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Case Report

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Spinal decompression and stabilisation in a cat with lumbar vertebral pathological fracture and subluxation, following discospondylitis and spinal epidural empyema

Adelina Proteasa[1], Myles Benjamin Walton[2], Ines Carrera[3], Laurent S Garosi[3], Emili Alcoverro[4], Menai Heyes[4] and Anna Tauro[4]

Abstract

Case series summary A 1-year-old castrated male Maine Coon cat was referred because of a 1-week history of progressive spastic non-ambulatory paraparesis. An MRI examination of the thoracolumbar spine showed multiple lytic lesions, with the most aggressive one centred on the adjacent endplates of L1–L2 and its associated disc. Ventral new bone formation, L1 vertebral body shortening and mild dorsal displacement of the caudal aspect of L1 were noted. Contrast enhancement of both paravertebral soft tissue and extradural lesion was present. These findings were compatible with L1–L2 discospondylitis (DS), spinal epidural empyema (SEE), with secondary L1 pathological vertebral fracture, subluxation and spinal cord compression. CT of the thoracolumbar spine, abdomen and thorax confirmed these findings. The patient deteriorated to paraplegia with absent nociception, despite initial medical therapy. A right-sided L1–L2 hemilaminectomy and spinal decompression were then performed, followed by application of a unilateral construct comprising four smooth arthrodesis wires and polymethylmethacrylate (PMMA). S*taphylococcus aureus* was isolated from both epidural material, intraoperatively sampled and blood culture. Antibiotic therapy was continued for 6 weeks, based on susceptibility results. The outcome was excellent, with a gradual improvement and complete neurological recovery at the 8-week postoperative check. Repeated spinal radiographs showed an intact apparatus and marked signs of vertebral fusion. At the 14-month follow-up examination, the cat remained free of clinical signs.

Relevance and novel information To the authors' knowledge, this is the first case report of SEE and DS in a cat that required surgical stabilisation. The outcome was still optimal, despite the rapid neurological deterioration.

Keywords: Discospondylitis; spinal epidural empyema; vertebral stabilisation; vertebral instability; MRI; CT; radiograph; endplate fracture; vertebral subluxation; lumbar vertebrae; lytic lesion; paraplegia; neurological deterioration; spinal cord compression; hemilaminectomy; culture; susceptibility testing; antibiotic therapy; *Staphylococcus aureus*; Maine Coon; pedicle screws; PMMA

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Introduction

Discospondylitis (DS) is an infection of the intervertebral disc and adjacent cartilaginous endplates of the vertebral bodies.[1,2] Spinal epidural empyema (SEE) is a suppurative, septic process, with accumulation of purulent material within the epidural space.[3]

At present, DS[4–8] and SEE[3,9] in dogs are well described and recognised. Contrary to the canine patients, reports of DS[10–16] and SEE[17–22] in cats are limited and thinly distributed around several case reports. Recently, more comprehensive reports have been made available on clinical and imaging features, treatment and outcome of feline DS.[23,24] Surgical treatment of feline DS and SEE with vertebral stabilisation has not yet been reported.

Case description

A 1-year-old, male castrated Maine Coon cat was initially presented to the referring veterinary surgeon with a right pelvic limb monoparesis and was referred to ChesterGates Veterinary Specialists with a 1-week history of progressive non-ambulatory spastic paraparesis. The cat had lost weight, despite having a good appetite.

On presentation, the cat weighed 6.85 kg and had a body condition score of 4/9. Physical examination was normal. The neurological examination showed spastic non-ambulatory paraparesis. When supported, paw placement was present, while hopping response was severely delayed in the pelvic limbs. Spinal reflexes and cutaneous trunci reflex were intact. Discomfort was elicited upon palpation of thoracolumbar spine. A T3–L3 myelopathy was suspected.

Haematology and serum biochemistry profile were unremarkable.

