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NEOPLASTIC DISEASE

Urachal Adenocarcinoma in a Dog

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Summary

An 8-year-old neutered female Labrador retriever was presented with a 3-year history of intermittent haematuria. Ultrasonographic evaluation of the urinary bladder revealed a 2 × 3 × 0.5 cm intraluminal mass arising at the dome. The mass was excised via partial cystectomy. Histopathological examination revealed neoplastic epithelial cells arranged in sheets, irregularly-branching tubules and acini within a fibrovascular stroma. Neoplastic cells were cuboidal to polygonal with abundant foamy amphophilic cytoplasm, typically with a single, large, clear intracytoplasmic vacuole and eccentric nucleus ('signet ring' cells). Neoplastic tubules were often ectatic and contained abundant mucin. Immunohistochemically, the neoplastic cells had weak, cytoplasmic immunoreactivity for cytokeratin 7 and rare, but strong, nuclear immunoreactivity for CDX2. Based on the cellular morphology, immunolabelling characteristics and anatomical location, a diagnosis of adenocarcinoma of urachal origin was made. To the authors' knowledge, this is the first reported case of urachal adenocarcinoma in a dog.

Keywords: dog; urachal adenocarcinoma; urachal remnant; urachus

Reported sequelae to human urachal remnant persistence include congenital urachal anomalies (e.g. patent urachus, umbilical–urachal sinus, vesicourachal diverticulum and urachal cyst), urachal remnant infections and urachal tumours (Jeong-Sik *et al.*, 2001). There are few reports of urachal disease in dogs; these include a congenital urachal diverticulum (Lojszczyk-Szczepaniak *et al.*, 2010), patent urachus (Osborne *et al.*, 1966), vesicourachal diverticula and urachal cysts (Groesslinger *et al.*, 2005). Canine urachal neoplasia has not been reported; however, it is recognized that urachal remnants represent a potential source for the development of urinary bladder adenocarcinoma (Meuten, 2002).

An 8-year-old neutered female Labrador retriever was presented to the Taylor Crossing Animal Hospital, Montgomery, Alabama, with a 3-year history of intermittent haematuria, overflow incontinence and presumptive urinary tract infection. The dog also had a history of low-grade chronic renal failure and mild eosinophilia (1.33×10^9/l; normal range $0.0–1.2 \times 10^9$/l). Immediately prior to presentation, a 1 × 0.5 × 0.5 cm soft, tan mass was voided in the urine. Ultrasonographic evaluation of the urinary bladder found an intraluminal, polypoid and sessile, 3 × 2 × 0.5 cm mass with echogenic (mineralized) spicules arising from the dome (Fig. 1). The mass was removed surgically via partial cystectomy (Fig. 2). Grossly, the mass was variegated grey–brown in colour and heterogeneous with discernible cystic structures and mucin accumulation on the cut surface.

The tissue was fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections (5 μm) were stained with haematoxylin and eosin (HE). Selected sections were subjected to immunohistochemistry (IHC) for detection of CDX2 (pre-diluted rabbit monoclonal antibody, Cell Marque, Rocklin, California, USA) and

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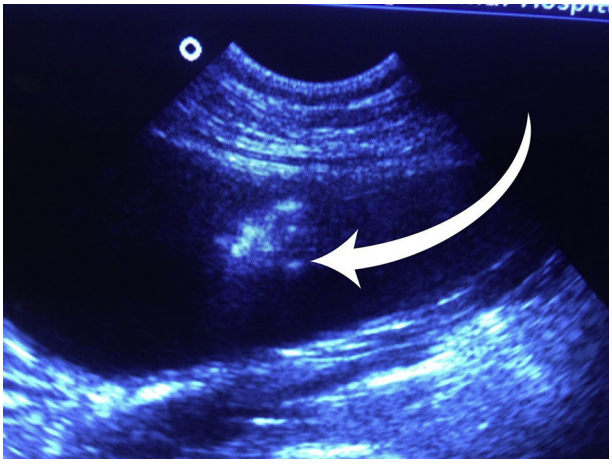

Fig. 1. Ultrasonographic image of the urinary bladder showing an irregularly-shaped intraluminal mass (arrow).

