CASE REPORT

Clinical presentation, diagnosis, treatment and outcome of spinal epidural empyema in four cats (2010 to 2016)

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This case series reviews previous publications and reports four feline spinal epidural empyema cases that presented with non-ambulatory thoracolumbar myelopathy. Two cats underwent myelography and two MRI. Bacteria were obtained in three cases, in two from epidural abscesses and from a tail base wound in one; histopathological examination of epidural tissue showed pyogranulomatous changes in the remaining cat. Three cats were treated by surgical decompression plus antimicrobial therapy and one cat was treated medically. All cats showed satisfactory improvement following treatment over a follow-up period of 3 months. Spinal epidural empyema is a rare condition but all cats in this series had favourable outcomes.

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INTRODUCTION

Spinal epidural empyema (SEE) is an accumulation of purulent material in the epidural space of the vertebral canal that causes spinal cord dysfunction due to mechanical compression and inflammation. In human SEE, most infections are bacterial with a minority of abscesses caused by mycobacteria, fungi or parasites (Reihsaus *et al.* 2000). Human SEE is a rare medical emergency and accounts for 0·2 to 2/10,000 hospital admissions (Grieve *et al.* 2000). It is even more rarely reported in animals, the majority have been in dogs (Remedios *et al.* 1996, Dewey *et al.* 1998, Jerram & Dewey 1998, Cherrone *et al.* 2002, Nykamp *et al.* 2003, Lavely *et al.* 2006, Cross *et al.* 2008, Sutton *et al.* 2010, Monteiro *et al.* 2016) and it has also been reported in two cats (Granger *et al.* 2007, Maeta *et al.* 2010). Clinical signs in dogs and cats are comparable to those in humans, varying from fever, spinal hyperaesthesia to spinal cord dysfunction (Hanigan *et al.* 1990, Rigamonti *et al.* 1999, Grieve *et al.* 2000, Reihsaus *et al.* 2000, Curry Jr *et al.* 2005, Duarte & Vaccaro 2013). Routes of infection include haematogenous spread, contiguous infections from adjacent tissues, direct inoculation and migration of foreign bodies or aberrant parasites (Reihsaus *et al.* 2000, Curry Jr *et al.* 2005, Barrs *et al.* 2007, Monteiro *et al.* 2016). Rapid progression of clinical signs is possible and delayed diagnosis and treatment may result in permanent neurological dysfunction and higher mortality. In humans surgical decompression combined with antimicrobial therapy has been the treatment of choice although there have been reports of successful treatment with antibiotics alone in selected patients (Rigamonti *et al.* 1999, Grieve *et al.* 2000, Soehle & Wallenfang 2002, Curry Jr *et al.* 2005, Duarte & Vaccaro 2013). Here we describe the clinical presentation, diagnosis, treatments and outcomes of four cats with SEE diagnosed in a private clinic from 2010 to 2016. The clinic received about 17,000 feline cases during this period, of which around 550 were neurological. The prevalence of spinal epidural empyema was therefore 0·02% of all feline patients and 0·7% of the neurologically affected cats.

CASE SERIES DESCRIPTION

Case 1

A 3-month-old, 1·5 kg female entire Exotic, was referred for investigation of progressive paraparesis. The cat was strictly indoor with a history of pododermatitis, which had slowly resolved, approximately 1 month before presentation. Twenty days before presentation she was transiently pyrexic (39·9°C), then became lethargic for a few days until 2 days before presentation she developed acute paraparesis and deteriorated rapidly.

On physical examination, the cat had normal vital signs and normal rectal temperature (38·7°C). Neurological examination demonstrated non-ambulatory paraparesis with preservation of "deep pain" sensation. Spinal reflexes of the pelvic limbs were exaggerated and thoracolumbar pain was evident. Neuroanatomical localisation was between T3 and L3 spinal cord segments.

Serum biochemical profile was normal. Complete blood count (CBC) revealed leukocytosis ($23{\cdot}42\times10^9$ cells/L; reference interval 5·5 to $19{\cdot}5\times10^9$ cells/L) and lymphocytosis ($16{\cdot}03\times10^9$ cells/L; reference interval 0·4 to $6{\cdot}8\times10^9$ cells/L). Plain radiographs of the vertebral column were unremarkable. Myelography was chosen over low-field MRI because of the small size of the patient. Cerebrospinal fluid (CSF) was collected from the lumbar cistern for routine analysis before the contrast agent [Iohexol 300 mgI/mL (Omnipaque; GE Healthcare) at 0·7 mL/kg) was injected. Post-contrast radiographs showed extradural compression on the left side from L1 to L3 vertebrae (Fig. 1A).