Upon intubation, a 2-cm, irregular, pinkish, soft tissue mass was identified in the sublingual area; this was biopsied and sent for histopathology. MRI (MAGNETOM Essenza 1.5 T MRI system; Siemens) of the thoracolumbar spine showed lysis of adjacent endplates of L1–L2 and irregular shape of the associated intervertebral disc. The endplates, intervertebral disc and paravertebral muscles at this level were T2-weighted (T2W) and Short Tau Inversion Recovery (STIR) hyperintense, and showed moderate enhancement in post-contrast T1 fat-saturated images. There was L1–L2 ventral new bone formation and the vertebral body of L1 was shorter with mild dorsal displacement of its caudal aspect, suggesting fracture and subluxation. In addition, there was T13–L3 moderate meningeal enhancement, and a circumferential extradural lesion at L1–L2, isointense in T2W and with marked enhancement, compressing the spinal cord. The spinal cord at this level was mildly hyperintense in T2W images (Figure 1), indicating spinal cord oedema. A similar lesion was affecting the adjacent endplates of T9–T10 and the physis adjacent to the cranial endplate of T10, without evidence of extradural lesions. In summary, the MRI showed an aggressive lesion centred on the endplates and associated disc at L1–L2, compatible with DS; this was associated with SEE and meningitis, spinal cord oedema and compression, and pathological fracture and subluxation of L1. The lesion at T9–T10 could represent another site of infection, but less aggressive, and with no signs of fractures, luxation or extradural compression. The presence of a pathological fracture and subluxation of L1 was suspected, given the L1 shortened vertebral body. CT (SOMATOM Scope 16-slice CT system; Siemens) of the thoracic and lumbosacral spine confirmed the above findings (Figure 2). Thoracic and abdominal CT failed to identify any extraneural source of infection.

Cerebrospinal fluid (CSF) and urine samples collected via lumbar puncture and cystocentesis, respectively, were submitted for both analysis and culture. Blood and urine cultures, and *Brucella canis* antibody test were requested. While awaiting test results, medical therapy was initiated and included amoxicillin-clavulanate (10 mg/kg IV q8h), marbofloxacin (2 mg/kg IV q24h) and itraconazole (5 mg/kg PO q24h).

Multimodal analgesia was provided: methadone (0.2 mg/kg IV q4h), meloxicam (0.05 mg/kg SC q24h) and gabapentin (10 mg/kg PO q8h). An indwelling urinary catheter was placed. Clinical signs deteriorated within 24 h into paraplegia with questionable nociception. A right-sided L1–L2 hemilaminectomy was performed: the epaxial and adjacent soft tissue were removed, uncovering a subluxated L1–L2 zygapophyseal joint. The pedicles and vertebral articular processes at this level were removed, exposing an extradural, firm, greyish material, adherent to the dura mater, L1 nerve root and inner periosteum. This was gently removed, exposing an oedematous spinal cord. The epidural material was submitted for culture and histopathology. A biopsy from adjacent epaxial muscles was also submitted for histology. The laminectomy site was copiously flushed with sterile saline, followed by vertebral stabilisation. This was achieved using a four-pin unilateral construct, with two smooth arthrodesis wires placed in each vertebra and subsequently embedded into gentamicin-impregnated polymethylmethacrylate (PMMA). Postoperative radiographic assessment demonstrated good apposition and alignment of the L1–L2 vertebrae (Figure 3 a1,a2).

CSF analysis showed increased protein level (67.3 mg/dl; reference interval <45 mg/dl) with no evidence of pleocytosis or infectious agent; CSF fungal and bacterial cultures were negative.

Urine analysis and cytology were unremarkable. Urine bacterial culture was negative.

Histopathology of the epaxial musculature showed fibrosis and mixed inflammatory infiltration, while histology of the mass under the tongue demonstrated inflammatory granulation tissue.

