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NOTE

Surgery

Chronic spinal epidural abscess in a cat: a case report with an unusual imaging finding

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ABSTRACT. A 1-year-old domestic shorthair cat was evaluated for a chronic history of back pain, dysuria, and paraplegia. Radiographic and computed tomographic examinations showed circumferential widening of the vertebral canal at T13 and T14. A spinal epidural abscess (SEA) compressing the spinal cord from the level of T11 to L1 was suspected following intravenous contrast administration, and was confirmed by surgical exploration and histopathological analysis. The cat recovered its motor and bladder functions following surgical decompression and antibiotic therapy. SEA is a neurological emergency requiring prompt treatment. However, the present case had a prolonged disease course and pressure atrophy of the vertebrae was strongly suspected. To our knowledge, this imaging finding has not been reported in dogs or cats with SEA.

KEY WORDS: computed tomography, delayed diagnosis, epidural empyema, feline, spinal cord compression

A spinal epidural abscess (SEA), otherwise referred to as spinal epidural empyema, is a rare disorder characterized by the accumulation of pus in the spinal epidural space. It can injure the spinal cord and often causes fever, spinal pain, and progressive neurological deficits [9, 10, 16, 24]. A prompt diagnosis and treatment, including systemic antibiotics with or without surgical drainage, are crucial for avoiding serious complications [7, 10, 19]. While most reported cases of canine and feline SEA have been diagnosed and treated within days to weeks after the onset of clinical signs [9, 10, 16, 24], limited information is currently available on SEA with chronic neurological deficits [13]. We herein report a feline case of chronic SEA.

A 1-year-old intact male domestic shorthair cat weighing 3.3 kg was referred to the Maizuru Animal Medical Center for an investigation of paraplegia. The cat was brought home 11 months previously when it was a stray kitten and had lived strictly indoors thereafter. Soon after adoption, the cat exhibited back pain, which resolved but recurred 4 months later and persisted thereafter. After 5 more months, the cat acutely developed hindlimb paresis and dysuria, which subsequently deteriorated into paraplegia.

Eighteen days before referral, the cat was presented to the referring veterinary clinic. Complete blood count and serum biochemical profile were unremarkable, but urinalysis revealed pyuria. Screening tests for feline leukemia virus antigens and anti–feline immunodeficiency virus antibodies were negative. The cat was treated for 1 week with prednisolone (0.5 mg/kg, PO, q12 hr), dantrolene (1 mg/kg, PO, q12 hr), and tamsulosin (0.01 mg/kg, PO, q12 hr). Since there were no signs of clinical improvement, the cat was referred to our center.

On admission, physical examination revealed hindlimb muscle atrophy and bladder overdistension. The rectal temperature was 37.7°C. A neurological examination showed paraplegia with preserved deep pain sensation and increased spinal reflexes in both hindlimbs without lateralization, which was consistent with a T3 to L3 spinal cord segment lesion. Complete blood count was unremarkable. Thoracic and abdominal radiographs were obtained. The cat had 14 thoracic vertebrae (with 14 pairs of ribs) and 6 lumbar vertebrae, and there was widening of the vertebral canal at T13 and the cranial portion of T14 (Fig. 1).

On the same day, the cat underwent a computed tomography (CT) examination using an 80-slice helical scanner (Aquilion Lightning TSX–036A; Canon Medical Systems, Otawara, Japan). Anesthesia was induced by propofol (6 mg/kg, IV) and maintained with 1.5–2.0% isoflurane in 100% oxygen. The cat was positioned in dorsal recumbency. Scans from the thoracic inlet to the pelvic inlet were obtained before and 40 sec after 600 mgI/kg iopamidol (Oypalomin 300; Fuji Pharma, Toyama, Japan) was manually injected via the cephalic vein.

Circumferential widening of the vertebral canal at T13 and the cranial portion of T14 was observed (Fig. 2A), with no other

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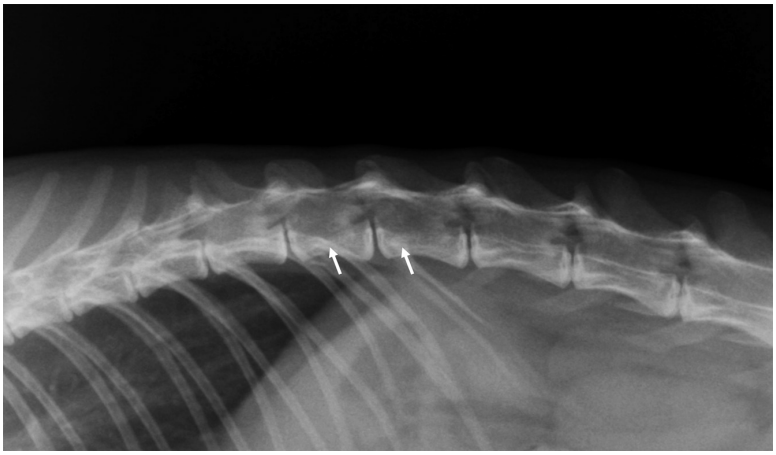