Preoperative antibiotic therapy with a dose of cephalexin 22 mg/kg (Stazolin; Standard Chem. & Pharm Co.) intravenously (iv) was started according to the spinal surgery protocol of the clinic. A left hemilaminectomy from L1 to L3 revealed abnormal pale-coloured epidural tissue. This tissue and parts of L2 lamina were submitted for histopathological examination. CSF analysis was consistent with mild blood contamination. Histopathological examination revealed reactive new bone formation with inflammation of L2 lamina (Fig. 1B) and pyogranulomatous inflammation of the epidural tissue (Fig. 1C); no aetiological agent was identified. Eubacterial fluorescence *in situ* hybridisation of the tissue revealed no bacteria. A blood sample was sent for PCR testing for feline coronavirus, which was negative.

Postoperative management consisted of antimicrobial therapy with doses of 22 mg/kg cephalexin (Stazolin; Standard Chem. & Pharm Co.) iv every 8 hours and analgesic of 0·02 mg/kg buprenorphine (Temgesic; Reckitt Benckiser Healthcare Limited) iv every 6 hours). The cat improved and was discharged 5 days after surgery with oral dose of 20 mg/kg cephalexin (Rilexine; Virbac) orally every 12 hours for 2 weeks. At a 9-day recheck, the cat was ambulatory with a mild proprioceptive deficit in the left pelvic limb. At 6-week and 5-month examinations, she was ambulating normally with no neurological deficits.

Case 2

A 3-year-old, 4·3 kg, male neutered Persian was presented for insidious onset of paraparesis. The cat had a traumatic accident when he was 1-month old that resolved with conservative management but resulted in kyphosis at T13 to L1 and L1 vertebral tilting. At 3 months of age, he developed a skin abscess, which was drained and from which *Clostridium* species were cultured.

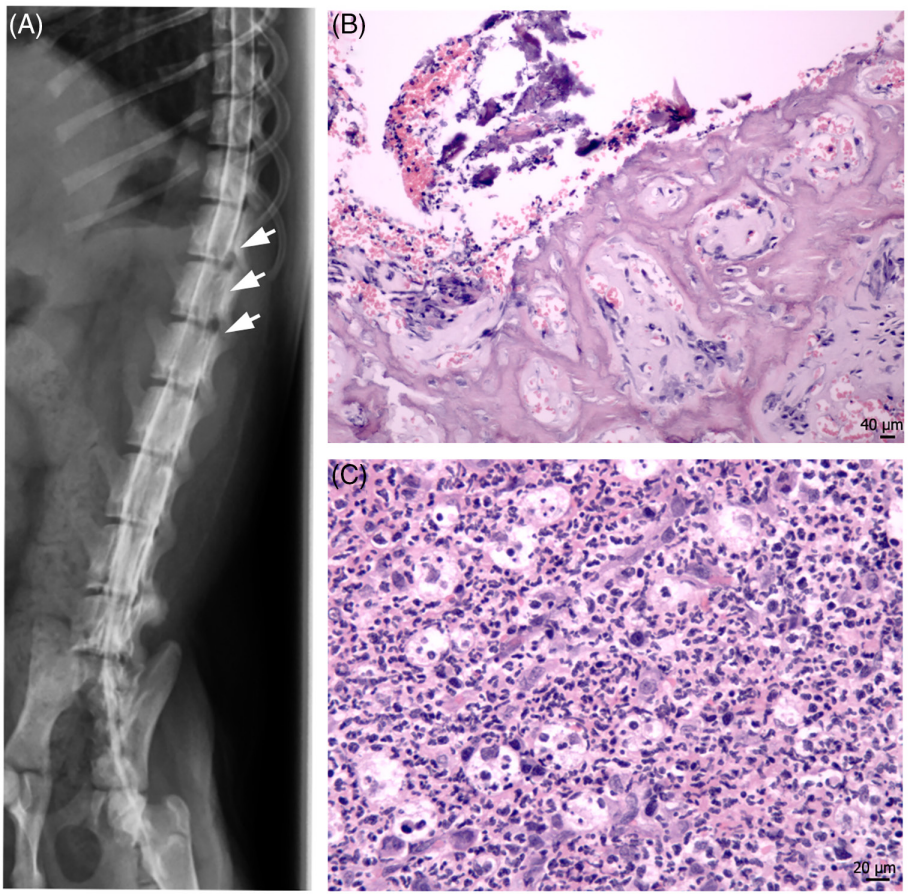

FIG 1. (case 1). (A). Lumbar myelography, oblique view: deviation of the left contrast column (white arrows). (B) Haematoxylin and eosin (H&E) staining of the lamina: reactive new bone among skeletal muscle and fibrocartilage. (C) Foci of inflammation of epidural tissue: neutrophils and macrophages admixed with fibrin, karyorrhectic debris and reactive fibroblasts

At 2 years of age, another abscess was drained from the right flank without repeated culture. A short period of spontaneously resolving paraparesis was reported a few months before presentation, during which the cat was non-ambulatory paraparetic with exaggerated spinal reflexes of the pelvic limbs. "Deep pain" sensation was intact and thoracolumbar hyperaesthesia was detected. Body temperature was 38·3°C. A T3 to L3 myelopathy was suspected.

Serum biochemistry showed mild elevation in total protein (91 g/L; reference interval 57 to 89 g/L) and globulin (60 g/L; reference interval 28 to 51 g/L). CBC was normal, FeLV (antigen test) and FIV (antibody test) tests were negative.