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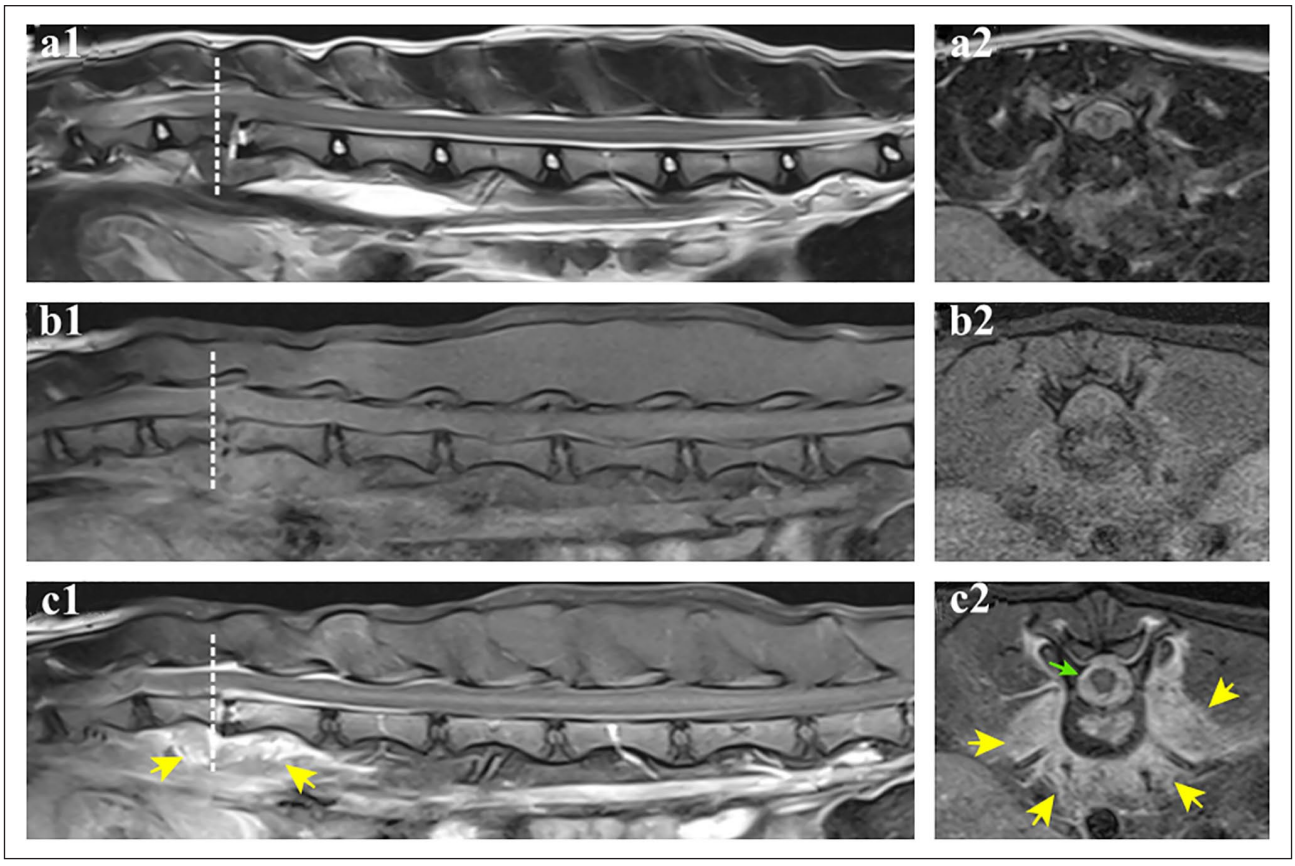

Figure 1 (a1) Sagittal and (a2) transverse T2-weighted, and (b1) sagittal and (b2) transverse pre-contrast, and (c1) sagittal and (c2) transverse post-contrast T1-weighted fat-suppressed MRI of the thoracolumbar spine, showing irregular L1–L2 intervertebral disc and endplates, shortening of the L1 vertebral body and mild dorsal displacement of the caudal aspect of L1. Paravertebral muscles (yellow arrows), disc and circumferential epidural lesion (green arrow) at L1–L2 demonstrate severe contrast enhancement

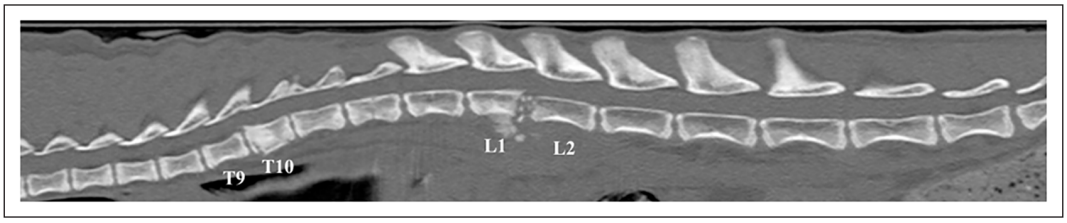

Figure 2 Mid-sagittal CT image of the thoracolumbar spine, showing irregular endplates at L1–L2, and milder at T9–T10. Note the shortening of L1 vertebral body and the mild dorsal displacement of its caudal aspect in relation to L2

Serology for *B canis* was negative. Bacterial cultures from both blood and epidural material demonstrated a *Staphylococcus aureus* infection and confirmed the susceptibility to amoxicillin-clavulanate. Fungal culture from the epidural material was negative. Marbofloxacin and itraconazole were discontinued. After 72 h of intravenous administration, antibiotic therapy with amoxicillin-clavulanate was continued orally for 6 weeks.

Eight weeks later, the patient was ambulatory with no neurological deficits. Repeated spinal radiographs showed an intact apparatus and marked signs of vertebral fusion (Figure 3, b1 and b2). At the 14-month follow-up examination, the cat was neurologically normal, and no signs of recurrence were reported.

Discussion

Vertebral instability secondary to DS and SEE has not previously been reported in a cat.