Fig. 1. Radiographic image of the thoracolumbar spine of the cat. The vertebral canal is widened at T13 and the cranial portion of T14 (arrows).

osseous involvement, such as endplate destruction. An elongated soft tissue structure was visualized within the right dorsal epidural space on post-contrast images (Fig. 2B and 2C). It extended from the level of the caudal endplate of T11 to the cranial endplate of L1, compressing the spinal cord most severely at the level of T13. The structure contained a poorly enhanced hypoattenuating center encapsulated with a thick strongly enhancing rim. Within the thoracic and the abdominal cavities, multifocal mediastinal and paraaortic lymphadenopathies were detected. Based on these imaging findings and the young age of the cat, differential diagnoses mainly included abscess and, to a lesser extent, hematoma and neoplasm.

Surgical exploration and decompression were performed the following day. The cat was administered cefazolin (20 mg/kg, IV), butorphanol (0.2 mg/kg, IV), and midazolam (0.2 mg/kg, IV), before anesthetic induction with propofol (5 mg/kg, IV) and intubation. Anesthesia was maintained with 1.5–2.5% isoflurane in 50% oxygen mixed with medical air. During the procedure, isotonic saline was administered intravenously at a rate of 5 ml/kg/hr. While preparing the surgical field, a small skin scar was found between the spinous processes of T13 and T14. A dorsal approach was applied to expose the vertebral column; no abnormalities were noted during the dissection of subcutaneous tissues and epaxial muscles. A right-sided hemilaminectomy was performed from the caudal end of T12 to the cranial end of T14 using a high-speed drill (RemB Micro Drill; Stryker Japan, Tokyo, Japan). After opening the spinal canal, pus surrounded by a capsule was visualized (Fig. 3), and a large amount of friable tissue and pus were retrieved from the epidural space. The exposed dural sac was reddish and irregularly thickened. The surgical field was irrigated with copious amounts of sterile saline and closed in a standard manner.

Tissue samples collected from the epidural space were submitted for histopathological analysis and aero/anaerobic bacterial culture and sensitivity. Histopathology revealed pyogranulomatous inflammation. Cultures showed the growth of *Streptococcus agalactiae*, *Bacteroides*, *Fusobacterium*, and *Porphyromonas* species, all of which exhibited sensitivity to amoxicillin, cefalexin, cefpodoxime, erythromycin, clindamycin, and chloramphenicol.

Postoperative management consisted of antibiotic therapy with cefazolin (20 mg/kg, IV, q12 hr) and analgesic therapy with buprenorphine (0.01 mg/kg, IV, q12 hr). Manual expression of the bladder was performed every 12 hr and continued at home after the cat was discharged on the 3rd day post-surgery. The oral antibiotic cefalexin (25 mg/kg, PO, q12 hr) was initially prescribed, but was not tolerated; therefore, 3 subcutaneous doses of 8 mg/kg cefovecin (Convenia; Zoetis Japan, Tokyo, Japan) were administered at 2-week intervals. Ten days after surgery, the cat was ambulatory with mild deficits in conscious proprioception in the hindlimbs and had regained urinary control. Six weeks after surgery, the cat had gained body weight to 4.0 kg and had recovered enough to run and jump, although mild proprioceptive ataxia remained in the hindlimbs.

SEA or empyema has rarely been reported in dogs and cats [9, 10, 16, 24]. The term abscess describes a localized collection of pus surrounded by inflammatory tissue, whereas empyema describes the accumulation of pus in a natural cavity [12, 16]. Although these terms have distinct definitions, they have been used interchangeably when describing lesions in the epidural space [12, 16]. In our case, findings consistent with an abscess were identified on contrast-enhanced CT and confirmed during surgical exploration by the presence of pus surrounded by a capsule. Thus, the diagnosis of SEA was considered to be more appropriate. Since most previous reports described no criteria for discriminating SEA and empyema, the term SEA is used for consistency in the present report.