The cat was anaesthetised for imaging. Due to suboptimal images obtained with low-field MRI (0·2 T, General Electric MR Goldseal), myelography was performed. On plain radiographs, fusion of the vertebral bodies of T13 and L1 was noted. CSF was collected from the lumbar cistern. Postcontrast radiographs showed extensive left-sided extradural compression from T10 to L2. CSF analysis showed elevation of white blood cells (WBC 49 cells/μL; reference interval <6 cells/μL) with lymphocytic pleocytosis.

Left hemilaminectomy between T11 and L2 was performed the following day. A large amount of pus and cream-coloured tissue were removed to decompress the spinal cord (Fig. 2A and B). Culture of these materials yielded heavy growth of *Pasteurella multocida*. Histopathological analysis showed chronic active pyogranulomatous inflammation.

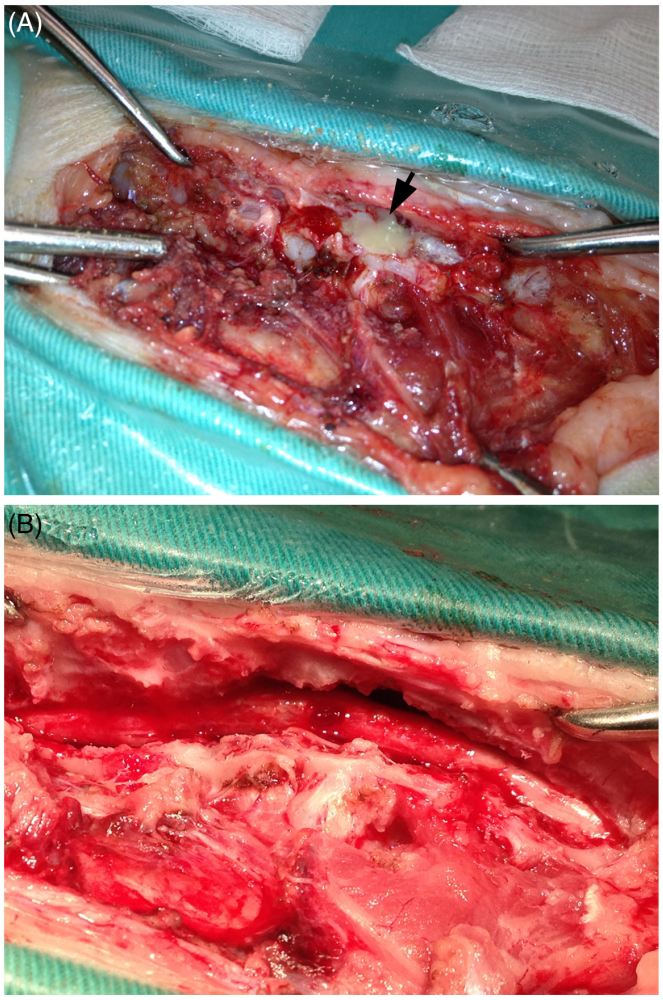

FIG 2. (case 2). (A) Intraoperative view: purulent discharge from intervertebral foramen (black arrow). (B) The spinal cord: decompressed following left hemilaminectomy from T10 to L2

The cat was treated with empirical antibiotic therapy initially with doses of 22 mg/kg cephalexin [Stazolin; Standard Chem. & Pharm Co.) iv every 8 hours and 25 mg/kg ampicillin (Unasyn IM/IV; Pfizer) iv every 8 hours and 0·1 mg/kg morphine (DBL; Hospira) iv every 6 hours. There was neurological deterioration; the cat became paraplegic and lost "deep pain" sensation. Improvement was observed from day 3 postoperatively. The cat was discharged on day 13 postoperatively with oral antibiotics prescribed according to culture and sensitivity test results [dose of 12·5 mg/kg clavulanic acid potentiated amoxicillin (Noroclav; Norbrook) orally every 12 hours for a total 8-week course]. At 3-week recheck, the cat was ambulatory with proprioceptive deficits in the pelvic limbs and at 6-week, 3-month and 2-year examinations, he was ambulating with a relatively normal gait and normal proprioception.

Case 3

A 1-year-old, 3·4 kg, female neutered domestic shorthair (DSH) was referred for evaluation of 2 days of progressive paraparesis. Four months before presentation she fought with another cat and was bitten on the left hock. The wound was subsequently infected and an abscess formed, which required draining and resolved. Two days before presentation the cat became less active and mild limping was noticed; this progressed rapidly to non-ambulatory paraparesis. Routine blood test at the referring clinic revealed elevation in total protein (90 g/L; reference interval 57 to 89 g/L) with hyperglobulinaemia (56 g/L; reference interval 28 to 52 g/L) and moderately low haematocrit (HCT) at 22% (reference interval 32·3 to 52·3%). FeLV and FIV tests were negative. Thoracolumbar radiographs taken by the referring veterinarian showed fusion of vertebral bodies L3 and L4.

