Metastatic anal sac adenocarcinoma in a dog presenting for acute paralysis

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Abstract — A 4-year old, female spayed terrier was referred for hind end paresis that rapidly progressed to paralysis. Spinal radiographs revealed vertebral collapse and bony lysis. Myelography confirmed spinal cord compression and surgical exploration found an extradural soft tissue mass. Metastatic anal sac adenocarcinoma was diagnosed at postmortem examination.

Résumé — Adénocarcinome métastasique des sacs anaux chez un chien présenté pour paralysie aiguë. Une chienne terrier stérilisée, âgée de 4 ans, fut référée pour une parésie postérieure qui a rapidement progressé vers la paralysie. Les radiographies de la colonne ont révélé un collapsus vertébral et de la lyse osseuse. La myélographie a confirmé une compression de la moelle épinière et l'exploration chirurgicale a démontré une masse extradurale de tissu mou. L'examen post mortem a permis de diagnostiquer un adénocarcinome métastasique des sacs anaux.

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4-year-old, 16-kg, female spayed terrier-cross was referred as an emergency to the Ontario Veterinary College with a 1-month history of progressive back pain and a 12-hour history of acute hind end paresis. Neurological examination of the recumbent dog revealed normal cranial nerve function; extensor rigidity of the thoracic limbs, with normal spinal reflexes; absence of purposeful movement bilaterally in the pelvic limbs, with normal to increased spinal reflexes; and normal pain perception in all limbs. Back pain was localized to the caudal lumbar and cranial thoracic regions; a reluctance to having the neck manipulated was also evident. Other body systems were within normal limits. Twenty minutes after the initial neurological evaluation, spinal reflexes could no longer be elicited in the right pelvic limb and were significantly decreased in the left pelvic limb. The neurological status continued to deteriorate rapidly, and approximately 1 h after admission, the patient was found to have complete loss of spinal reflexes and pain sensation in the pelvic limbs and tail region. Because of the rapid neurological deterioration, myelomalacia secondary to intervertebral disc disease, vascular compromise, or neoplasia was suspected. A poor prognosis, regardless of the etiology, was given to the owners, but they opted to proceed with diagnostic testing. Oxymorphone (Numorphan; Dupont Pharma, Mississauga, Ontario), 0.05 mg/kg bodyweight (BW), IV, and dexamethasone (Vetoquinol, Lavaltrie, Quebec), 0.25 mg/kg BW, IV,

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were administered to provide analgesia and sedation and decrease spinal cord inflammation, respectively.

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Three radiographic views of the thorax, taken under sedation, did not show any evidence of metastatic lung disease. Propofol (Rapinovet; Abbott Laboratories, Montreal, Quebec), 4 mg/kg BW, IV, was administered for anesthetic induction; anesthesia was maintained with isoflurane (Aerrane: Janssen, Toronto, Ontario) and oxygen via endotracheal intubation. Survey spinal radiographs of the thoracolumbar area taken under general anesthesia showed that the 9th thoracic (T) vertebral body had collapsed and suggested that bony lysis involving the body of the 5th and, possibly, the 3rd lumbar (L) vertebra was also present (Figure 1). Lumbar myelography was performed at the L5-6 intervertebral space using a 20 G, 1.5-inch spinal needle. Under fluoroscopic guidance, 4 mL of a noniodinated contrast agent (Omnipaque; Amersham Health, Oakville, Ontario) was injected and confirmed a right-sided compression of the spinal cord at the level of L2 and L3. Dye absorption by the spinal cord at the level of L5 and the presence of dye in the central canal was suggestive of myelomalacia (Figure 2). Euthanasia was recommended based on the severity of the neurological status and likelihood of neoplasia; the owners declined and elected for surgical exploration and biopsy to confirm the diagnosis.