Based on the existing literature, the authors suggest features to help clinicians identify signs related to DS and SEE in cats. Gomes et al[24] described the feline population affected by DS as old (ie, median age of 9 years)

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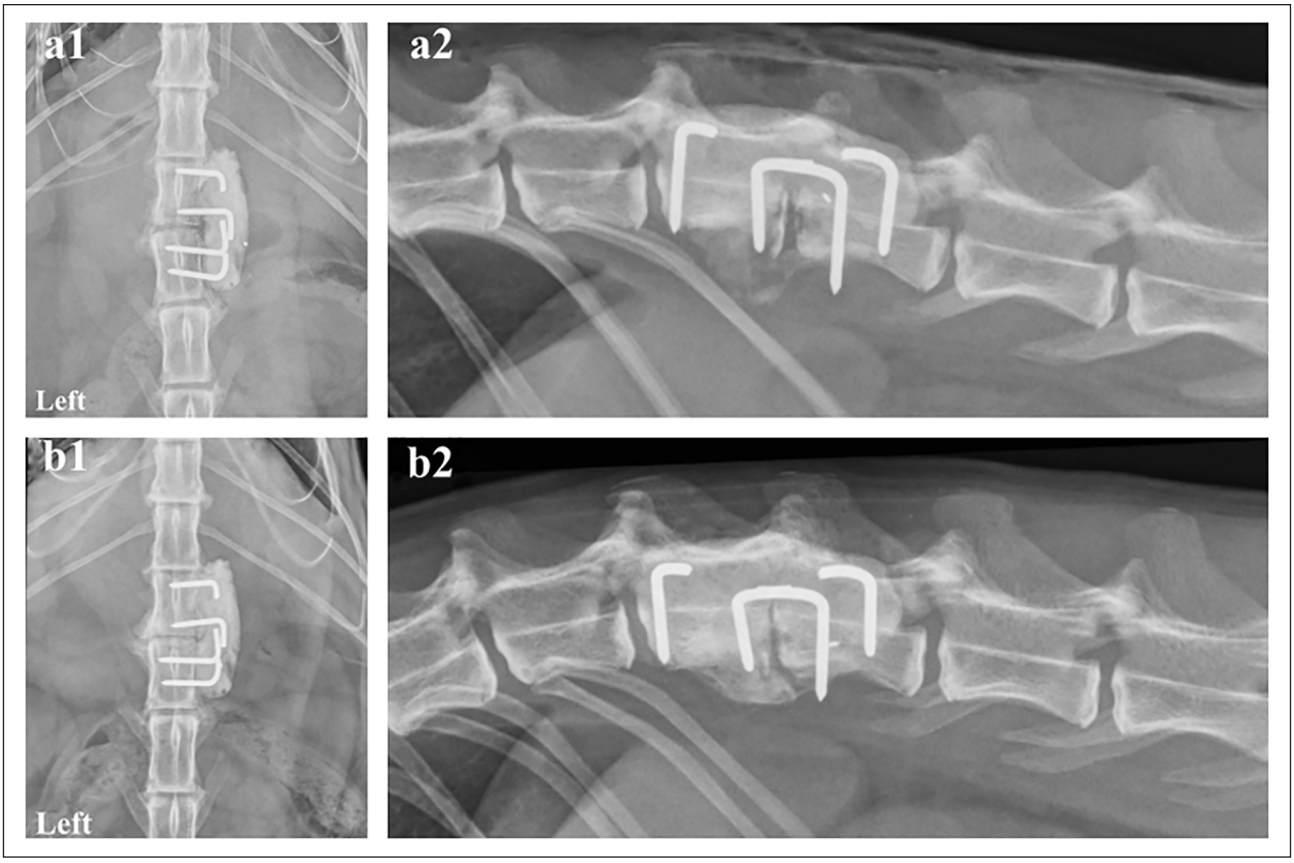

Figure 3 (a1) Dorsoventral and (a2) right lateral postoperative radiographs, showing adequate placement of the construct and alignment of the vertebral column. (b1) Dorsoventral and (b2) right lateral 8-week follow-up radiographs, showing partial L1–L2 vertebral fusion, good alignment and apposition of the structures involved, and intact apparatus

male cats. Hyperaesthesia was always present and, although DS can affect the spine anywhere, L7-S1 was the most common site.[24]

Guo and Lu[20] reported that SEE is instead commonly found in young cats (ie, aged <3 years). Spinal pain was not a common feature. Thoracolumbar spine was mostly affected, leading to more severe neurological progression. Pyrexia and haematological changes were not consistent findings, and there was often evidence of haematogenous spread of bacteria from a distant site (such as pododermatitis or gingivitis) or direct extension from a nearby infected area (such as bite wound or vertebral osteomyelitis).[20]

The current case has more features related to SEE, being a young cat with a thoracolumbar myelopathy, and lack of spinal pain. Pyrexia and haematological changes were not present.