At presentation, the cat was non-ambulatory, with left pelvic limb plegia and paresis of the right pelvic limb. "Deep pain" sensation was present in both pelvic limbs. Moderate lumbar pain was detected. Vital signs were normal including a normal rectal temperature (39·1°C). A T3 to L3 myelopathy was suspected.

The cat was anaesthetised for MRI (1·5 T General Electric Signa Explorer). The intervertebral disc between L2/3 appeared hypointense and misshapen in T2-weighted and short tau inversion recovery (STIR) images compared with adjacent normal hyperintense discs. There was distortion of the contour of the vertebral bodies of L2, L3 and L4, where L2 was mildly hyperintense and L3 and L4 appeared to be fused. There was mild contrast enhancement of L2 vertebral body and epaxial musculature from L2 to L4 level (Fig. 3A and B). Extradural compression was demonstrated on the dorsolateral aspect on the left side of the vertebral canal from L2 to L3 (Fig. 3C). Extradural material appeared to have mixed intensity in the T2-weighted sequence (Fig. 3D) and was contrast-enhancing (Fig. 3E). The MR findings were supportive of discospondylitis and osteomyelitis with formation of SEE.

Hemilaminectomy was performed immediately after MRI. Antibiotic therapy of 22 mg/kg cephalexin [Stazolin; Standard

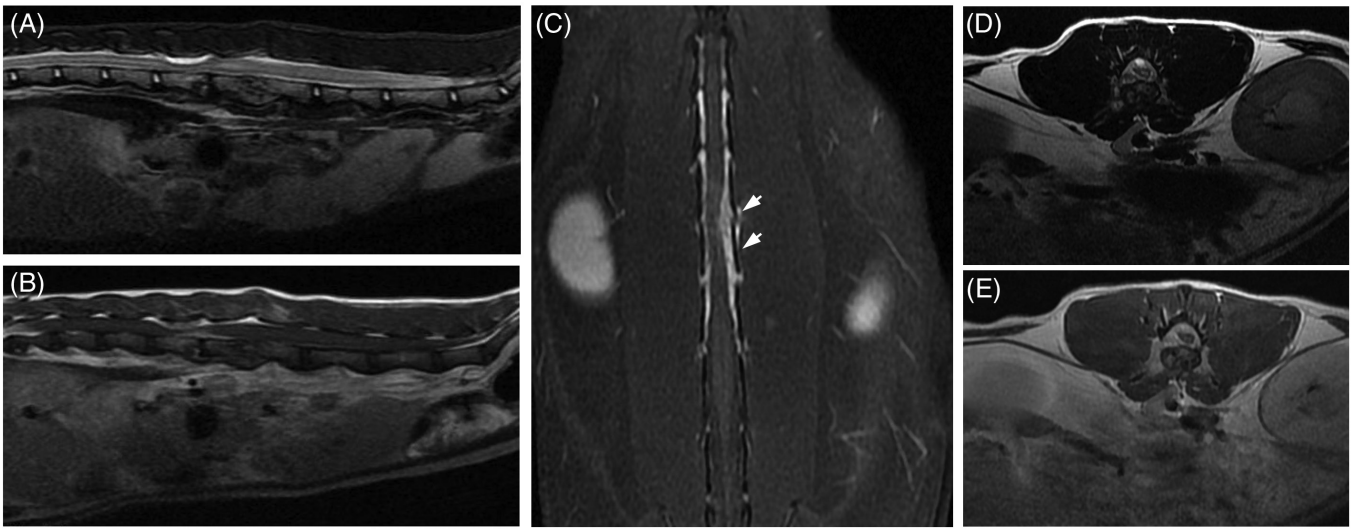

FIG 3. (case 3). (A) Sagittal STIR image: hypointense change of intervertebral disc between L2/L3; distorted vertebral bodies of L2, L3 and L4; hyperintense change in L2 vertebral body. (B). Sagittal T1W postcontrast image: contrast enhancement of L2 vertebral body, epidural material from L2 to L3 and epaxial muscle from L2 to L4. (C) Dorsal T1W postcontrast image: extradural compression on the left side from L2 to L3 vertebrae, by contrast-enhanced material (white arrows). (D) Transverse T2W image: extradural compression from dorsolateral, by tissue with mixed intensity, at the level of L3. (E) Transverse T1W postcontrast image: enhancement of the abnormal epidural tissue at the level of L3

Chem. & Pharm Co.] iv every 8 hours; potentiated 25 mg/kg ampicillin [Unasyn IM/IV; Pfizer] iv every 8 hours was initiated peri-operatively after urine and blood samples were collected and sent for bacterial culture and sensitivity testing. At surgery, purulent discharge was noticed to ooze from intervertebral foramen and a large amount of purulent discharge was released after hemilaminectomy. This was collected for bacterial culture and sensitivity testing. Abnormal tan-coloured epidural tissue was removed to achieve decompression, and the tissues were sent for histopathological examination. Urine culture generated *Escherichia coli*, while blood culture and epidural material culture generated *Staphylococcus aureus*. Histopathological examination of the epidural tissue was consistent with chronic active inflammation.