The major causes of canine and feline SEA are *Staphylococcus* species, *Streptococcus* species, *Pasteurella* species, and *Escherichia coli* [7–10, 12, 16, 19, 20, 24, 29]. Bacteria can gain access to the spinal epidural space through hematogenous spread [9, 29], extension of an adjacent infection [16, 20, 24], penetrating injuries [26], or migrating foreign bodies [8, 12, 25]. In the present case, *S. agalactiae*, *Bacteroides*, *Fusobacterium,* and *Porphyromonas* species were isolated from the epidural material. The small skin scar above the lesion implies trauma as a possible route of infection. However, potential sources of infection were not thoroughly investigated using blood or urine cultures despite pyuria being reported by the referring veterinarian. Since *S.*

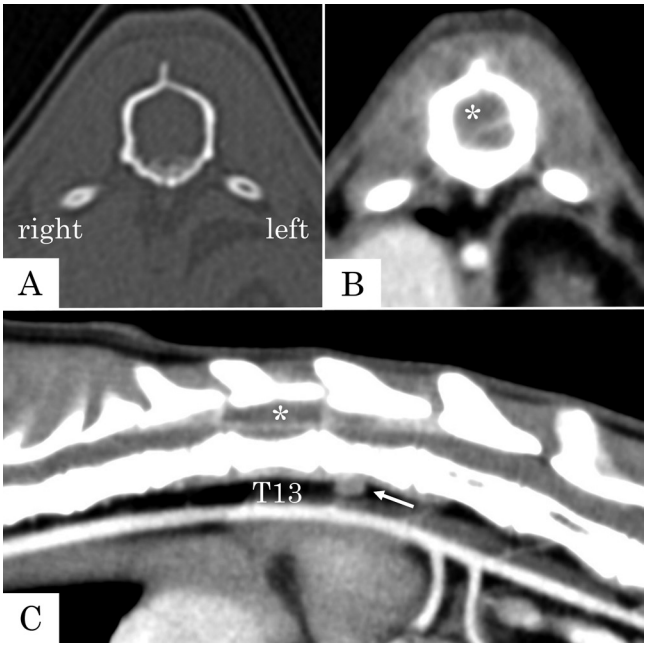

Fig. 2. Computed tomography images of the thoracolumbar spine of the cat. (**A**) Pre-contrast transverse image reconstructed with a bone algorithm (window level: 400 Hounsfield units [HU], window width: 3,000 HU). The transverse image is at the level of T13 mid-body. Note circumferential widening of the vertebral canal with smooth margins. Post-contrast (**B**) transverse and (**C**) sagittal images reconstructed with a soft tissue algorithm (window level: 50 HU, window width: 300 HU). The transverse image is at the same level as (A). There is an elongated, soft tissue structure with a thick strongly enhanced rim (asterisks) displacing the spinal cord to the left ventral direction. Additionally, there is an ovoid soft tissue structure ventral to the T14 vertebra (arrow), which may be a mildly enlarged lymph node.

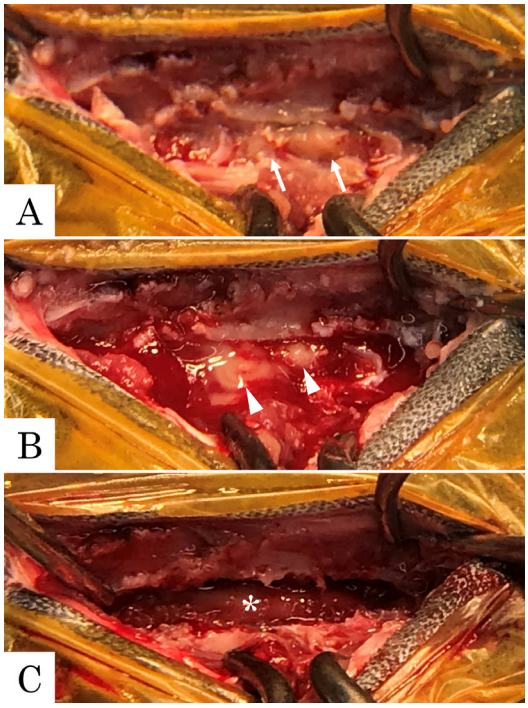

Fig. 3. Intraoperative views. (**A**) Pus surrounded by a capsule (arrows) was visualized after opening the spinal canal. (**B**) The capsule was easily ruptured and pus (arrowheads) and blood flowed out from the epidural space. (**C**) The dural sac (asterisk) was reddish and irregularly thickened.

agalactiae may cause septicemia and genitourinary infections [14, 15], hematogenous spread from a distant infectious focus, such as bacterial cystitis, was also possible. In contrast, impaired urinary function may have predisposed the cat to urinary tract infection and subsequent pyuria. The other isolated bacteria were obligate anaerobes that have generally been implicated in necrotic and suppurative lesions, often as mixed infections [10, 12, 14, 25].