An initial pediculectomy was performed at the level of L4 and L5 on the right side through grossly normal bone; it revealed no evidence of spinal cord compression. A durotomy was performed at that level and was not supportive of a diagnosis of myelomalacia. A second pediculectomy was performed at the level of L2 and L3 on the right side; this indicated bony lysis of the body of L3 and the presence of an extradural, soft tissue mass compressing the spinal cord at that level. The mass was

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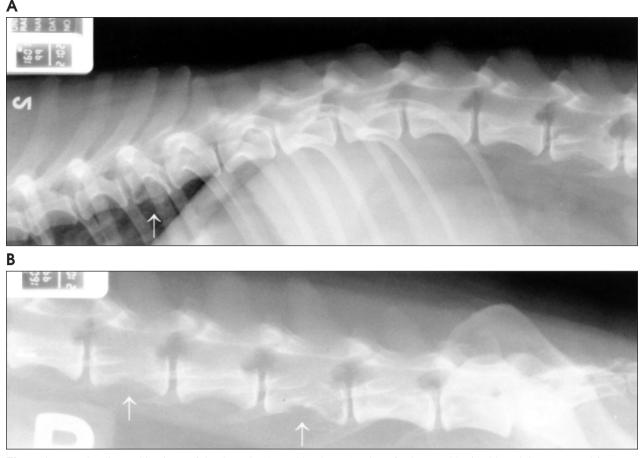
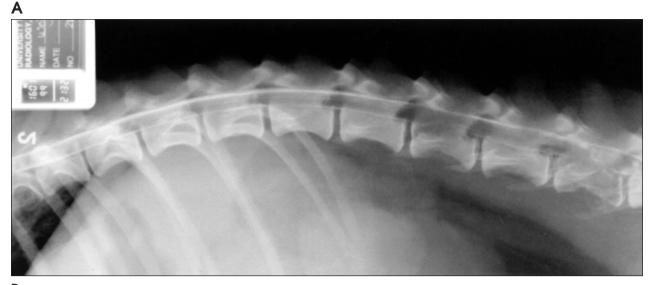


Figure 1. Lateral radiographic views of the thoracic (A) and lumbar (B) spine of a 4-year-old mixed breed dog presented for acute paresis. Note a pathological compression fracture of T9 vertebra (white arrow) (A) and bony lysis affecting the vertebral bodies of L5, and possibly L3 (white arrows) (B).

biopsied and intraoperative cytological evaluation of imprints revealed high cellularity with sheets of epithelial cells containing secretory granules and forming acini. Anisokaryosis was noted, but no evidence of mitoses or inflammation was present in these samples. Based on the surgical and cytological findings, as well as the severity of the neurological status, the dog was euthanized.

Postmortem examination showed bony lysis, pathological fracture, and collapse of vertebral bodies T4 and T9 and bony lysis with tumor invasion of vertebral bodies L3 and L5 (Figure 3). In addition, multiple nodular lesions of varying diameters were found within the spleen (2 to 10 mm in diameter) and liver (5 to 30 mm in diameter). Grossly, all abdominal lymph nodes were normal in size, and the pancreas and urinary bladder were also normal, making metastatic pancreatic or transitional cell carcinomas unlikely. The right anal sac was enlarged to approximately twice the size of the left and had a multinodular texture, with nodules ranging in size from 1 to 3 mm in diameter. Histological evaluation of tissue samples confirmed adenocarcinoma of the apocrine glands of the right anal sac, with metastasis to the liver, spleen, vertebral bodies, and vertebral canal. All histological sections were composed of highly cellular lobules separated by a fibrovascular stroma. A solid pattern predominated with some acini and tubular formation. Three-fold anisokaryosis was evident and there were 1 to 2 nucleoli per nucleus. Mitotic figures ranged from 2 to 3 per high power $(400 \times)$ field. The spinal cord in the thoracic region (in sections made at the level of pathological fractures) was most severely affected by Wallerian degeneration. Similar changes, although less severe, were seen in sections of the spinal cord from the lumbar region.