The cat improved rapidly after surgery. She was ambulatory when discharged from the hospital at 5 days after surgery with continuing oral antibiotic treatment dose of 15 mg/kg clavulanic acid-potentiated amoxicillin [Noroclav; Norbrook] orally every 12 hours. At 2-week recheck, she was ambulatory with only mild paresis of the left pelvic limb. At 2-month recheck, she was ambulatory with a normal gait. Antibiotics were continued for a total course of 12 weeks. The owner was contacted again at the time of writing this report (3 months from first presentation), who reported the cat was normal.

Case 4

A 2-year-old, 3·1 kg, female spayed DSH cat was referred for investigation of paraplegia. The cat lived outdoors and went missing for 2 weeks before presentation. Abnormalities during physical and neurological examinations included a tail base wound with purulent discharge, paraplegia, exaggerated spinal reflexes in the pelvic limbs, flaccid tail, flaccid easily expressible bladder and thoracolumbar hyperaesthesia. "Deep pain" sensation in both pelvic limbs and tail were equivocal. Rectal temperature was normal (37·8°C). Neuroanatomical localisation was from T3 to S3 spinal cord segments.

Biochemical profile showed mild elevation in blood glucose (9·18 mmol/L; reference interval 4·11 to 8·84 mmol/L) and amylase (2201 U/L; reference interval 500 to 1500 U/L). CBC showed leukocytosis (25·70×10^9 cells/L; reference interval 5·5 to 19·5×10^9 cells/L) with neutrophilia (18·00×10^9 cells/L; reference interval 1·48 to 10·29×10^9 cells/L).

The cat was anaesthetised for radiography and MRI (0·2 T General Electric MR Goldseal). Survey radiographs were unremarkable. MRI sequences performed were T2-weighted, STIR, T1-weighted and postcontrast T1-weighted after iv injection of 0·1 mmol/kg gadopentetate dimeglumine (Magnevist; Bayer). There was material within the subcutaneous region and epaxial musculature on the right side in the mid lumbar to sacral region, which corresponded to the superficial skin wound and extended into the extradural space (Fig. 4A to C). Extensive extradural compression of the spinal cord from T13 to L7 by material suspicious of epidural abscess was observed. This extradural lesion appeared hyperintense in T2-weighted and STIR images and was contrast-enhancing. Presumptive SEE was diagnosed and a poor prognosis was given due to the severity of the neurological deficits, prolonged time lag before presentation and extent of the extradural compression. The owner declined euthanasia and wished to try medical treatment. CSF was collected from the lumbar cistern and sent for routine analysis. The skin wound was explored, cleaned and a drain was placed. Urine, blood, CSF and discharge from the wound were sent for bacterial culture and sensitivity testing. Antibiotic therapy with doses of 22 mg/kg cephalexin (Stazolin; Standard Chem. & Pharm Co.) iv every 8 hours, 10 mg/kg metronidazole (Metronidazole 500 mg; Hospira) every 12 hours, 5 mg/kg enrofloxacin (Baytril; Bayer HealthCare) every 8 hours) was initiated while waiting for laboratory results and the bladder was expressed regularly. Scanty *Sphingomonas paucimoti-*

