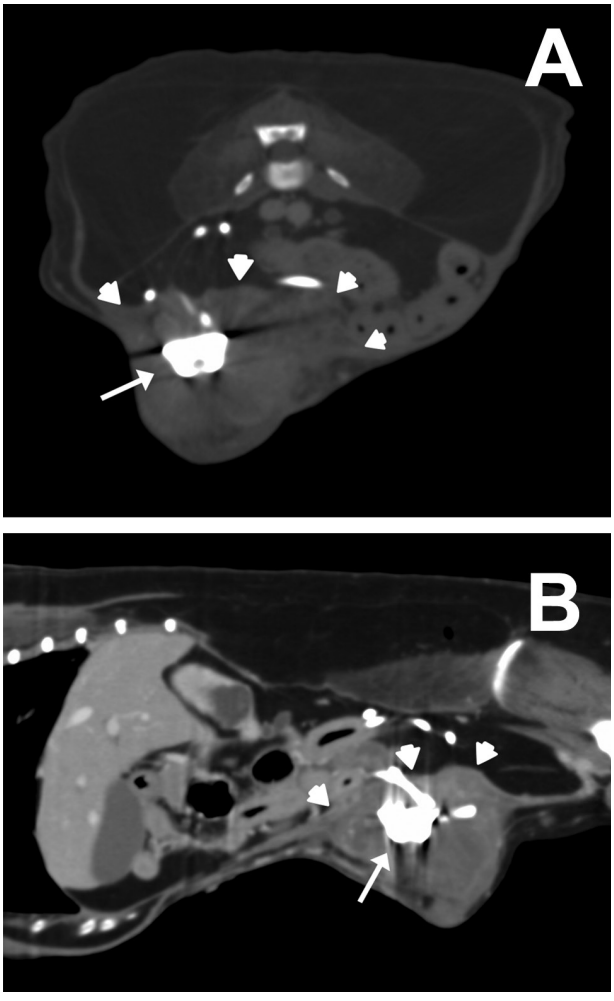

Figure 3—Postcontrast transverse (A; right to the left) and sagittal (B; cranial to the left) CT images of the dog in Figure 2 with a hypoattenuating, heterogeneous contrast-enhancing mass (arrowheads) that developed around the SUB shunting port (arrow). Images are displayed in a modified soft tissue window to minimize metal artifact from the SUB shunting port. Window width, 400 HU; window level, 40 HU; 2-mm slice thickness.

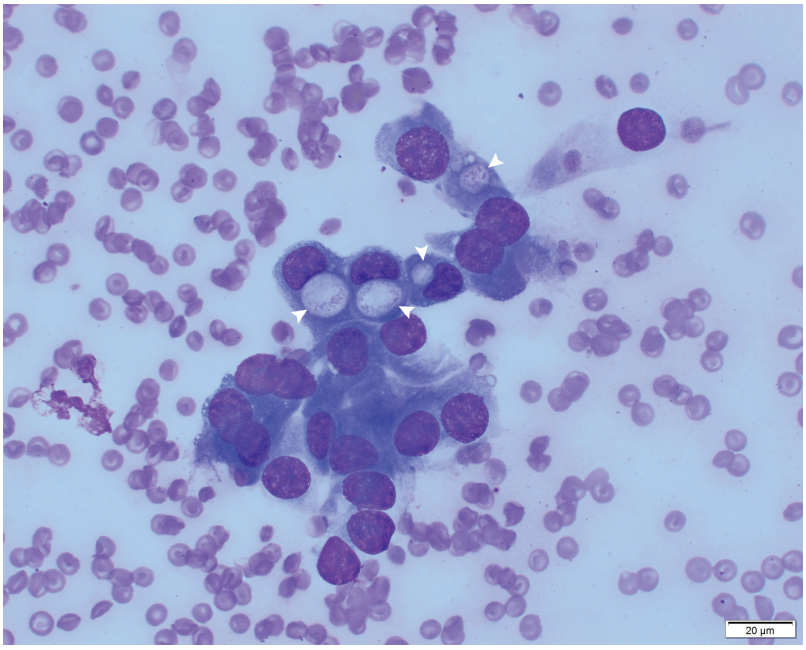

Figure 4—Photomicrograph of cells aspirated from the body wall mass of the dog in Figure 2. In addition to RBCs and peripheral blood-associated WBCs, polygonal to plump to spindyloid atypical cells are noted. These atypical cells are characterized by moderate amounts of basophilic to gray granular cytoplasm and prominent small, round to ovoid nucleoli. Eosinophilic inclusions are present within multiple cells (arrowheads). H&E stain. Bar = 20 µm.

Discussion

Primary malignancies of the lower urinary tract are rare in dogs, accounting for 0.5% to 1% of all canine cancers; however, TCC accounts for up to 75% of primary lower urinary tract tumors.[3] Canine TCC is an aggressive neoplasm with a moderate risk of metastasis to lymph nodes and lungs, and reported median survival times are < 1 year regardless of treatment,[3–10] except in cases of neoplasms confined to the apex of the urinary bladder in which partial cystectomy and adjuvant chemotherapy may yield a median survival time of approximately 2 years.[11] Ten percent of dogs with TCC reportedly[12] have metastases to the skin, through direct extension of neoplastic cells from the urinary bladder, indirect extension from the blood stream and lymphatic system, and iatrogenic implantation of neoplastic cells during partial cystectomy.[12] Cutaneous metastasis of TCC of the urinary bladder appears more common (10%)[12] in dogs versus people (< 1%),[13] possibly owing to the high incidence of aggressive, muscle-invasive TCC in dogs.[4]

Cutaneous metastasis of TCC for the dog of the present report may have developed because of dissemination of neoplastic cells in the urine to the skin directly above the shunting port during port aspirating and flushing sessions. Neoplastic cells may have also tracked along the SUB device or urine leakage or neoplastic cell seeding during surgery may have led to metastasis to the skin. However, the route of metastasis could not be definitely determined because necropsy was not performed. Because the bulk of the cutaneous mass was not at the ventral abdominal incision (ie, on midline) and instead was at the lateralized site of the shunting port, surgical dissemination was considered less likely.