Anal sac adenocarcinoma is a rare tumor that, typically, affects older female dogs (1-3). The dog in the current report was younger than those previously reported in the literature and had an unusual clinical presentation. Rather than the clinical signs usually associated with the primary tumor or metastasis to local lymph nodes, the signs in this dog consisted solely of acute and rapidly progressing pelvic limb paralysis, secondary to metastasis to the vertebral bodies and vertebral canal. Signs of progressive paresis that developed for 1 mo prior to presentation were likely secondary to growing metastatic tumor at the level of L3 and L5, resulting in progressive spinal cord compression. Acute progression of neurological signs seen just prior to presentation was likely due to pathological fracture of T4 and T9 vertebral bodies, resulting in compressive myelopathy. The anal sac mass was small and was identified only during postmortem examination after



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Figure 2. Lateral (A) and ventrodorsal (B) radiographic views of a myelographic study in a 4-year-old mixed breed dog presented for acute paresis. Note the loss of ventral dye column at the level of L3 and L5 vertebrae as well as evidence of dye in the central spinal canal and absorbed into the spinal cord parenchyma at the level of L5 vertebra (A). The dye column also appears deviated to the left at the level of L3 vertebra (white arrow) (B).

documenting cellular acinar formation on cytological examination.

Spinal metastasis has rarely been reported with anal sac adenocarcinoma. A review of the veterinary literature turned up 1 case that was reported to have spinal cord metastasis identified during postmortem examination, although there were no clinical signs attributable to this lesion (1). Another dog was reportedly diagnosed with anal sac adenocarcinoma after surgical resection of a pulmonary metastasis; it subsequently developed a lytic lesion in the lumbar spine (2). In a recent retrospective study evaluating 43 cases of canine anal sac adenocarinoma, vertebral metastasis was identified in only 1 dog (3).

Anal sac adenocarcinoma is a highly malignant and locally invasive tumor. Metastasis typically occurs locally via lymphatic drainage to sacral, iliac, and lumbar lymph nodes and is frequently present at the time of diagnosis (1–3). Distant metastasis has been reported less commonly and usually affects the spleen, liver, and lungs (1–3). One study reported that 22 of 23 cases evaluated for metastatic lesions had local, and, sometimes, also distant metastases (1). In 2002, Bennett et al (3) reported that 79% of their cases had metastases documented at the time of diagnosis.

Other tumors, such as carcinomas of mammary, lung, and prostatic origin, along with transitional cell carcinoma of the urinary bladder, have been reported to metastasize frequently to the vertebral column (4–7). One study that focused on skeletal metastasis of carcinomas reported that skeletal metastasis was the initial clinical manifestation in 79% of 19 dogs: 4 dogs presented for neurological signs associated with spinal metastasis and 10 had radiographic or postmortem evidence of spinal metastasis; none of these cases were diagnosed with anal sac adenocarcinoma (6).

Metastatic or secondary spinal neoplasia is identified frequently in humans and often develops secondary to carcinomas of the breast, lung, prostate, and kidney (8,9). Radical resection is often limited in such cases due to local anatomy and is not warranted given that dissemination is already present (10). Chemotherapy, radiation therapy, and spinal decompression, with or without stabilization, are commonly used as palliative measures <image>

Figure 3. Postmortem photographs of the thoracic (A) and lumbar (B) spine of a 4-yearold mixed breed dog presented for acute paresis. Note the marked bony lysis affecting the bodies of T4 and T9 and L3 and L5 vertebrae. The bodies of T4 and T9 vertebrae also appear compressed cranio-caudally suggesting pathological fracture and collapse.

(10,11). In dogs, curative intent spinal surgery for tumor resection and spinal stabilization has been attempted to treat primary vertebral neoplasia (12), but this has not been reported in the treatment of spinal metastasis. Palliative treatment was not considered to be an option in this case due to the severity of the neurological signs and because multiple vertebrae were affected.

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Though uncommon, vertebral metastasis of adenocarcinoma of the apocrine glands of the anal sac is possible and may result in neurological signs being the first clinical signs associated with this type of tumor. It is also possible that vertebral metastasis is underdiagnosed in cases of anal sac adenocarcinoma, as the spine may not be evaluated routinely during postmortem examination in cases where neurological deficits did not develop prior to death or euthanasia.

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