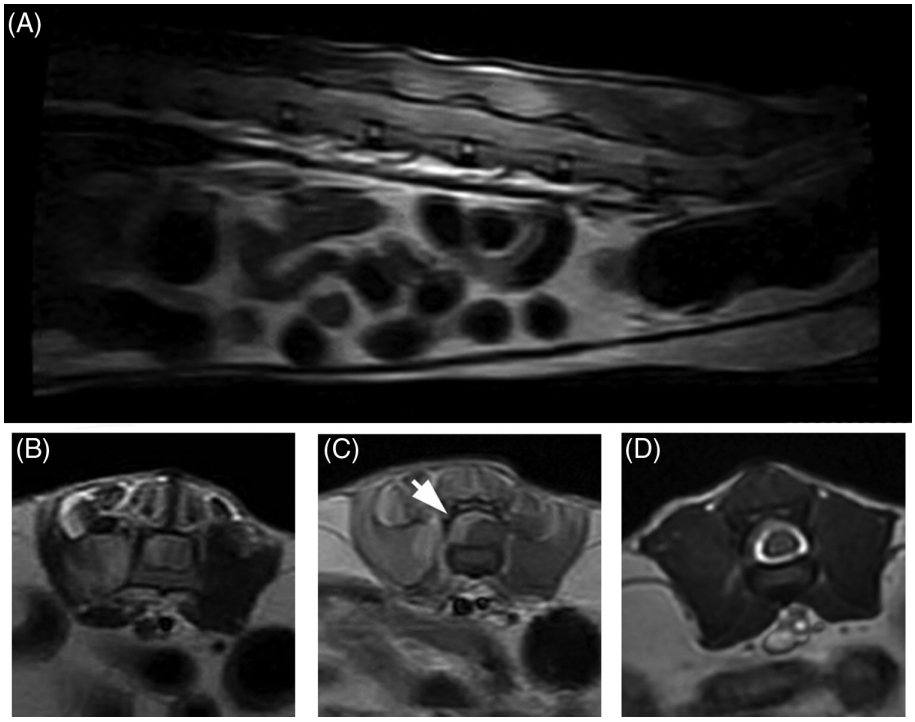

FIG 4. (case 4). (A) Sagittal T2W image: hyperintensity in spinal cord, epaxial muscle and subcutaneous region from the level of L3 to sacrum. (B) Transverse T2W image: extradural compression of the spinal cord on the right side at the level of L3 to L4 intervertebral disc space and hyperintense signal in the epaxial musculature and subcutaneous region on the right side. (C) Transverse T1W postcontrast image: enhancement of the epidural material at the level of L3/L4 (white arrow). (D) Transverse T2W image (6 days after the first MRI): disappearance of extradural compression at the level of L3 to L4 intervertebral disc space and resolved hyperintensity in epaxial muscle

lis was isolated from wound discharge and showed sensitivity to most antibiotics, while the rest of the culture results were negative. CSF analysis showed blood contamination.

The cat improved slowly and voluntary movement of its left pelvic limb returned. MRI was repeated after 6 days. The epidural lesion had almost disappeared, with the spinal cord minimally compressed and the hyperintensity in the epaxial muscle also resolved (Fig. 4D). The cat was discharged with oral antibiotics: 20 mg/kg cephalexin (Rilexine; Virbac) every 12 hours, 10 mg/kg metronidazole (Flagyl; Sanofi] every 12 hours, 5 mg/kg enrofloxacin (Baytril; Bayer) every 24 hours). At 5-day recheck, the cat had regained voluntary movement in both pelvic limbs; its bladder was distended and easily expressed; the skin wound was clean and the drain was removed. Antibiotics were continued for a total of 8 weeks. At 2-month recheck, the cat was ambulatory paraparetic, with proprioceptive deficits; the bladder was still distended and could be expressed easily.

The owner was contacted at the time of writing this report (39 months from last visit), who reported the cat had a normal gait and there was no urinary or faecal incontinence.

DISCUSSION

SEE is a rare condition in dogs and cats and only a few cases have been reported (Remedios *et al.* 1996, Dewey *et al.* 1998, Jerram & Dewey 1998, Cherrone *et al.* 2002, Nykamp *et al.* 2003, Lavely *et al.* 2006, Granger *et al.* 2007, Cross *et al.* 2008, Maeta *et al.* 2010, Sutton *et al.* 2010, Monteiro *et al.* 2016). Table 1 summarises previously reported SEE and our current cases. All of our cases were followed up for at least 3 months and all had satisfactory outcomes.

In previously reported veterinary SEE cases, clinical signs varied from spinal pain only to plegia with loss of nociception. Progression was observed in most cases. Pyrexia was not a consistent presenting sign (Dow *et al.* 1988, Remedios *et al.* 1996, Dewey *et al.* 1998, Jerram & Dewey 1998, Klopp *et al.* 2000, Cherrone *et al.* 2002, Lavely *et al.* 2006, Barrs *et al.* 2007, Granger *et al.* 2007, Cross *et al.* 2008, Monteiro *et al.* 2016). The four cases reported here were all progressive and non-ambulatory by the time of presentation, with normal body temperature. Normal core body temperature does not preclude the diagnosis of SEE.

In our cases, three cats were believed to have haematogenous spread of infection from a distant site (case 1, chronic pododermatitis; case 2, skin abscess; case 3, cat bite wound) and one case (case 4) was considered to have contiguous infection from the tail base wound. All cats in this series were young (ranging from 3 months to 3 years old) and both cats in the previous reports were 2 years old (Granger *et al.* 2007, Maeta *et al.* 2010), making young age a possible risk factor for SEE. The active nature of young cats may make them more susceptible to acquiring fight wounds.

Early diagnosis of SEE is imperative to initiate appropriate treatment and prevent permanent neurological dysfunction. Diagnosis of SEE is greatly facilitated by the increased