Another possibility for cutaneous metastasis was that the inflammatory microenvironment around the SUB device encouraged hematogenous or lymphatic dissemination of neoplastic cells; inflammation has been implicated in creating a microenvironment favorable to metastasis.[14] Authors of 2 reports[15,16] detailed metastasis of squamous cell carcinoma of the hypopharynx and spindle cell carcinoma of the hard palate along totally implantable venous access ports in people. These authors speculated that the inflammatory microenvironment around the venous catheter in conjunction with its proximity to the primary tumor allowed for direct invasion of the catheter tract by neoplastic cells.[16]

Human and canine cases of needle-tract seeding of carcinoma cells caused by percutaneous aspiration or biopsy have been reported,[17–26] especially with removal of the needle from the mass through the skin. Although needle-tract seeding during fine-needle aspiration is rare, TCC may be more likely to cause needle-tract seeding than other tumors.[23,24] The incidence of needle-tract seeding of neoplastic cells for men with prostatic neoplasia is < 1%[26]; the incidence of needle-tract seeding of neoplastic cells for dogs with TCC is unknown but this complication has been described.[20,21] Several factors may affect the prevalence of seeding of neoplastic cells following biopsy. In people, the focus has been on the occurrence following core-needle aspiration rather than fine-needle aspiration because the former is more common and allows for molecular analysis. Coaxial techniques may have lower seeding rates, compared with techniques that create a new path with each biopsy.[22–25] Similar to needle-tract seeding, dissemination of neoplastic cells to the skin at sites of laparoscopic trocarization in people with muscle-invasive bladder cancer undergoing radical cystectomy has occurred.[27] The development of laparoscopic-port metastasis may be caused by contamination of the port with neoplastic cells through tumor extraction, contamination of surgical instruments, and decompression of the peritoneal cavity.

A SUB device was placed in the dog of the present report because of progressive ureteral obstruction. The device consisted of a subcutaneous shunting port connecting 2 nephrostomy catheters and 1 cystostomy catheter, thereby allowing urine to bypass the obstructed ureters. The port allowed for regular flushing of the device and facilitated repeated urine sampling. Subcutaneous ureteral bypass de-

vices are commonly and successfully used with cats with benign ureteral obstructions,[28] but they are infrequently used with dogs with urinary tract disease; therefore, complications associated with SUB devices in dogs are less known. A device similar to the SUB device used for the dog of the present report has been used in people with extensive neoplasia of the urinary tract when ureteral stenting is ineffective or conventional surgery is contraindicated.[29,30] For the dog of the present report, the decision was made to place a SUB device following unsuccessful ureteral stenting. The dog was unstable under general anesthesia; therefore, speed was an important consideration, and the time to complete a SUB procedure was relatively shorter than the time to complete bilateral ureteroneocystostomies. Additionally, avoiding exposing the TCC during a cystotomy for bilateral ureteroneocystostomies was thought to reduce the risk of dissemination of neoplastic cells during surgery.[31] In retrospect, however, considering that subcutaneous dissemination of neoplastic cells occurred after placement of a SUB device for the dog of the present report, open cystostomy with ureteroneocystostomy or open surgical placement of ureteral stents may have been an equally suitable option for this dog.

To the authors' knowledge, the dog of this report with advanced TCC of the urinary bladder and bilateral ureteral obstruction was the first with dissemination of neoplastic cells at and adjacent to the shunting-port site of a SUB device. Cutaneous dissemination was suspected secondary to repeated urine sampling through the port or to SUB device placement by direct implantation. Urothelial carcinomas are highly exfoliative tumors and associated with seeding related to needle puncture and surgical manipulation in dogs and people. The risk of seeding of neoplastic cells should be considered when choosing available treatment options.

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Footnotes

a. SonoSite Edge II ultrasound with C11x probe, Fujifilm, Bothell, Wash.
b. Optiray 350 (Ioversol), Mallinckrodt Inc, Hazelwood, Mo.
c. Aquilion 64, Toshiba America Medical Systems Inc, Tustin, Calif.
d. Pinnacle, version 16.2, Philips, Fitchburg Wis.
e. MIM Maestro, version 6.12, MIM Software Inc, Cleveland, Ohio.
f. Storz visual field rigid endoscope (2.7 mm; 30°), Karl Storz SE & Co KG, Tuttlingen, Germany.
g. Wedgewood Pharmacy, Swedesboro, NJ.
h. Norfolk Vet, Skokie, Ill.
i. LigaSure Impact, Medtronic, Minneapolis, Minn.
j. Lactated Ringer solution, Abbott Laboratories, North Chicago, Ill.
k. ProPlan Veterinary Diets HA Hydrolyzed Canine Formula, Purina, St Louis, Mo.
l. West-Ward Pharmaceuticals Corp, Eatontown, NJ.
m. PLIVA HRVATSKA d.o.o., Zagreb, Croatia.
n. Cerenia, Zoetis, Kalamazoo, Mich.
o. Clavamox, Zoetis, Kalamazoo, Mich.

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