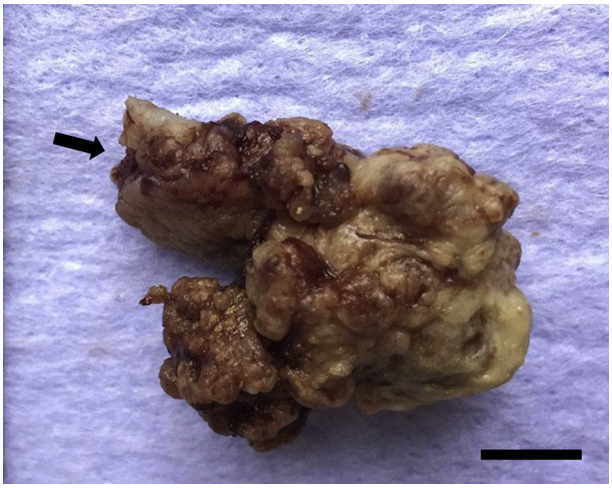

Fig. 2. Gross image of the surgically excised polypoid mass (surgical margin indicated by the arrow) showing the variegated luminal surface. Bar, 5 mm.

cytokeratin 7 (CK7; pre-diluted rabbit monoclonal antibody, Hoffmann–La Roche, Tucson, Arizona, USA) using an automated system (BenchMark Ultra, Ventana Medical Systems, Tuscon, Arizona, USA) with a polymer-based horseradish peroxidase-conjugated detection kit with 3, 3′ diaminobenzidine as the chromogen (Hoffmann-La Roche). Antigen retrieval was performed by using a Tris-based reaction buffer (pH 7.6) (Reaction Buffer Concentrate × 10, Ventana Medical Systems). Slides were counterstained with haematoxylin. Sections of normal canine colon and a canine mammary carcinoma were used as positive controls for CDX2 and CK7, respectively. For negative reagent controls, the primary antibodies were replaced with equivalent non-homologous immune sera. Negative internal tissue controls in immunolabelled slides consisted of non-epithelial tissue elements such as blood vessels and smooth muscle, which did not demonstrate any immunoreactivity for CDX2 or CK7.

On histological examination, effacing and expanding the lamina propria of the urinary bladder wall, and elevating the overlying urothelium, was an unencapsulated, fairly well-demarcated mass composed of neoplastic epithelial cells arranged in sheets, irregularly-branching tubules and acini within a fibrovascular stroma (Fig. 3). Individual neoplastic cells had abundant, foamy, amphophilic cytoplasm (Fig. 4), often with a single large clear vacuole and, frequently, an eccentric, round to irregularly-shaped nucleus ('signet ring' cell) with multifocal, delicate, intraluminal, papillary fronds. Approximately 50% of the neoplastic tubules were ectatic and contained abundant, often mineralized, amphophilic intraluminal material (mucin) admixed with sloughed epithelial cells, foamy macrophages, neutrophils and eosinophils. The intratumoural connective tissue stroma was expanded multifocally by oedema fluid, haemorrhage, dense aggregates of eosinophils and low numbers of lymphocytes, plasma cells and neutrophils. Overlying the mass, the urothelium was composed of a single layer of attenuated epithelial cells that lacked contiguity with the neoplasm and merged, peripherally, into normal epithelium (Supplementary Fig. 1). The neoplastic cells had weak cytoplasmic immunoreactivity for CK7 and rare, but strong, nuclear immunoreactivity for CDX2 (typically associated with tubules and acini that were more differentiated) (Supplementary

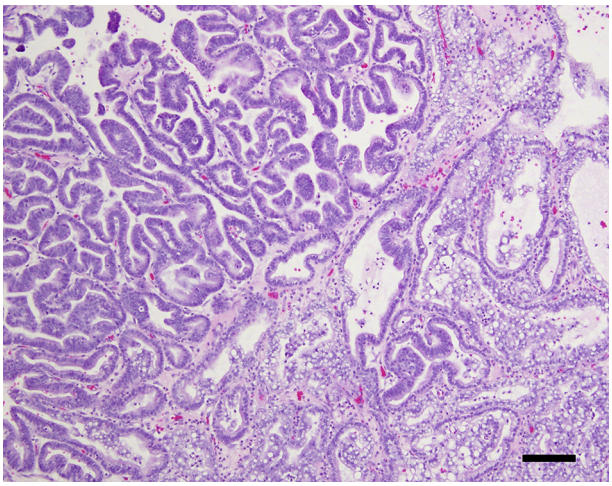

Fig. 3. Photomicrograph showing an unencapsulated mass within the urinary bladder lamina propria composed of neoplastic epithelial cells arranged in irregularly-branching tubules and acini. HE. Bar, 100 μm.

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