Table 1. Summary of SEE cases

Species	Breed	Location of SEE	Bacterial culture (source of culture)	Treatment	Outcome
SEE cases from previous literature					
Dog (Remedios *et al*. 1996)	German shepherd	Lumboscral	*Enterococcus faecalis* + *E. coli* (A)	Antibiotics	Recovered
Dog (Dewey *et al*. 1998)	Irish wolfhound	C1-C3, T3-L1	*Streptococcus canis* (A)	Epidural lavage+Antibiotics	Euthanased
Dog (Dewey *et al*. 1998)	Irish wolfhound	T4-L6	*Staphylococcus intermedius* + *Clostridium perfringens* (A)	None	Euthanased
Dog (Jerram & Dewey 1998)	Australian heeler dog	T13-L4	No growth (A)	Left hemilaminectomy T13-L4 + Antibiotics	Recovered
Dog (Cherrone *et al*. 2002)	Mastiff	L1	No growth (A)	Dorsal laminectomy over L1 antibiotics	Recovered
Dog (Nykamp *et al*. 2003)	Boxer	C3-4	*Streptococcus* group G (A)	Dorsal laminectomy C3-4	Died postop
Dog (Lavely *et al*. 2006)	Labrador retriever	L3-L6	*Klebsiella pneumonia* + *Pasteurella hemolytica* (A)	Hemilaminectomy L2-L3 dorsal laminectomy L6-7 + Antibiotics	Recovered
Dog (Lavely *et al*. 2006)	Great Dane	T2-L2	*E. coli* + *Bacteroides* species + *Prevotella* species (A)	Hemilaminectomy L2-L3 + Antibiotics	Euthanased
Dog (Lavely *et al*. 2006)	Australia cattle dog	L2-3	*Bacteroides* species + *Prevotella* species (A)	Hemilaminectomy L2-3 + Antibiotics	Recovered
Dog (Lavely *et al*. 2006)	Rhodesian ridgeback	T10-13	*Streptococcus canis* (B)	Hemilaminectomy T9-L1 + Antibiotics	Recovered
Dog (Lavely *et al*. 2006)	Mixed breed dog	T9-10, T12-13	No growth (C)	Hemilaminectomy T9-T10, T12-T13 + Antibiotics	Recovered
Dog (Lavely *et al*. 2006)[6]	Bernese mountain dog	T5-6 T10-11	*Staphylococcus intermedius* (B) + *E coli* (U)	Hemilaminectomy T10-T11 + Antibiotics	Euthanased (unrelated)
Dog (Lavely *et al*. 2006)	Labrador retriever	L2-3	*Staphylococcus intermedius* (B)	Hemilaminectomy L2-L3 + Antibiotics	Recovered
Dog (Sutton *et al*. 2010)	Dobermann	L2-3	*Prevotella* species + *Clostridium perfringens* (A)	Hemilaminectomy L2-L3 + Antibiotics	Recovered
Dog (Monteiro *et al*. 2016)	Golden retriever	L1-7	No growth (U)	Antibiotics	Recovered
Dog (Monteiro *et al*. 2016)	Labrador retriever	C7-T1	*E. coli* (U + B + P1)	Antibiotics	Recovered
Dog (Monteiro *et al*. 2016)	English bulldog	L5-S1	No growth (U)	Antibiotics	Recovered
Dog (Monteiro *et al*. 2016)	Boxer	T1-4	*Pasteurella* species (A)	Antibiotics	Recovered
Dog (Monteiro *et al*. 2016)	Cocker spaniel	C4-7	*Corynebacterium* species (P2)	Antibiotics	Recovered
Cat (Granger *et al*. 2007)	Domestic shorthair	C3-4	*Pasteurella multocida* (A)	Hemilaminectomy C3-C4 + Antibiotics	Recovered
Cat (Maeta *et al*. 2010)	Domestic shorthair	L3-4	No growth (A)	Dorsal laminectomy L3-L4 + Antibiotics	Recovered
SEE cases from this series					
Cat	Exotic cat	L1-3	Not submitted	Hemilaminectomy L1-3 + Antibiotics	Recovered
Cat	Persian	T11-L2	*Pasteurella multocida*	Hemilaminectomy T11-L2 + Antibiotics	Recovered
Cat	Domestic shorthair	L2-3	*Staphylococcus aureus* + *E. coli*	Hemilaminectomy L2-3 + Antibiotics	Recovered
Cat	Domestic shorthair	T13-S	*Sphingomonas paucimobilis*	Antibiotics	Recovered

A Abscess, B Blood, C CSF, U Urine, P1 Prostatic wash, P2 Pleural effusion

availability of cross-sectional imaging (CT, MRI). In humans, the combination of back pain and abnormal inflammation parameters (*e.g.* leukocytosis, accelerated erythrocyte sedimentation rate) is considered characteristic of SEE. Gadolinium-enhanced MRI is the imaging modality of choice over conventional radiography or CT-myelography as this provides a 91% sensitivity in diagnosing SEE in human patients, helps to identify alternative lesions and avoids lumbar or cerebellomedullary cisternal puncture during myelography, thus minimising the risk of iatrogenic intrathecal contamination (Reihsaus *et al*. 2000, Curry Jr *et al*. 2005). In humans, the characteristics of SEE MR images include hyperintense extradural lesion on T2-weighted images, generalised or ring contrast enhancement after iv injection of gadolinium-based agent on T1-weighted images, changes in adjacent tissue such as discospondylitis, epaxial muscle changes and sinus tracts (Rigamonti *et al*. 1999, Reihsaus *et al*. 2000, Curry Jr *et al*. 2005, Duarte & Vaccaro 2013). Similar MR changes were reported in canine SEE cases (Holloway *et al*. 2009, Carrera *et al*. 2011) and hyperintensity within the spinal cord grey matter at the site of the lesion was

reported in all five dogs in one case series (De Stefani *et al*. 2008). Nevertheless, there are no reliable MRI changes that would differentiate SEE from a possible neoplasm. Histopathological examination of the abnormal tissue remains crucial to reach a definitive diagnosis.