SEA may pose a diagnostic challenge in part due to its rarity [7, 8, 12]. In most reported cases of canine and feline SEA, clinical signs are progressive, beginning with fever and spinal pain [7–10, 16, 25, 29]. However, the absence of fever does not preclude the diagnosis of SEA [9, 16]. The acute onset and worsening of neurological deficits are frequently observed with disease progression [7–10, 16, 19, 25, 29], as in the present case. Laboratory assessments may show elevations of inflammatory markers such as white blood cell count [9, 10, 16, 24], but they are not specific for the disease. Thus, the diagnosis of SEA depends mainly on imaging studies. In humans, gadolinium-enhanced magnetic resonance imaging (MRI) is the imaging test of choice for the diagnosis of spinal infection [17]. The MRI features of SEA include hyperintense epidural lesions on T2-weighted images and diffuse or peripheral enhancement on gadolinium-enhanced T1-weighted images [18, 27]. Additionally, diffusion-weighted imaging has been used to facilitate a differential diagnosis by showing restricted diffusion in an abscess [17]. In dog and cats, the T2-weighted and gadolinium-enhanced T1-weighted imaging findings of SEA were similar to those reported for human SEA [8, 9, 12, 16, 24]. When MRI is unavailable, myelography, CT myelography, or contrast-enhanced CT has been performed on dogs and cats suspected of having SEA [7–10, 13, 19, 20, 25, 26, 29]. Myelography or CT myelography is a sensitive technique for the detection and localization of epidural lesions. However, it has limitations such as an inability to characterize epidural material and a risk of introducing bacteria into the subarachnoid space upon the injection of a contrast agent [21]. In the present case, contrast-enhanced CT was performed to investigate the cause of vertebral canal widening. It sufficiently localized the lesion and facilitated differential diagnoses by showing findings that are consistent with SEA, namely, a central area of low density with rim enhancement. Similar findings were previously reported in humans with SEA who underwent contrast-enhanced CT [1, 28]. On the other hand, contradictory findings were reported in both a dog and cat with SEA [8, 29]. SEA consists of either a liquefied purulent collection or granulation tissue with microabscesses [5, 27]; therefore, the imaging features of SEA may vary depending on its composition [18, 27].

In the present case, vertebral canal widening with smooth margins presumably represented pressure atrophy of the vertebrae, which indicated slowly expansile lesion within the vertebral canal [3]. In dogs and cats, neoplastic lesions, such as peripheral nerve sheath tumors and intraparenchymal spinal cord ganglioneuroblastoma [2, 23], and non-neoplastic lesions, including spinal arachnoid diverticula [11], have been reported as causes of pressure atrophy of the vertebrae. However, to the best of our knowledge, this finding has not been described in dogs or cats with SEA presumably because the clinical signs of SEA typically progress rapidly [9, 10, 16, 24].

Delayed diagnosis of SEA may result in serious consequences; neurological deficits may become irreversible, or an expansion of pus in the epidural space or development of sepsis may be fatal [7, 10, 19]. In human SEA, the severity and duration of neurological deficits prior to surgery closely correlate with the final neurological outcome [21]. In cases of paralysis persistent for >24–36 hr, a surgical intervention is unlikely to improve neurological function [5, 6, 22]. In our case, the muscle atrophy in the hindlimbs accompanied with upper motor neuron signs probably resulted from disuse [4]. Physical and imaging findings substantiated the prolonged disease course of the cat, although the actual onset of disease was unknown. Nevertheless, surgical decompression and antibiotic therapy sufficiently improved its motor and bladder functions. Similarly, it was reported that a cat with SEA who presented with paraplegia that persisted for >1 month and severe muscle atrophy in the hindlimbs recovered independent ambulation following surgical decompression and antibiotic therapy [13]. These cases suggest that the clinical courses and outcomes of a similar disease process may differ greatly between humans and animals, and feline SEA may respond well to treatment even if it is delayed. Furthermore, the remaining reports of feline SEA (3 single case reports and a case series of 4 patients) have noted the recovery of independent ambulation in all cases [8, 9, 20, 26]. The durations of their clinical signs ranged from a few days to a few weeks, and 4 cases had developed paralysis with or without the loss of nociception. Based on these observations, the prognosis of feline SEA appears to be very encouraging, regardless of the severity and chronicity of clinical signs. However, a publication bias (a tendency to submit and publish positive findings rather than negative findings) may exist, and, thus, a timely diagnosis and therapy are still highly desirable to minimize complications.

In conclusion, SEA is an infrequent but important disorder that needs to be included in the differential diagnosis of cats exhibiting fever, spinal pain, and/or progressive myelopathy. In chronic cases, the expansion of an epidural abscess may cause pressure atrophy of the vertebrae and severe neurological impairment. However, delays in the treatment of SEA are not necessarily associated with a poor prognosis in this species.

CONFLICT OF INTEREST. The authors declare that there were no conflicts of interest.

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