Previous studies reported L7–S1 subluxation associated with DS that did not require surgical intervention.[14,23] In our case, we hypothesised vertebral instability as cause for rapid neurological deterioration. Our theory was supported by acknowledgement of the level of the lesion (lumbar) and patient size as contributing co-factors for vertebral instability. Moreover, failure of the intervertebral disc and the lysis of the endplates and adjacent vertebral bodies, secondary to the infectious process, resulted in loss of the ventral buttress, therefore loss of ability to resist axial load and bending. Progression of the SEE, although considered to be an aggravating cause for the neurological signs, could not be verified due to the lack of repeated imaging studies.

The suspected multifocal DS in our case may suggest haematological spread, and the inflammatory granulation mass under the tongue could be the primary infection site; however, this could not be confirmed as tissue culture was not performed.

We also noted new bone formation and vertebral body shortening. We speculate that these changes, as previously described in humans and animals,[15,24–26] can relate to chronic changes and lack of early recognition of clinical signs.

In accordance with the previous literature,[19,20] the pathogen was isolated from both blood and epidural material, supporting the presence of bacteraemia.

Owing to the multifocal lesion localisation and the zoonotic risk, *B canis* was considered; however, cats seem to be resistant to infection with most strains of *Brucella* species.[27] In our case, serology was negative. In cats, infection with *Brucella suis*[28] and *Brucella abortus*[29] were reported outside the UK. However, owing to the

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lack of contact of our case with farm animals and the strict UK surveillance programme for imported livestock, other *Brucella* species serotypes were considered unlikely, hence this cat was not tested for these.

In humans as well as in animals, MRI is considered the modality of choice to investigate spinal infections.[7,18,20,23,30,31] MRI features in our case were similar to those previously reported.

In addition, CT is applicable to localise extraneural sources of infection and identify osseous lesions.[23,31] In our case, it better defined the vertebral lesions and favoured surgical planning.

The treatment of feline DS and SEE has been reported to be predominantly medical[10–16,24] and sporadically associated with spinal decompression surgery.[15,17–20] In our case, the decision for surgical intervention was dictated by the severity of both spinal cord compression and neurological dysfunction.

The most common bacteria identified in DS and SEE are *Escherichia coli*[10–12,20,24] and *S aureus*,[19,20,24] followed by *Pasteurella multocida, Streptococcus* species, *Actinomyces viscosus, Enterococcus* species and *Clostridium perfringens*.[10,13,17,20] The suggested antibiotic monotherapy is cephalosporin or amoxicillin-clavulanate.[24] If an anaerobic infection is suspected, metronidazole can be added at 7.5–10 mg/kg q12h, to reduce risk of neurotoxicity.[32,33] Fluoroquinolones and third-generation cephalosporins should be reserved for infections where no other effective alternative exists, owing to increased risk of antibiotic resistance.[34] In our case, a combination of amoxicillin-clavulanate, marbofloxacin and itraconazole were initially used to cover a wide range of bacterial and fungal agents. Amoxicillin-clavulanate (12.5 mg/kg PO q12h) was continued for 6 weeks, based on the susceptibility results. Unfortunately, no inflammatory biomarkers, such as feline serum amyloid A, were used to help diagnose the disease or monitor treatment efficacy.[35]

There are no studies to support the optimal duration of the antibiotic therapy, but 3–5 days of initial intravenous administration followed by 6–12 weeks of oral antibiotics has been recommended to reduce the risk of recurrence.[1,2,24] In our case, surgical decompression also allowed the use of gentamicin-impregnated PMMA, which released the antibiotic locally. Gentamicin is considered effective against some Gram-positive organisms[36]; however, this was not included in our antibiotic susceptibility testing. Further, insertion of implants in infected sites can be disputed; optimal outcomes in people[37,38] and animals[39,40] have been previously reported.

The main limitations of this report were the lack of screening for additional cervical osseous lesions and repeated preoperative imaging to demonstrate lumbar instability. Therefore, we could not be certain whether the rapid neurologic deterioration was owing to instability or to the progression of the epidural empyema. The mass under the tongue should have been cultured to confirm it as a primary site of infection.

Conclusions

This case demonstrates the benefits of combined MRI and CT to diagnose DS and SEE, detect whether multiple infection sites are present and their clinical relevance, identify possible extraneural sources of infection, and determine the most appropriate treatment. The decision of medical vs surgical treatment should be based on both imaging findings and neurological assessment. Blood and epidural material cultures were essential for diagnosis.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

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