In the current study, myelography was performed in two cats, because their small size would have led to suboptimal images on low-field MRI. Myelography located the extradural compression but did not provide information on the extradural material, the spinal cord itself and the surrounding tissue. Low-field MRI was performed in Case 4 and high-field MRI in Case 3. In both cases, extradural lesions appeared as mixed- or high-signal material in T2-weighted images and showed diffuse strong contrast enhancement, comparable with MR features in humans and dogs. These changes were not apparent on survey radiography. Therefore MRI is considered superior in diagnosing SEE to myelography.

CSF collection is controversial in human SEE patients because it carries the theoretical risk of introducing bacteria from the epidural space into the subarachnoid space and CSF commonly displays non-specific inflammatory changes (Rigamonti *et al*. 1999, Reihsaus *et al*. 2000, Curry Jr *et al*. 2005). CSF was collected in three cases in this study and the results were non-specific. With the increased availability of high-field MRI, a standardised diagnostic procedure for SEE should be recommended to include MRI (T2-weighted, T1-weighted with postcontrast sequences), urine and blood bacterial culture and sensitivity testing. CSF collection is probably unnecessary and should be avoided if lesions identified on MRI are supportive of SEE and patients are scheduled for surgical decompression and biopsy.

Pasteurella multocida, *Staphylococcus aureus* and *Sphingomonas paucimotilis* were isolated in Cases 2, 3 and 4, respectively. In Case 1, at the time of surgery and tissue collection no bacterial culture was submitted because feline infectious peritonitis (FIP) was suspected and antibiotics were given before sample collection. The cat subsequently had negative PCR for coronavirus and continued to improve following treatment, making FIP extremely unlikely. For human and dog SEE the most frequently isolated causative bacteria are *Staphyloccocus* and *Streptococcus* species (Dewey *et al*. 1998, Rigamonti *et al*. 1999, Grieve *et al*. 2000, Reihsaus *et al*. 2000, Cherrone *et al*. 2002, Soehle & Wallenfang 2002, Curry Jr *et al*. 2005, Duarte & Vaccaro 2013). In the cervical SEE reported in one cat, *Pasteurella multocida* was cultured (Granger *et al*. 2007). However there was negative yield on culture in the other cat despite a large amount of bacteria identified on cytological examination of the epidural tissue (Maeta *et al*. 2010). In a recent literature review of human SEE, no microbiological diagnosis was identified despite blood and intraoperative cultures in 13% of patients (Suppiah *et al*. 2016). In another study of human SEE, the early use of antibiotics before culture and insufficient tissue for bacteria culture were suggested reasons leading to 24·4% cases with an unidentified causative pathogen and up to 50% of negative blood cultures (Ma & Kim 2012). Histopathological examination revealing an inflammatory extradural lesion is highly diagnostic of SEE (Cases 1, 2 and 3). Real-time PCR for bacterial species DNA, performed on EDTA anti-coagulated plasma and freshly excised portions of the extradural lesion may be more sensitive in reaching a definitive diagnosis (Cross *et al*. 2008).

The treatment options for SEE are also controversial; in humans, full recovery was estimated to be between 41 and 47% and mortality 16% for all SEE cases (Reihsaus *et al*. 2000, Curry Jr *et al*. 2005). A recent human literature review recommended that neurologically symptomatic SEE should be offered early surgical intervention with adjuvant antibiotic therapy to provide the best likelihood of neurologic recovery (Suppiah *et al*. 2016). However, some authors reported successful medical management of SEE in humans (Hanigan *et al*. 1990, Grieve *et al*. 2000) and dogs (Monteiro *et al*. 2016). In veterinary SEE both surgical and medical management have been documented with variable outcomes. In this case series, three cats were treated with decompressive surgery and a course of antibiotics. Two of them improved immediately postoperatively and one initially deteriorated but eventually recovered. We suspected the initial deterioration might be related to excessive manipulation of the spinal cord during surgery in order to remove epidural material adhered to the spinal cord. Case 4 presented with the most severe neurological deficits, of unknown duration. Despite the severity of the neurological dysfunction, the extent of the spinal cord involved, and the likely prolonged time lag before presentation, empirical antimicrobial therapy resulted in satisfactory outcome.

In conclusion, SEE is a rare progressive myelopathy that results in severe spinal dysfunction in young cats. It should be included in the differential diagnosis of cats exhibiting relevant clinical signs and prompt investigation should be initiated. The value of collecting CSF is questionable and should probably be avoided if lesions identified on MRI are supportive of SEE and patients are scheduled for surgical decompression and biopsy. In carefully selected patients, antimicrobial therapy alone might also result in satisfactory outcome, although a protracted course might be necessary.

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Conflict of interest

Part of the information was presented at previous congresses (ECVN & ESVN 26th Symposium and the World Feline Veterinary Congress 2013).

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